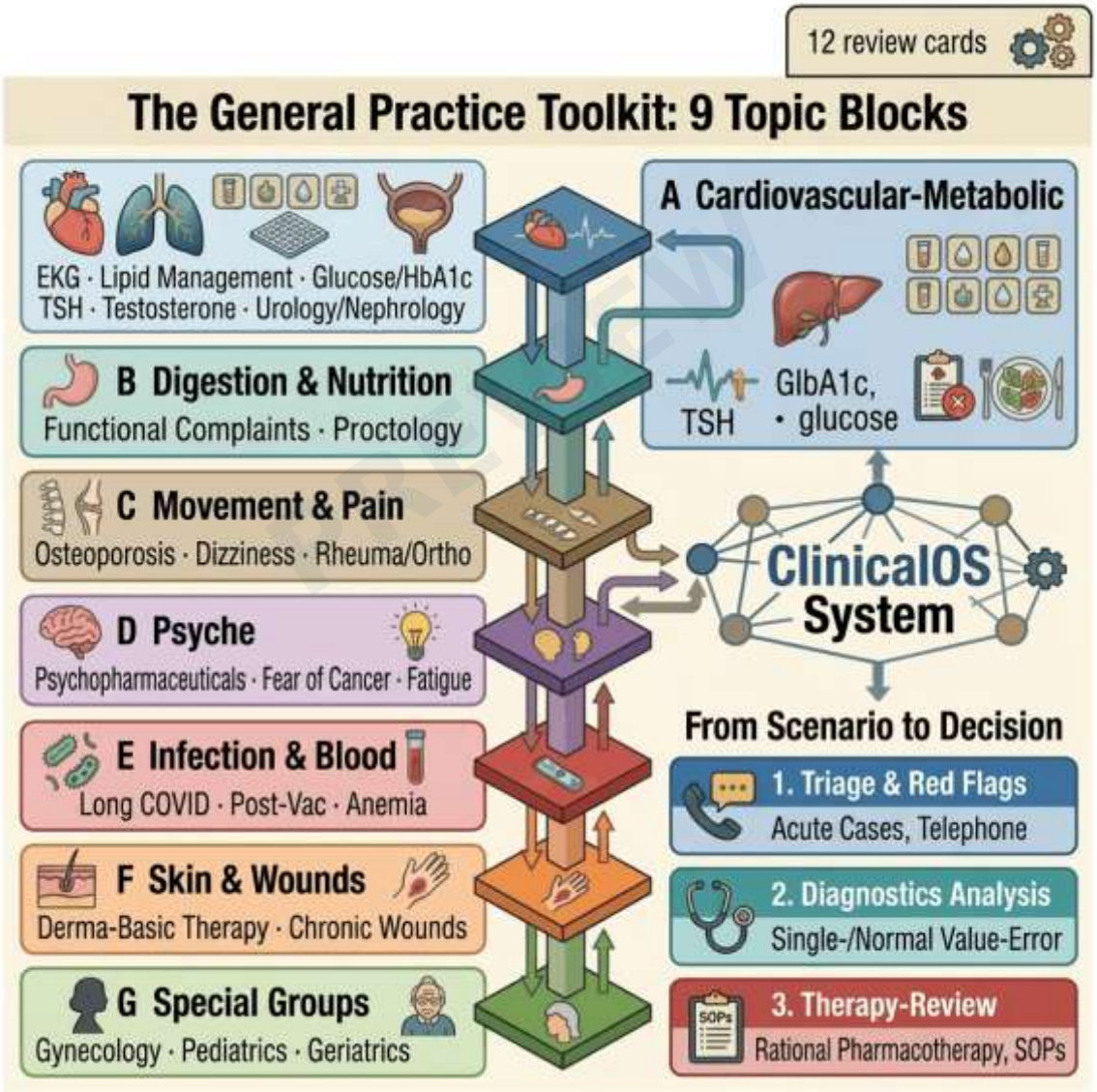
ClinicalOS – Structured decision-making in general practice

Practice Annex (C)

Complementary to Volume 4: Patient Management



Practical Management
for 20 clinical chapters and healthcare assistant (HCA) modules

Imprint

Dr Goetz Huber, MD

Practice Annex (C), complementary to Volume 4: Patient Management

Note: The information contained in this book has been carefully researched and verified. As AI tools and regulatory frameworks change rapidly, some details may already be out of date by the time you read this. You can find the latest updates at the URL provided in the book. The mention of products and manufacturers is without guarantee and does not constitute a recommendation to purchase or use them.

Note on Translation, Content, Use and Liability

This English edition is a translation of the original work written in German. The concept, structure, and core medical content of ClinicalOS were originally developed for German general practice and the German healthcare system, to provide practising GPs, trainee GPs, healthcare assistants (HCAs) and medical students with a structured, practice-oriented learning and reference resource. The content is based on current medical literature, guideline-based sources and practical experience in general practice.

During the linguistic adaptation, references to guidelines, clinical practices and regulatory frameworks in other English-speaking settings were taken into account and highlighted where relevant and feasible; in some areas, differences from German recommendations are also presented explicitly. However, the translation does not constitute a systematic adaptation of every medical, therapeutic or regulatory aspect to all English-speaking countries. Professional guidelines, marketing authorisations, approved dosages, prescribing procedures, reimbursement rules and regulatory requirements may vary between countries;

the guidelines, official prescribing information and regulatory provisions currently applicable in the respective country or healthcare system always take precedence. Dosage information applies to adults without significant renal impairment; individual adjustments are required for elderly patients, those with polypharmacy and during pregnancy. **Current national/regional guidelines (e.g. NICE, ESC, ADA) must be followed as a priority.**

Digital tools, including AI-assisted text generation, were used in the creation of this manual. All content was subsequently checked by the author for technical accuracy, consistency and potential errors. **No guarantee can be given for the correctness, currency or completeness of the information provided;** the complete absence of errors cannot be excluded, including with regard to guidelines or dosage recommendations that may have been updated in the meantime.

The contents of this manual do not replace an individual medical assessment and should not be construed as binding treatment recommendations. Diagnostic and therapeutic decisions must always be made on a case-by-case basis for each individual patient, taking into account the full clinical picture and the currently valid guidelines.

Doctors bear full and ultimate medical responsibility: any use of content from this manual — whether as a text module, SOP or treatment recommendation — requires independent professional review by the treating doctor. **AI-supported or digitally generated content cannot and must not replace medical judgement.**

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Detailed Contents

Key question: 'What is the safe and sensible course of action now?

Section	Contents
A10 – Erectile dysfunction	PDE-5 inhibitors, contraindications with nitrates, traffic light system, comparison of sildenafil and tadalafil, ruling out testosterone deficiency, treatment failure, ED in patients with depression/SSRIs, red flags, referral pathways between urology and cardiology
B Digestion & Nutrition	**Gastroenterology – vitamin deficiencies / trace elements – food allergies & intolerances**
B1 – Functional digestive complaints	Irritable bowel syndrome, irritable stomach, red flags, Rome IV criteria, gut-brain axis, low-value medicine, core GP-related responsibilities
B2 – GLP-1 and GIP/GLP-1 analogue therapy for obesity	Tirzepatide/Mounjaro, Semaglutide/Wegovy, Liraglutide/Saxenda/Nevolat, 6-step model, titration schedule, off-label risk with Ozempic, DMP for obesity **Management of gastrointestinal side effects** - Nausea, vomiting, diarrhoea, constipation, - Severity classification: mild/moderate/severe, - Titration rate, criteria for discontinuation of treatment **Interactions and drug safety** - Levothyroxine interaction, oral contraceptives - delayed gastric emptying, - insulin and sulphonylureas, risk of hypoglycaemia - Pancreatitis, ileus, medullary thyroid carcinoma **Appendix A – Patient Information Leaflets** - Treatment information for patients, differences between preparations, missed doses, - costs and reimbursement, warning signs
B3 – Proctology in GP practice	Haemorrhoids, anal margin thrombosis, anal fissure, perianal abscess, anal eczema, pruritus ani, table of leading symptoms and differential diagnoses
C Movement & Pain	**Orthopaedics / Rheumatology – Neurology – Pain Medicine**
C1 – Self-management exercises for common musculoskeletal complaints	14 prioritised clinical presentations, safety check with red flags, pain traffic light system and activity management, lower back and neck pain, osteoarthritis (knee/hip), tendinopathies and tendon complaints, fall prevention and sarcopenia, SOPs, healthcare assistant (HCA) module
C2 – Osteoporosis screening, diagnosis and management	DXA measurement, fracture risk, glucocorticoid-induced osteoporosis, fall risk, DMP Update 2025, risk constellations
C3 – Management of vertigo in GP practice	ADAC scheme (type, duration, triggers, associated symptoms), DEGAM S2k AWMF 053-018, HINTS triad, Dix-Hallpike test, Schellong test, head impulse test, BPPV, vestibular neuritis

Section	Contents
C3 – Dizziness algorithm	4 key questions: history-taking, temporal course and probable diagnoses, risk stratification: Pathway A (older/multimorbid) / Pathway B (younger), HINTS triad, Dix-Hallpike test, head impulse test, red flags, management decision
C4 – A. Pain management in GP practice: **Selection, dosing, titration and safety**	30-second pain triage, five principles of treatment, non-opioids and neuropathic pain, opioid initiation and titration, opioid rotation (six-step algorithm), management of side effects, geriatrics and renal insufficiency, tapering off and misuse
C5 - B. Cancer pain and palliative pain management **Basic analgesia, breakthrough pain, special situations and cannabinoids**	Cancer pain by mechanism, breakthrough pain and rapid-onset opioids, bone metastases, neuropathic and visceral cancer pain, alternative routes of administration (s.c.), malignant bowel obstruction, cannabinoids, geriatric cancer pain, AAPV/SAPV
D Mental Health	**Psychiatry / Psychosomatics + Substance Use Disorders**
D1 – Management of psychotropic drugs in GP practice	Depression, panic disorder, escitalopram, sertraline, mirtazapine, St John's wort, avoiding long-term benzodiazepine therapy
D2 – Cancer-related anxiety and health-related fears in GP practice	Tumour marker thresholds, NURSE model, SET model, CALM approach, health anxiety disorder, watchful waiting, red flags
D3. Functional physical symptoms and basic psychosomatic care in GP practice	Red/Yellow/Green flags, basic psychosomatic assessment in a 10- to 20-minute consultation, simultaneous diagnosis, levels of care (initial, extended, multimodal), challenging patient interactions, traffic-light algorithm, consultation phrasing, case vignettes
D4 – Management of fatigue and exhaustion in GP practice	DEGAM S3 guideline AWMF 053-002, basic laboratory tests, vitamin B12 algorithm, ME/CFS criteria (IOM), pacing, biopsychosocial model
E Infection & Blood	**Infectious diseases (& antibiotic therapy) – Haematology**
E1 – Long COVID and post-vaccination syndrome	Post-COVID syndrome, ICD U09.9, post-vaccination syndrome, differentiation by index event, vaccination as a risk-reduction measure
E2 – Diagnosis of anaemia and haemoglobinopathies	Microcytic anaemia, iron-deficiency anaemia, anaemia associated with chronic diseases, β-thalassaemia, sickle cell disease, haemoglobin electrophoresis, stepwise diagnosis
F Skin, sensory organs & allergies	**Dermatology – ENT – Ophthalmology – Allergology**
F1 – Basic dermatological treatment in GP practice	Atopic dermatitis, psoriasis, acne, rosacea, tinea, intertrigo, urticaria, 10 dermatological safety rules

Section	Contents
F2 – Acne treatment in GP practice	Classification by severity, stepwise treatment in GP-related settings, topical and systemic treatments, doxycycline, indications for isotretinoin, adherence, psychosocial burden, indications for referral to a dermatologist
F3 – Ectoparasites in GP practice	Scabies, head lice, pubic lice, body lice, fleas, bedbugs, management of contacts, hygiene measures
F4 – Treatment of alopecia in GP practice	5 patterns of alopecia, androgenetic alopecia, female pattern hair loss, alopecia areata, telogen effluvium, scarring and inflammatory alopecias, laboratory diagnostics, referral matrix
F5 – Skin cancer screening in GP practice	Statutory skin cancer screening under Section 25 of the German Social Code, Book V (SGB V), the ABCDE rule, 7-point checklist, melanoma, basal cell carcinoma, squamous cell carcinoma, actinic keratosis
F6 – Management of chronic wounds in GP practice	venous hypertension, peripheral arterial occlusive disease, diabetic neuropathy, venous leg ulcer, diabetic foot syndrome, pressure ulcers, compression therapy
G Special groups	**Gynaecology (& screening) – Paediatrics (+ adolescent medicine) – Oral and maxillofacial medicine**
G1 – PCOS – Polycystic ovary syndrome in GP practice	Rotterdam criteria, hyperandrogenism, metabolic risk, lifestyle intervention, metformin, interdisciplinary care
H Cross-disciplinary	**Geriatrics / Rational Pharmacy – Emergency Medicine**
H1 – Urgent non-classical emergencies	Sudden hearing loss, temporal arteritis, retinal artery occlusion, peripheral facial nerve palsy, testicular torsion, amaurosis fugax, missed treatment windows
I End-of-life care	**Oncology – Palliative care**
I2 – Symptom management and medication at the end of life: tools for GP practice	Pain, dyspnoea, nausea/vomiting, anxiety/agitation/delirium, palliative sedation, routes of administration and controlled substances documentation, escalation matrices, case vignettes and quick reference
ANNEX	
Annexes: GLP-1/GIP-GLP-1 therapy for obesity as a structured long-term treatment	6-step model, mini-SOP with 5 stages of practice workflow, allocation of tasks between doctor and healthcare assistant (HCA), dose extension, click-counting, long-term management, end of treatment

Table of Contents

Introduction and Instructions for Use: Bonus Annex

This bonus material is not intended as light evening reading – it is a practical tool for day-to-day consultations. Each chapter answers a specific question for GP's: What should I do now, safely and sensibly? When should I act, when should I wait, and when should I escalate?

PLEASE NOTE

GPs cannot spend half their nights and weekends keeping up to date with specialist topics. This manual takes that work off their hands – condensed, structured and ready for immediate use.

Part of the ClinicalOS system

The bonus material supplements Manual 4 (Patient Management) of the 11-volume ClinicalOS General Practice Knowledge Base – the clinical operating system for general practice:

Volume	Title
1	Decision-Making Architecture in general practice
2	Rational Diagnosis
3	Practice Pearls for General Practice
4	Patient Management + Bonus Annex (this document)
5	Digital Tools & AI
6	Multimorbidity & Rational Pharmacotherapy
7	Practice Architecture & Team Processes
8	Professional Patient Communication
9	GP-specific topics – Organisational aspects
10	Introduction and Work Manual (Students, healthcare assistants (HCA))

What makes this format special

Each chapter follows the same structure: from a common practice scenario to a confident decision. No theory without practical application.

Format element	Function in practice
Red Flag	Immediately recognisable: when not to wait, when to escalate
Practice Pearl	Clinical short-circuit: a simple insight, a major impact
Key Points Box	Key rule of the chapter – worth learning by heart
Practical mistakes	Common pitfalls – and how to avoid them
SOPs	Numbered, delegable, directly applicable in practice
Tables	Decision-making logic at a glance – no continuous text
HCA module	Clear tasks for the team: who does what, when and with what
PMS modules	Documentation texts ready to copy and paste
Case vignettes	Typical scenarios – practising classification
Online resources	Clickable links: guidelines, outpatient clinics, self-help groups in Germany, Austria and Switzerland

Contents: 9 thematic sections, 20 chapters

The bonus material is organised by clinical domain. Each chapter can be read independently – there is no need to work through the material in a linear fashion.

Block	Topic area	Chapters (selection)
A	Cardiovascular and Metabolic Medicine (including urology and nephrology)	ECG assessment · Lipid management · Glucose & HbA1c · TSH · Testosterone · PSA
B	Digestion & Nutrition	Proctology in GP practice
C	Exercise & Pain	Osteoporosis · Dizziness
D	Mental Health	Psychotropic drugs · Fear of cancer · Tiredness & exhaustion
E	Infection & Blood	Long COVID / Post-vaccination syndrome
F	Skin & Wounds	Basic dermatological treatment · Chronic wounds
G	Special Groups	PCOS – Polycystic ovary syndrome
H	Cross-disciplinary	Cross-thematic content

Living document

The supplementary material is not a finished work. It is continuously updated as new GP-related topics are added or guidelines change (work in progress). The most common and clinically challenging topics encountered in everyday GP practice have already been covered. Additions are made as required and according to clinical relevance.

HOW TO USE IT?

– Navigate directly to a topic: use the table of contents or search function.
– Each chapter stands on its own: no need to read in a linear fashion.
– Read the ‘Red Flags’ section first: a safety net before delving into the details.
– Incorporate SOPs and PMS modules directly into your day-to-day practice.
– HCA modules: Share with the team and delegate tasks.

Block A – Cardiovascular and Metabolic Medicine

CARDIOLOGY

A1 – ECG interpretation in GP practice

ECG Interpretation in GP practice

Manual 4 – Bonus Annex | Including long-term ECG

1. Basic framework and technical check

The systematic interpretation of a 12-lead ECG is a core competence of general practice. This chapter provides a reproducible basic framework, guiding the reader from technical assessment through to clinical interpretation.

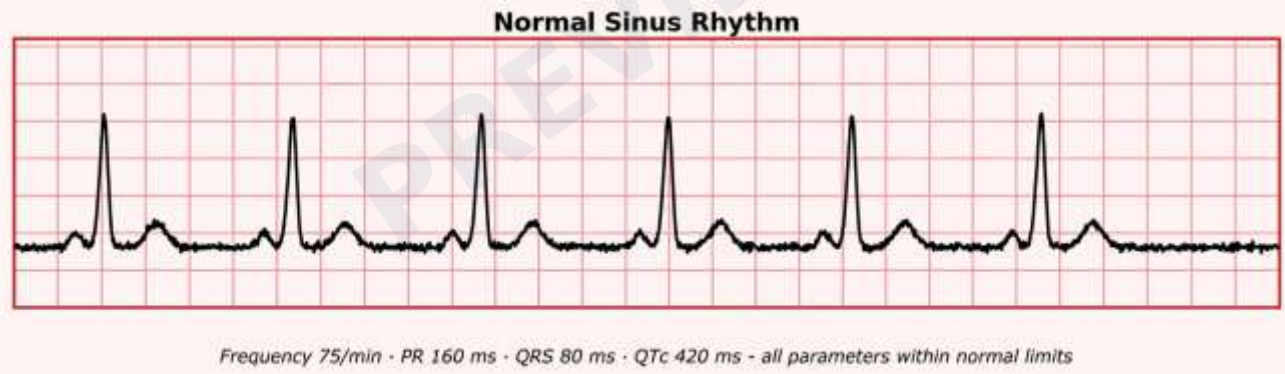

Fig. 1.1 – Normal sinus rhythm: all parameters within the normal range

1.1 Technical check prior to interpretation

Before any substantive assessment, technical parameters are checked:

- Patient identification: Are the name, date of birth, date and time correct?
- Paper speed: Standard 25 mm/s (large square = 200 ms)
- Calibration: Standard 10 mm/mV (1 cm deflection = 1 mV)
- Signal quality: stable baseline, no artefacts, no channel loss
- Electrode position: are all 10 electrodes correctly positioned and firmly attached?

Key point

A technically faulty ECG must not be interpreted. Repeating the test is not a waste of time – it prevents misdiagnoses.

1.2 The 90-second basic procedure (10 steps)

Step	Assessment	Normal values
1. Rhythm	Sinus rhythm? P wave before every QRS complex?	P positive in lead II, negative in aVR; PQ constant
2. Rate	300 ÷ number of large squares (BP)	60–100/min
3. Axis	Lead pattern I and aVF	Normal axis: I+, aVF+
4. P wave	Morphology, duration, amplitude	≤120 ms; ≤0.25 mV
5. PQ interval	AV conduction	120–200 ms, constant
6. QRS	Width, morphology, pathological Q?	<120 ms; Q <40 ms and <25% of R
7. ST segment	Elevation/depression at the J-point	±0.5 mm in the extremities, ±1 mm in the chest
8. T-wave	Polarity, morphology	Positive in leads I, II, V3–V6; negative in lead aVR
9. QTc	Bazett/Fridericia correction	♀ <460 ms; ♂ <450 ms
10. Context	Comparison with previous ECG, clinical presentation	Dynamics = Alarm

1.3 Healthcare Assistant Module: ECG recording in the practice

HCA task	When / Trigger	Important note
Set up the ECG machine	Before each recording	Check cable colours; V1/V2 exactly 4. ICR
Prepare the electrodes	Degrease and dry the skin	Shave hair short if present; do not use cream
Check the quality	Before printing	All 12 channels visible, no flatlines
Archive ECG	Immediately after recording	Label with patient details; file in the patient record system
Inform the doctor	For any abnormal findings	Never interpret the results yourself; observe the traffic light system
Service the equipment	Weekly	Check the gel; inspect cables for damage

2. Chest pain, ischaemia and heart attack

Chest pain is the most common potentially life-threatening presenting symptom in GP practice. The ECG is indispensable – but with clear limitations.

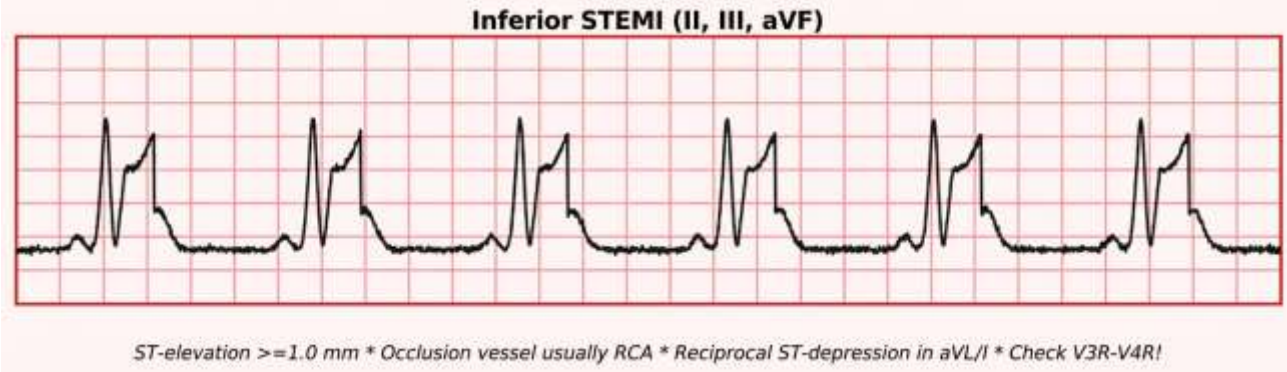

Fig. 2.1 – Inferior STEMI: ST-segment elevation in leads II, III, aVF – occluded vessel usually the RCA

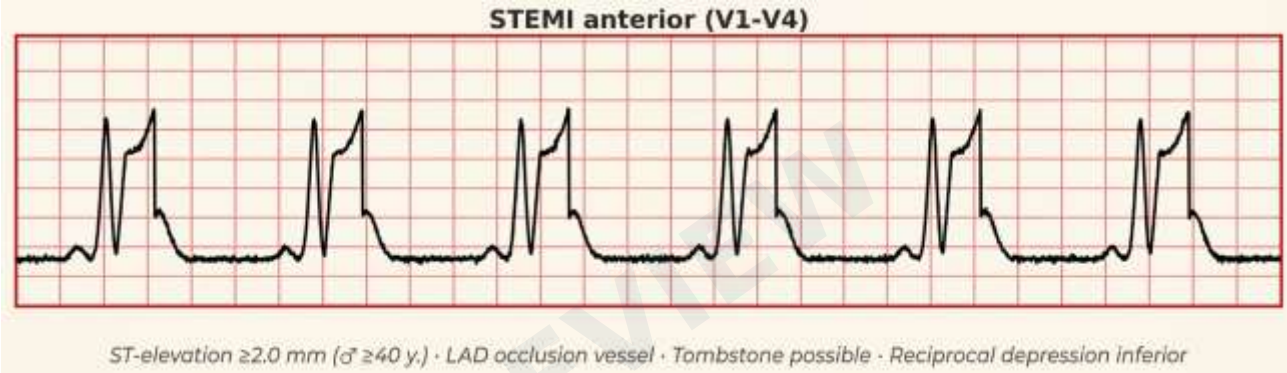

Fig. 2.2 – Anterior STEMI: ST-segment elevation in V1–V4 – proximal LAD occlusion

2.1 'Safety First' algorithm for chest pain

SOP: Chest pain in GP practice

1. Check vital signs immediately: BP, pulse, SpO_2, respiratory rate – instability?
2. 12-lead ECG within 10 minutes (start the timer!)
3. Establish venous access; administer 300 mg aspirin orally if ACS is suspected
4. ECG interpretation: STEMI criteria + clinical presentation? → Call 112 immediately
5. Troponin (hs-cTn): level + kinetics; NSTEMI possible without ECG criteria
6. Risk stratification: HEART score or GRACE score
7. Safety net: discuss and document discharge criteria

2.2 STEMI criteria (ESC 2023)

Lead	Threshold for men	Threshold for women
V2–V3 (<40 years ♂)	≥2.5 mm	≥1.5 mm
V2–V3 (men aged 40 and over)	≥2.0 mm	≥1.5 mm
Other leads	≥1.0 mm	≥1.0 mm
V7–V9 (posterior)	≥0.5 mm	≥0.5 mm
V3R–V4R (RV)	≥0.5 mm (men under 30: ≥1.5 mm)	≥0.5 mm

2.3 High-risk NSTEMI pattern without classic STEMI criteria

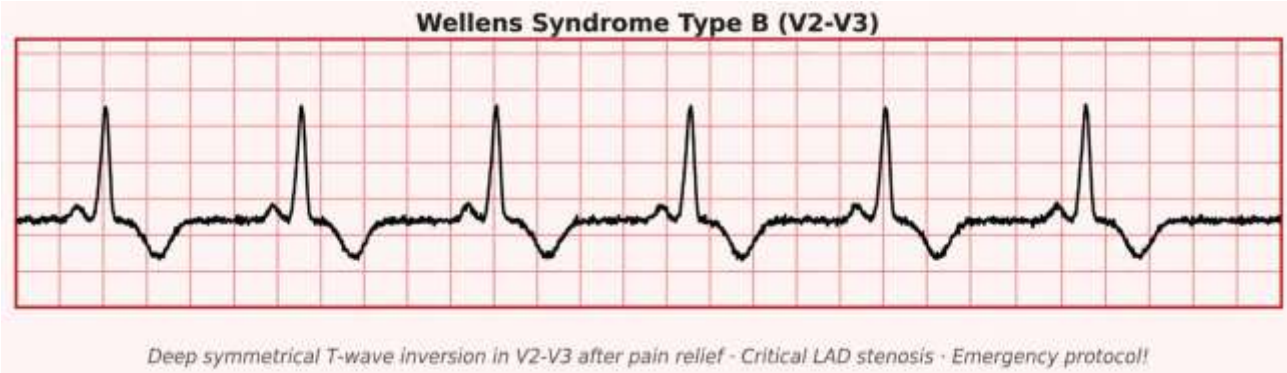

Fig. 2.3 – Wellens' syndrome type B: deep symmetrical T-wave inversion in V2–V3 – critical LAD stenosis!

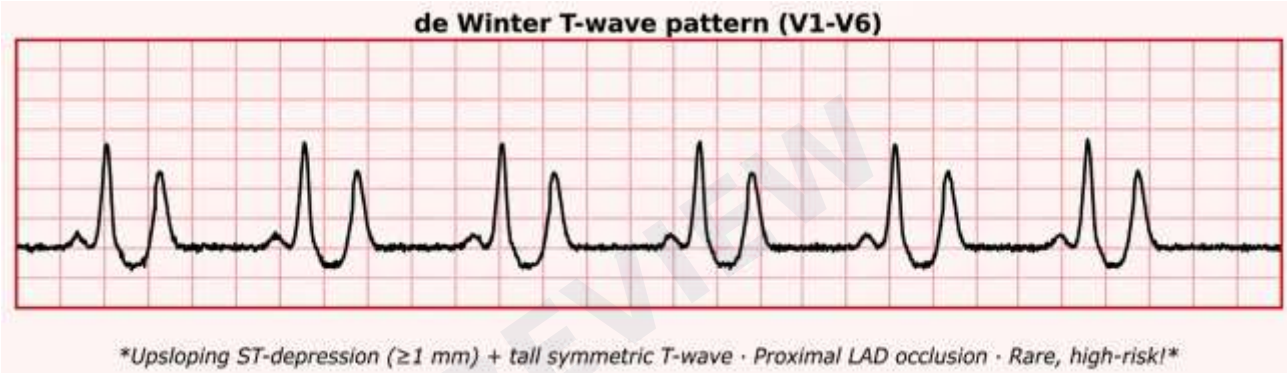

Fig. 2.4 – de Winter pattern: ascending ST-segment depression + tall T-waves – proximal LAD occlusion!

- Wellens Type A: biphasic T-waves in V2–V3 following resolution of pain
- Wellens Type B: deep symmetrical T-wave inversions in V2–V3
- de Winter pattern: ascending ST-segment depression + tall symmetrical T-waves in V1–V6 = proximal LAD occlusion!
- aVR ST elevation ≥1 mm = main trunk or proximal LAD

Red Flag

Wellens and de Winter patients often present with few symptoms and a negative troponin result – yet they require immediate emergency management. A heart attack has occurred or is imminent!

2.4 STEMI mimics

Condition	ECG differences from STEMI	Clinical presentation
Pericarditis	Diffuse (concave/saddle-shaped), PR depression, no reciprocal changes	Respiration-dependent, friction rub, young
Early repolarisation	J-point notch in V2–V5, stable, no dynamic changes	Asymptomatic, young man
Left bundle branch block	Discordant ST/T changes → check for Sgarbossa's sign!	QRS ≥120 ms, wide
LVH strain	Lateral asymmetry, high stresses, no acute dynamics	Hypertension, LVH
Brugada type 1	V1–V2 coved, terminal negative T wave	Syncope, family history
Pulmonary embolism	Sinus tachycardia, S1Q3T3, T-wave negative in V1–V4	Dyspnoea, D-dimer, clinical presentation

⚠ **Clinical error**

In cases of LSB + chest pain, ALWAYS apply the Sgarbossa criteria. Automated ECG interpretation regularly fails to recognise ACS behind LSB.

3. Conduction disturbances and bundle branch block

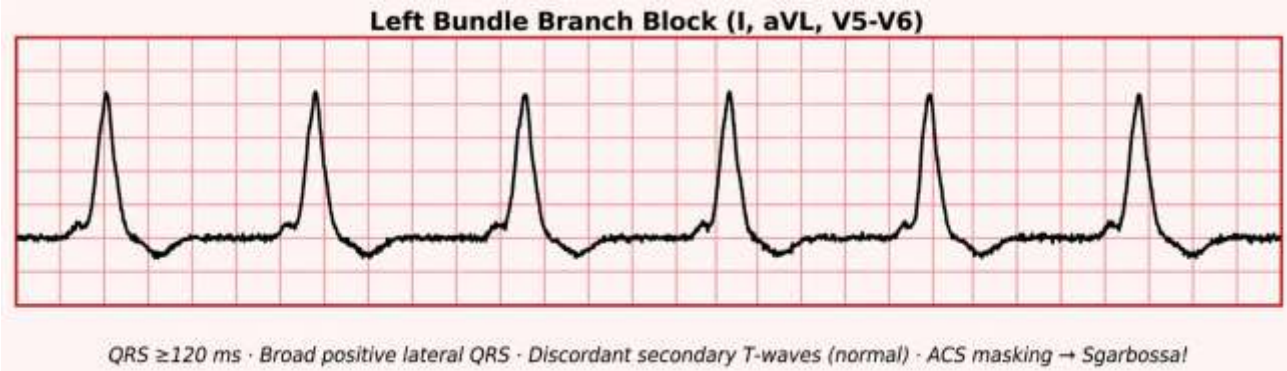

Fig. 3.1 – Left bundle branch block (LBBB): Wide, positive QRS complex in the lateral leads, discordant T-waves

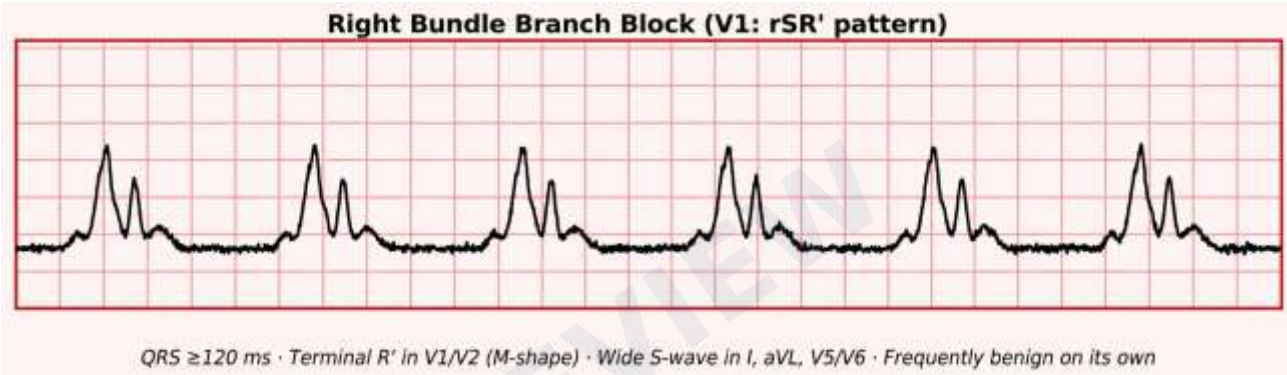

Fig. 3.2 – Right bundle branch block (RBBB): Terminal R’ in V1 (rSR’ pattern/M-wave)

3.1 Right bundle branch block (RBBB)

QRS ≥120 ms. Delayed conduction in the right ventricle.

- Terminal R’ in V1/V2 (rSR’ pattern or M-wave)
- Wide, deep S wave in I, aVL, V5/V6
- Secondary T-wave inversion in V1/V2 = normal and expected

Key points

Isolated RSB is often of no clinical significance. New onset + syncope / dyspnoea / suspected ACS → emergency pathway.

3.2 Left bundle branch block (LBBB)

- Wide, positive QRS in leads I, aVL, V5/V6 (BP’ or monophasic)
- No Q waves in leads I, aVL, V5/V6
- Discordant ST/T changes (expected = secondary)
- Poor or absent R-wave progression in V1–V4

⚠ Clinical error

LSB completely masks signs of ACS! In cases of LSB + chest pain, always check for Sgarbossa criteria and compare with a previous ECG.

3.3 Sgarbossa criteria

Criterion	Definition	Clinical significance
Concordant ST elevation	≥1 mm in the same direction as the QRS	Highly specific for occlusion
Concordant ST depression	≥1 mm in V1–V3	Specific for anterior ischaemia
Discordant ST elevation	≥25% of the S-wave (Smith modification)	Disproportionately high = suspicious

3.4 Fascicular blocks and high-risk patterns

Block	ECG criteria	Clinical significance
LAFB	Left axis deviation −45° to −90°, qR in I/aVL, rS in II/III/aVF, QRS <120 ms	Common, often isolated and benign
LPHB	Right axis >+90°, differential diagnosis (rule out RVH/PE first)	Rare, usually structural
RSB + LAFB	Bifascicular block, wide QRS + left axis	Syncope → electrophysiological investigation!

Red flag

Syncope + bifascicular block = urgent electrophysiological assessment. Risk of intermittent third-degree AV block.

4. Arrhythmias on the resting ECG

4.1 Systematic rhythm check (20 seconds)

Question	Findings	Consequence
Pulse/circulation present?	No / unstable	Call 112 immediately / CPR
Narrow QRS (<120 ms) or wide?	Wide	Presumption of validity until proven otherwise
Regular or irregular?	Irregular + narrow	AFib? Variable flutter?
P-waves detectable?	Absent	AFib likely
P-QRS relationship?	AV dissociation	Third-degree AV block or VT

4.2 Atrial fibrillation (AFib)

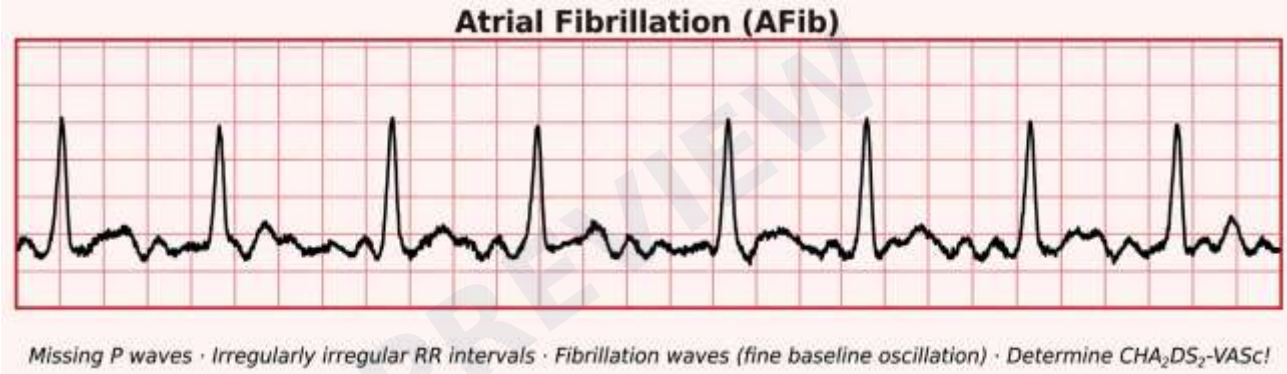

Fig. 4.1 – Atrial fibrillation: Absent P-waves, irregular-irregular BP intervals, fibrillation waves

- Absence of consistent P waves, irregular-irregular BP intervals
- Confirmation: check leads II and V1, analyse long strips
- Stroke risk: determine CHA_2DS_2-VA score
- Anticoagulation: if score ≥2 (♂) / ≥3 (♀), start DOACs

Mnemonic

Atrial flutter with 2:1 conduction → ventricular rate ~150/min – may present like sinus tachycardia. Look for flutter waves (sawtooth) in leads V1 and II!

4.3 Ventricular tachycardia (VT)

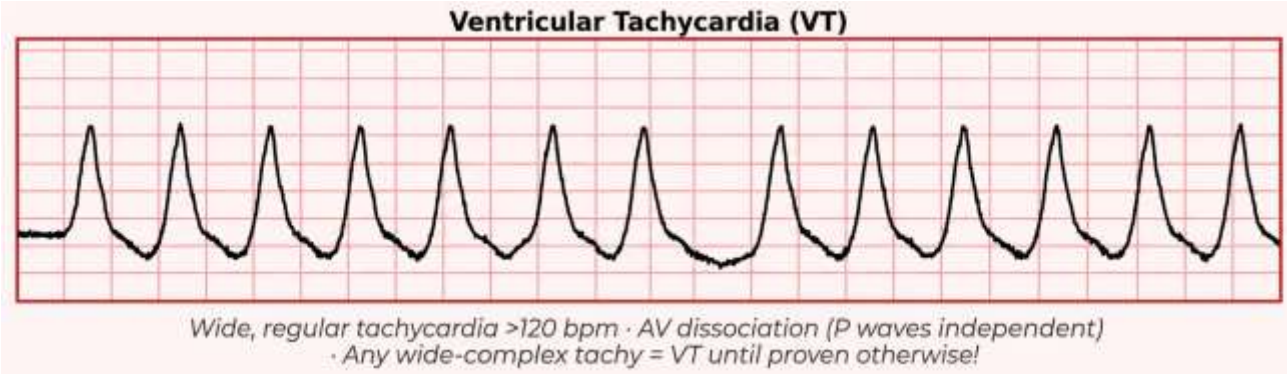

Fig. 4.2 – Ventricular tachycardia: wide, regular tachycardia ~160/min, AV dissociation

Principle: Any wide tachycardia = VT until proven otherwise.

- Indications of VT: AV dissociation, capture beats, fusion beats
- Positive or negative concordance between V1 and V6
- Morphology does not fit the RSB/LSB pattern → VT more likely

> ⚠ **Clinical errors**
>
> *Verapamil in cases of wide-complex tachycardia of unclear aetiology is potentially fatal. Do not administer AV node blockers without a confirmed diagnosis of SVT.*

4.4 AV blocks

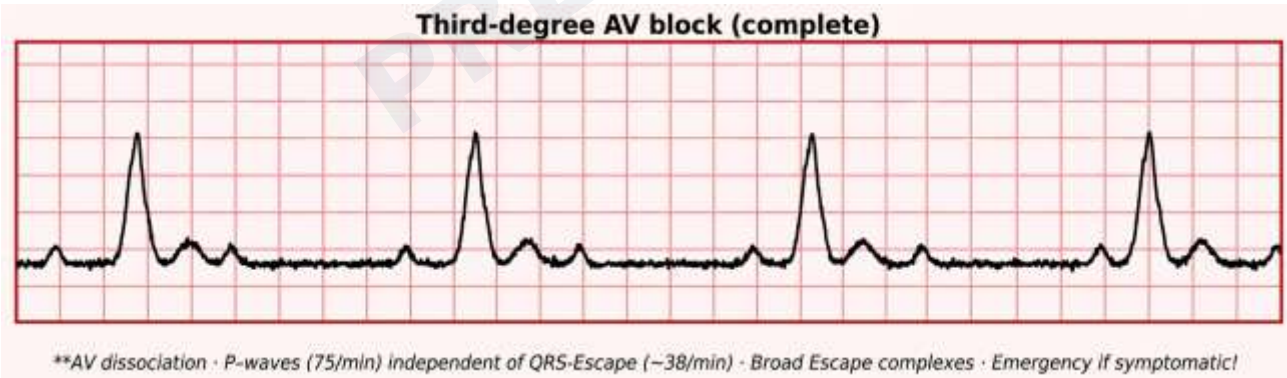

Fig. 4.3 – AV block III: P waves (75/min) and QRS escape complexes (~38/min) completely independent of one another

Type	ECG pattern	Urgency
First-degree AV block	PQ >200 ms, constant, every P wave is conducted	Course and context
Wenckebach (Mobitz I)	Progressive PQ prolongation, followed by loss of conduction	Depends on symptoms
Mobitz II	Constant PQ interval, sudden QRS drop-out without warning	Urgent!

Type	ECG pattern	Urgency
2:1 block	Every second P wave is conducted; type cannot be definitively determined	Risk context
AV block III	AV dissociation, independent P-wave and QRS frequencies	Acute, often a medical emergency

Red flag

Syncope + bifascicular block / Mobitz II / third-degree AV block = urgent to immediate cardiological assessment or call 999 (or 112).

5. QT interval, electrolytes and medication

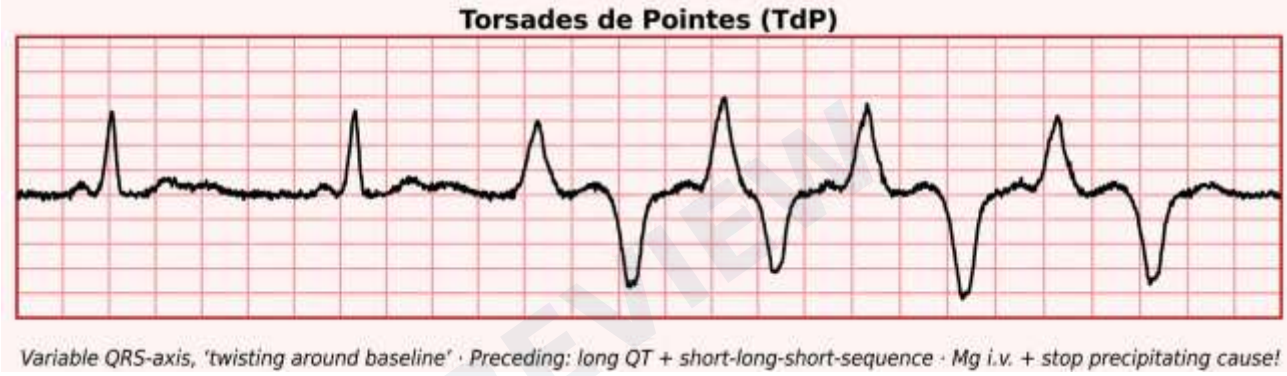

Fig. 5.1 – Torsade de Pointes: Varying QRS axis against a background of a prolonged QT

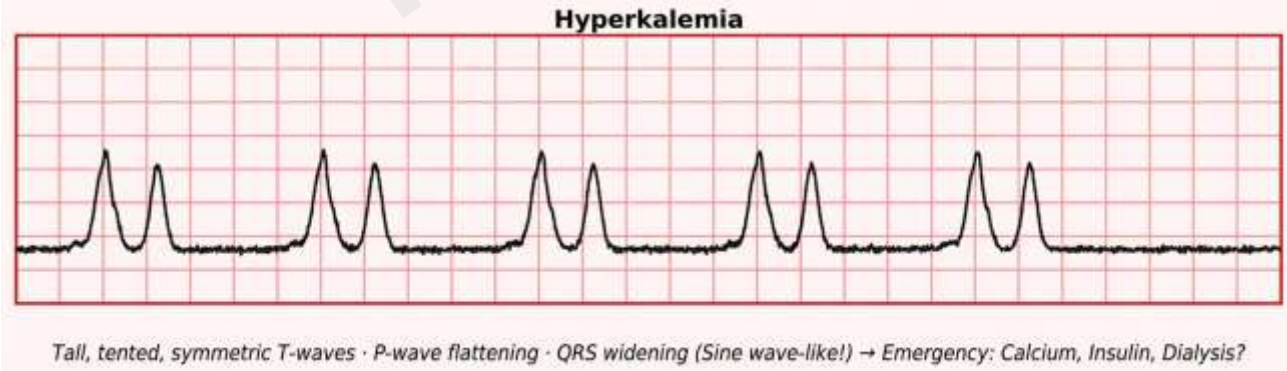

Fig. 5.2 – Hyperkalaemia: tall, pointed tent-shaped T-waves, flattened P-waves, incipient QRS widening

5.1 Correctly measuring and interpreting the QTc

- Bazett: QTcB = QT / √BP (overestimates in tachycardia)
- Fridericia: QTcF = QT / ³√BP (more accurate at extreme heart rates)
- Abnormal: ♂ ≥450 ms, ♀ ≥460 ms; critical ≥500 ms or increase ≥60 ms
- Measurement: Lead II or V5; tangent method for flat T waves

5.2 Electrolyte ECG patterns

Electrolyte imbalance	Typical ECG signs	Critical sign
Hyperkalaemia	High, pointed T wave; flattened P wave; prolonged PQ interval	QRS widening + sinus wave → medical emergency
Hypokalaemia	Flattened T-wave; ST depression; prominent U-wave	U > T + VES/TdP
Hypocalcaemia	ST prolongation → long QTc	QTc >500 ms + symptoms
Hypercalcaemia	Short ST segment → short QTc	AV block, bradycardia
Hypomagnesaemia	No specific pattern; risk of TdP	Combination with hypokalaemia

5.3 QT-critical medicines in GP practice

Group	Examples	Typical pitfall
Antiarrhythmics	Sotalol, quinidine, amiodarone	QT prolongation + bradycardia; amiodarone rarely causes TdP
Macrolides	Clarithromycin, erythromycin	Infection + electrolyte loss + interaction
Fluoroquinolones	Moxifloxacin, levofloxacin	Renal insufficiency + combinations
Antidepressants	Citalopram, escitalopram, TZA	Age, dose, electrolytes
Antipsychotics	Haloperidol, quetiapine	Dose, intravenous administration, polypharmacy
Anti-emetics	Ondansetron, domperidone	Vomiting = concurrent hypokalaemia!

Clinical Pearl

Before starting a QT-relevant medication: baseline ECG + K/Mg/Ca + renal function + medication list. www.crediblemeds.org provides the current risk level.

6. Long-term ECG, VES and SVES

6.1 Methods and indications

Method	Duration	Suitable for	Limit
24–48-hour Holter	1–2 days	Daily symptoms, burden assessment	Rare events missed
Patch/long-term	3–14 days	Several times a week, AFib	Fewer leads
Event recorder	Weeks	Less frequent, longer-lasting symptoms	Syncope may be missed
Implantable loop	Months–years	Very rare syncope, unexplained	Invasive, cardiological indication

Key point

A negative long-term ECG without typical symptoms simply means: 'Nothing documented during this period.' It does not rule out a rhythm disturbance.

6.2 Evaluation scheme: Q-G-F-T-B-S-V-S-K

- 1. Quality: recording duration, evaluable portion, number of channels
- 2. Baseline rhythm: Sinus, AFib, pacemaker?
- 3. Frequency profile: minimum, mean, maximum + context (exercise, sleep)
- 4. Day-night profile: physiological variation or rigidity?
- 5. Bradycardia: pauses, AV block, episodes of bradycardia
- 6. Supraventricular: SVES, bursts, SVT, AFib/flutter
- 7. Ventricular: VES, morphology, couplets, triplets, NSVT
- 8. Symptom correlation: diary vs. rhythm
- 9. Implications: Immediate / today / in the near future / long-term

6.3 Clinically classifying VES and SVES

Findings	Generally reassuring	Requires further investigation
VES	Isolated, monomorphic, asymptomatic, no structural heart disease	Frequent >1000/24h, polymorphic, couplets/NSVT, exercise-induced
SVES	Isolated, no structural heart disease	Multiple bursts, SVT, AFib trigger
Pauses	Sleep bradycardia <2 s, sinus arrhythmia	Pauses >3 s whilst awake, AV block, blocked SVES

Red flag

VES + syncope + structural heart disease = urgent electrophysiological assessment. VES burden (e.g. 10%) on its own, without context, is not an indication for treatment.

6.4 PMS documentation module: Long-term ECG

Long-term ECG [device], recording [X] hours, technically evaluable [Y]%. Basic rhythm: [sinus rhythm/AFib]. Frequency: Min. [__]/min, Mean [__]/min, Max. [__]/min. Pauses: [none / longest __s]. SVES [__], VES [__], [no bursts / NSVT present]. Symptom correlation: [present / no symptoms documented]. Conclusion: [__].

7. Artifacts and technical misclassification

Artifact	ECG trace	Pseudo-diagnosis	Remedy
Muscle tremor	High-frequency irregular baseline in the extremities	Pseudo-AFib	Warm up, relax, proximal electrodes
Motion artefact	Large baseline jumps, no QRS pattern	Pseudo-VT	Stop movement, re-establish contact
Baseline drift	Slow upward/downward drift, breath-dependent	Pseudo-ST elevation	Electrodes, breathe calmly
50 Hz mains	Smoothly jagged baseline, regular	ST disturbance	Check electrode contact and earthing
Poor contact	Flat line, intermittent channel	Pseudopause, asystole	Clean the skin, fit a new electrode
Arm reversal	Lead I negative, aVR positive	Misdiagnosis of heart attack	Reapply electrodes
V1/V2 too high	RSB-like, suspected Brugada syndrome	Pseudo-Brugada	Standard position 4. ICR exact

⚠ Clinical errors

'Pseudo-AFib due to tremor' and 'pseudo-VT due to movement' are the two most common sources of error. Always check the raw strip before initiating treatment.

SOP: ECG quality check prior to interpretation

1. Are all 12 leads present and complete?
2. Paper speed 25 mm/s and calibration 10 mm/mV confirmed?
3. Is the baseline stable (no drift >0.5 mm)?
4. Are there any artefacts? → Identify and eliminate the cause
5. Possible electrode reversal? (Lead I negative?)
6. If in doubt: repeat the test under better conditions

8. Red flags and the traffic light urgency system

8.1 The four-colour urgency system

Level	Findings / Clinical presentation	Action
🔴 **RED**	Loss of consciousness/no pulse / STEMI + clinical presentation / Unstable tachy-/bradyarrhythmia / Persistent VT / Third-degree AV block with reduced perfusion / TdP or polymorphic VT / Hyperkalaemia + ECG / Pre-excited AFib	Call 112 immediately. Be prepared to perform CPR. Monitor the patient. No delay in diagnosis.
🟠 **ORANGE**	New suspected STEMI without instability / Wide-complex tachycardia following spontaneous termination / Syncope + cardiac warning signs / Mobitz II / NSVT + structural heart disease / QTc ≥500 ms + symptoms	Direct referral to cardiology/hospital. Transport method based on risk. No self-drive.
🟡 **YELLOW**	New asymptomatic RSB/LAFB / Initial diagnosis of stable AFib / Frequent VES without acute symptoms / Stable QTc prolongation / Possible asymptomatic Brugada/WPW	Elective cardiology appointment in 1–4 weeks. Discuss discharge criteria.
🟢 **GREEN**	Known stable conduction disorder unchanged / Sinus bradycardia in athletes / Isolated monomorphic VES without risk / Clear sinus arrhythmia / Explained artefact	Document management plan. Safety net discussed.

8.2 Situations requiring immediate action

Findings	Risk	Immediate action
STEMI + clinical presentation	Acute MI	Call 112, take aspirin, pre-register with PCI centre
New LSB + chest pain	Masked ACS	Sgarbossa test → positive: 112
Wide-complex regular tachycardia	VT	Emergency protocol, no verapamil
Third-degree AV block + bradycardia	Circulatory failure	112, atropine, pacemaker on standby
TdP/VF	Cardiac arrest	CPR + defibrillation
Pre-excited AFib	VF caused by AV node blockers	112, no AV node blockers

☑ Clinical Pearl

The 30-second stability assessment takes priority: consciousness → breathing → pulse → BP → chest pain. Only once the patient is stable: detailed ECG interpretation.

9. Pacemaker ECG and device findings

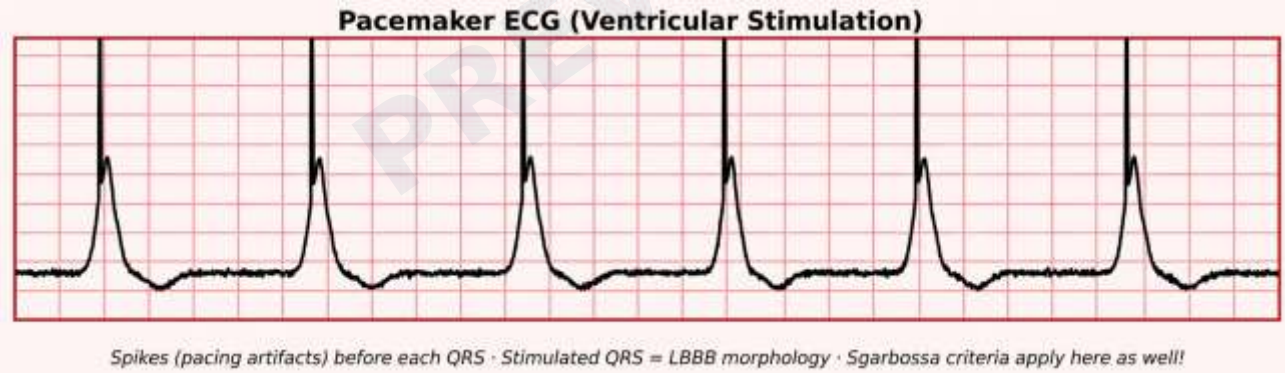

Fig. 9.1 – Pacemaker ECG: pacing spike before each QRS complex, LSB-like morphology

9.1 Four key questions regarding pacemaker ECGs

- 1. Are pacing spikes visible? (Digital ECGs sometimes suppress spikes!)
- 2. Are the atria, ventricles or both being stimulated?
- 3. Is each spike followed by depolarisation (capture)?
- 4. Does the electrical activity correspond to the clinical condition and pulse?

Problem	ECG trace	Possible cause	Urgency
Capture loss	Spike without P/QRS	Probe fault, battery, hyperkalaemia	Acute in the case of bradycardia
Undersensing	Unnecessary spikes within the patient's own QRS/T	Lead fault, fibrosis	Device monitoring
Oversensing	Necessary pacing inhibited → Pause	T-wave sensing, muscle potentials	Bradycardia possible

Red flag

Pacemaker emergency: syncope + spike without capture + long pauses + wide tachycardia in a device patient → immediate cardiological device interrogation.

Mnemonic

Pacemaker QRS = LSB morphology → Sgarbossa's rule applies here too! Automatic ECG analysis does not reliably detect stimulation spikes.

10. Specific ECG patterns in GP practice

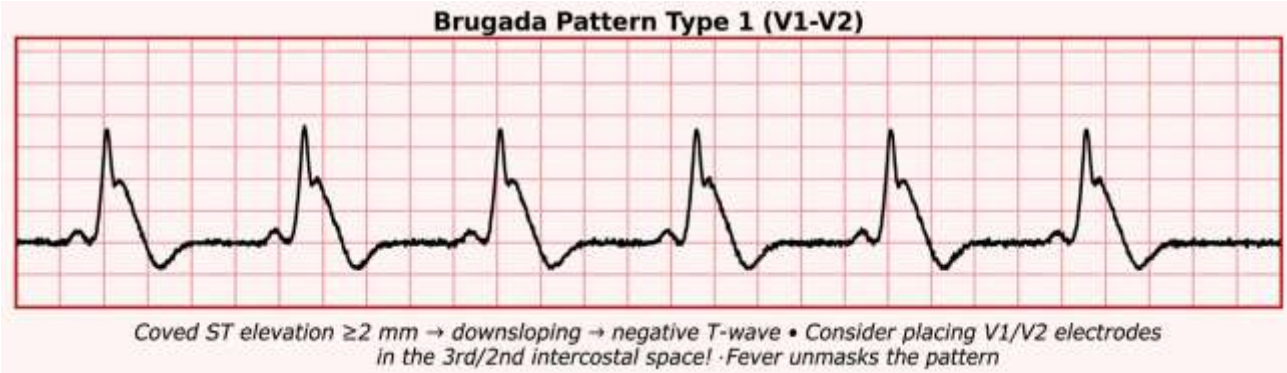

Fig. 10.1 – Type 1 Brugada pattern: Coved ST-segment elevation ≥2 mm in V1/V2, descending, terminally negative T-wave

10.1 Brugada pattern

Type 1 Brugada: Coved ST-segment elevation in V1 (≥2 mm), descending, followed by a negative T-wave. Diagnosis = ECG pattern + clinical context.

Aspect	Brugada Type 1	Common mimic
ECG	Coved, arched, >2 mm, then negative T	Wedge-shaped or saddle pattern
Electrodes	Standard or higher (V1/V2 in 3rd/2nd ICR)	Placing V1 too high mimics Brugada syndrome!
Triggers	Fever increases ECG expression	Ischaemia, electrolytes, medication
Red flags	Syncope + Type 1 + family history	No red flag without clinical symptoms

Red flag

Fever + suspected Brugada syndrome type 1 = urgent investigation. Fever can unmask ECG patterns and trigger ventricular fibrillation.

10.2 LVH and strain

- Sokolow-Lyon: S(V1) + R(V5 or V6) ≥35 mm
- Cornell: R(aVL) + S(V3) >28 mm (♂) / >20 mm (♀)
- LVH strain: descending ST depression + asymmetric T-wave inversion laterally

Mnemonic

LVH strain can resemble NSTEMI. New lateral ST-segment depression: compare with previous ECG + clinical presentation. ACS and LVH strain can occur simultaneously!

10.3 Pericarditis vs. STEMI

Characteristic	Pericarditis	STEMI
ST-segment distribution	Diffuse (>2 territories), concave/saddle-shaped	Territorial, convex (tombstone)
Reciprocal depression	Absent (except aVR/V1)	Present in opposite leads
PR depression	Characteristic	Absent
Clinical presentation	Respiration-dependent, rubbing, young	Typical ischaemic pain

10.4 Pulmonary embolism (PE)

The ECG in PE is often normal or non-specific. Signs: sinus tachycardia (most common!), new RSB, S1Q3T3 pattern, T-wave inversions in V1–V4.

⚠ Clinical errors

A normal ECG does not rule out severe PE. If PE is suspected: D-dimer + Wells score + CT angiography. S1Q3T3 alone is not a diagnosis.

11. Documentation and patient communication

11.1 PMS documentation modules

Normal findings: 12-lead ECG technically interpretable. Sinus rhythm, rate approx. __/min, PQ __ ms, QRS __ ms, QTc __ ms. Normal lead configuration. No ST/T changes, no conduction disturbance, no signs of hypertrophy. Findings unremarkable compared with the previous ECG dated __.

High risk: ST elevation __ mm in [lead(s)], clinically consistent with STEMI. Emergency protocol initiated; 112 alerted at __. Aspirin 300 mg administered. PCI centre [Name] notified in advance.

AFib: Irregular-irregular ventricular rhythm, absence of consistent P-waves, consistent with AFib. Ventricular rate approx. __/min. Symptoms since [__], onset [known/unclear]. Anticoagulation: [in place / initiated / discussed]. Other: [__].

11.2 Automatic device readings: Handling

Automatic report	Problem	Correct action
'Myocardial infarction cannot be ruled out'	Over-sensitive; clinical presentation and previous ECG missing	Check raw ECG trace + clinical presentation + previous ECG
'Left bundle branch block'	Often fails to recognise pacemaker QRS	Look for spikes; distinguish between left bundle branch block and pacemaker
'Atrial fibrillation'	False positive in cases of tremor, SVES	Check the raw trace; auscultate the pulse
'Prolonged QTc'	Wide QRS → QTc is overestimated	Note QRS width; JTc if necessary
'Normal'	Fails to detect de Winter, Wellens, or posterior MI	Do not adopt blindly – assess for yourself!

SOP: Communicate discharge criteria

1. Explain the findings: ‘Your ECG shows [finding]. This means for you...’
2. State the return-to-hospital criteria: ‘Come in straight away or call 999 (or 112) if...’
3. Specific symptoms: new or worsening chest pain, shortness of breath, fainting, palpitations
4. Document the follow-up appointment, including the date and purpose
5. Check understanding: ‘What will you do if the pain returns?’
6. PMS: ‘Safety net discussed, patient understood: yes’

Online resources for Germany, Austria and Switzerland

Guidelines and professional societies

- **ESC – Cardiology guidelines (Germany):** https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines
- **AWMF – S3 guideline on chest pain (D):** https://www.awmf.org/leitlinien/detail/ll/053-023.html
- **DEGAM – General Practitioner Guidelines (D):** https://www.degam.de/leitlinien.html
- **ÖGK – Austrian Society of Cardiology (A):** https://www.oekg.at/fachinfo/leitlinien/
- **SGK – Swiss Society of Cardiology (CH):** https://www.sgk.ch/de/fachleute/leitlinien-empfehlungen/

ECG learning platforms and tools

- **ECG Library – Life in the Fast Lane (EN):** https://litfl.com/ecg-library/
- **EKG.academy – Interactive ECG learning platform (German):** https://www.ekg.academy/
- **QTc calculator – MDCalc (EN):** https://www.mdcalc.com/calc/3164/corrected-qt-interval-qtc
- **HEART Score Calculator (EN):** https://www.heartscore.nl/
- **Crediblemeds – QT drug list (EN):** https://crediblemeds.org/

Continuing professional development and CME

- **DGK e.Akademie – ECG training (German):** https://eakademie.dgk.org/
- **University of Münster Online ECG Course (German):** https://ekg-kurs.uni-muenster.de/
- **Amboss – ECG basics (German):** https://www.amboss.com/de/wissen/ekg-grundlagen/
- **UpToDate – ECG Clinical Topics (EN):** https://www.uptodate.com/contents/search?search=electrocardiogram

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A2 – Tailored treatment of hypertension in GP practice: when and which investigations to carry out to determine the cause

Learning objectives of this chapter

- To apply the four-stage treatment model (stages 1–4) – from monotherapy to resistant hypertension – with confidence.
- Know when basic and advanced diagnostic tests are indicated, and distinguish between pseudo-resistance and true treatment resistance.
- Systematically consider secondary hypertension (CHARLES algorithm) and initiate initial diagnostic investigations in GP practice.
- Safely dose diuretics and mineralocorticoid receptor antagonists, recognise typical electrolyte imbalances and monitor them closely.
- Recognise and avoid the risks of drug interactions in cases of polypharmacy (Triple Whammy, CKD).
- Apply specific, guideline-based stepwise treatment regimens for CKD, frail patients and metabolic syndrome.
- Use strategies to promote adherence (fixed-dose combinations, the KISS principle) in a targeted manner during consultations.

✔ Note: The key decision for GPs

- Before any escalation of treatment, follow this sequence: check measurement quality → check adherence → consider secondary hypertension → only then escalate.
- From stage 2 onwards, the fixed-dose combination (single-pill) is the standard, not the exception – it is the most effective tool for improving adherence.
- Potassium and eGFR are the safety parameters for any diuretic/MRA treatment: before starting, 1–2 weeks after each dose adjustment, and thereafter every 3–6 months, depending on risk.

Quick overview: Who to target, when to act?

Situation	GP-related action
Newly diagnosed hypertension	Complete basic diagnostic work-up (lab tests, urine analysis, ECG, HBPM/ABPM), risk stratification, initiate Stage 1 treatment.
BP not within target range despite Stage 2 (dual therapy)	Rule out pseudo-resistance, then proceed to Stage 3 (triple therapy including a diuretic).
BP not within target range despite optimised triple therapy	Resistant hypertension: Re-assess adherence and measurement quality, consider MRA (Stage 4), actively investigate secondary causes.
Hypertension + hypokalaemia (including borderline levels)	Determine the aldosterone-renin ratio (ARQ) – consider primary hyperaldosteronism.
Increase in creatinine >20% following initiation of RAS blockers	Suspected renovascular hypertension – renal ultrasound; consider duplex scanning or angiography if necessary.
New initiation/increase in spironolactone/eplerenone	Check potassium and eGFR before starting treatment and again after 1–2 weeks.
New prescription of NSAIDs in the presence of RAAS blockade + diuretic	'Triple whammy' – risk of acute kidney failure; consider an alternative or monitor closely.

Case vignette – The initial case

Mr M., aged 65, type 2 diabetes mellitus, left ventricular hypertrophy (LVH) on echocardiogram. Despite treatment with an ACE inhibitor, a calcium antagonist and a thiazide-type diuretic at the maximum tolerated dose, his blood pressure measured in the GP's surgery is 158/96 mmHg; the weekly home blood pressure monitoring (HBPM) confirms 152/92 mmHg. Before diagnosing 'resistant hypertension', the quality of blood pressure measurements, adherence (collection of prescriptions, pill burden) and medication history (regular use of NSAIDs for knee pain) are assessed – the use of NSAIDs is identified and discontinued. As the hypertension remains uncontrolled thereafter, spironolactone 25 mg 1-0-0 is added; prior to commencement, potassium is 4.3 mmol/l and eGFR is 68 ml/min/1.73 m². Follow-up after 1 and 4 weeks shows stable values; blood pressure falls to 138/86 mmHg. In the absence of a response, the aldosterone-renin ratio would also have needed to be determined to ensure that primary hyperaldosteronism was not overlooked.

1. Stages of hypertension, risk stratification and blood pressure targets

Arterial hypertension remains the most important modifiable risk factor for cardiovascular disease and premature mortality. In GP practice, most patients' blood pressure can be controlled using standard diagnostic and therapeutic approaches; however, in cases of difficult-to-control blood pressure, atypical clinical courses or possible secondary hypertension, structured, differentiated approaches are crucial to ensure that opportunities for improving prognosis are not missed [1,3].

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	≥ 140	and < 90

In addition to the classification, the 2023 ESH guidelines define three stages that reflect the prognostically relevant hypertension-mediated organ damage (HMOD) and overall cardiovascular risk: Stage 1 (uncomplicated hypertension without HMOD or CVD, CKD 1–2), Stage 2 (existing HMOD, CKD 3 or diabetes) and Stage 3 (cardiovascular end-organ damage or CKD ≥ 4) [3]. SCORE2 or SCORE2-surgery (for older patients) is recommended for risk stratification [3,6].

Blood pressure target values by age (ESH 2023)

Age group	Systolic target	Diastolic target
18–64 years	< 130 mmHg	< 80 mmHg
65–79 years	< 140 mmHg (< 130 mmHg if well tolerated)	< 80 mmHg
≥ 80 years	140–150 mmHg (< 140 mmHg if well tolerated)	< 80 mmHg
Frail patients	Individual target based on tolerance	individual target
For all patients	do not aim for < 120 mmHg	–

The NVL Hypertension 2023 guidelines set a mandatory target of a clinic blood pressure < 140/90 mmHg for virtually all patients and recommend, where tolerated, values around 130/80 mmHg [1]. Drug therapy is initiated for readings > 140/90 mmHg, and from as low as 130/80 mmHg in cases of established cardiovascular damage [3,6].

2. Staged model of diagnosis and treatment (stages 1–4)

Stage	Clinical situation	Preferred approach	Typical pitfalls
Stage 1	Grade 1 hypertension, low risk, or very elderly/frail patients	Lifestyle intervention plus, if necessary, monotherapy (ACE inhibitor/ARB or CCB)	Do not allow uncontrolled blood pressure to persist for too long – the target remains a mandatory target.
Stage 2	Most patients requiring treatment	Early dual therapy, preferably as a fixed-dose combination: ACE inhibitor/ARB + CCB or + thiazide-like diuretic	Fixed-dose combinations significantly improve adherence and should be the norm, not the exception.
Stage 3	Inadequate control despite optimised dual therapy	Triple therapy: ACE inhibitor/ARB + CCB + thiazide or thiazide-like diuretic (preferably chlortalidone or indapamide)	Before classifying the patient as ‘difficult to control’, rule out pseudo-resistance and measurement errors.
Stage 4	Resistant hypertension despite optimised triple therapy at an adequate dose and confirmed adherence	Add an MRA (spironolactone preferred, eplerenone as an alternative); followed by individualised reserve classes	Monitor potassium/eGFR before and after initiation; exercise particular caution in cases of CKD or risk of hyperkalaemia.

A genuine non-response despite full utilisation of Stage 4 justifies referral to a hypertension centre; interventional procedures such as renal denervation may also be considered there [1,3]. In practice, the model should be consistently linked to home/24-hour BP measurements, risk stratification and assessment of HMOD.

3. Differentiated diagnosis: when and which investigations?

3.1 Basic diagnostics – mandatory and indication-specific investigations

Test	Must/May	Specific details	Practical note
Standardised clinical measurement	Must	Multiple measurements after 3–5 minutes' rest, correctly fitted cuff, arm at heart level, no talking	The number one source of error in pseudoresistance – record the average value.
Home blood pressure monitoring (HBPM)	Essential, usually as a supplement	7 days, two readings each in the morning and evening; discard Day 1	Threshold for diagnosis/monitoring: ≥ 135/85 mmHg.
24-hour blood pressure monitoring (ABDM)	Optional/mandatory if required	Daytime, night-time and 24-hour average values	Particularly useful in cases of apparently resistant hypertension and suspected white-coat or masked hypertension.
Serum laboratory tests	Must	Sodium, potassium, creatinine/eGFR, fasting glucose, HbA1c if applicable, lipid profile, uric acid	Repeat before starting treatment and whilst on diuretics/MRA.
Urinalysis	Must	Urine dipstick test, albumin-to-creatinine ratio (ACR)	ACR is still too rarely measured in clinical practice – request this early on.
12-lead ECG	Must	Rhythm, conduction disturbances, signs of left ventricular hypertrophy (LVH), signs of ischaemia	Basic diagnostic work-up for every initial diagnosis of hypertension.
Echocardiography	May be performed/if indicated	LV mass, EF, diastolic function, left atrium	Consider in cases of treatment-resistant hypertension, dyspnoea or oedema.
Renal ultrasound	May be performed if indicated	Kidney size, asymmetry, parenchyma	Consider early in cases of elevated creatinine, CKD or suspected renovascular cause.

3.2 Advanced diagnosis of hypertension-mediated organ damage (HMOD)

Advanced diagnostics are indicated in cases of manifest or suspected HMOD (proteinuria, decline in eGFR, ECG-detected LVH, clinical signs of heart failure, neurological symptoms), high or very high cardiovascular risk, or a rise in blood pressure that is difficult to control or is progressing rapidly. These investigations are frequently initiated in GP practices; the subsequent management strategy is often determined in collaboration with cardiology or nephrology [1,3].

4. Secondary hypertension: consider systematically, investigate specifically

Secondary hypertension is generally less common, but significantly more frequent in specific patient groups (resistant hypertension, hypokalaemia, hypertension + atrial fibrillation, adrenal incidentaloma) and is often underdiagnosed, particularly in primary hyperaldosteronism (PA) – prevalences of over 20% have been reported in resistant hypertension cohorts [2,7].

CHARLES – Mnemonic for secondary hypertension

- C – Conn/Cushing/catecholamines (primary hyperaldosteronism, Cushing's syndrome, pheochromocytoma)
- H – Hyperparathyroidism, hyperthyroidism/hypothyroidism
- A – Aortic isthmus stenosis
- R – Renovascular and renoparenchymal hypertension
- L – Liquorice abuse and other exogenous substances
- E – Oestrogen/the pill (oral contraception, hormone therapy)
- S – Sleep apnoea syndrome

Red flags in clinical practice	Probable cause	Initial diagnosis at the GP practice
Grade 3 hypertension (≥180/110 mmHg), particularly in patients under 60 years of age	Secondary hypertension in general	Basic laboratory tests (Na, K, creatinine/eGFR, glucose, lipids), urinalysis + ACR, ECG, medication history, HBPM/ABPM.
Treatment-resistant despite 3 active substances, including a diuretic	Pseudoresistance or secondary hypertension (CKD, renovascular, Conn's syndrome, sleep apnoea)	Check measurement quality and adherence, baseline laboratory tests + ACR, renal ultrasound.
Spontaneous/diuretic-induced hypokalaemia	Primary hyperaldosteronism (Conn's syndrome)	Na/K/creatinine, medication review, aldosterone-renin ratio (ARQ).
New-onset severe hypertension in patients aged <30–40 years	Monogenic hypertension, aortic isthmus stenosis, renal cause	BP in all 4 limbs, pulse status, baseline laboratory tests + ACR, renal ultrasound.
Rapid worsening of previously stable hypertension	Renovascular hypertension, change of medication	Medication review (NSAIDs etc.), creatinine/eGFR, Na, K, ACR, renal ultrasound.
CKD, proteinuria/haematuria + hypertension	Renoparenchymal hypertension	eGFR trend, ACR, urinalysis, renal ultrasound, early involvement of a nephrologist.

Red flags in clinical practice	Probable cause	Initial diagnosis at the GP practice
Snoring, breathing pauses, daytime sleepiness	Obstructive sleep apnoea syndrome	STOP-BANG history-taking, BMI/neck circumference, referral to a sleep laboratory if necessary.
Paroxysmal episodes, tachycardia, sweating	Pheochromocytoma/paraganglioma	Plasma-free or 24-hour urine metanephrines (usually by a specialist).
Trunk obesity, livid striae, myopathy	Cushing's syndrome	1 mg dexamethasone suppression test or cortisol measurement (endocrinology).
Weight change, tremor, intolerance to cold or heat	Hyperthyroidism/hypothyroidism	TSH screening; if abnormal, fT4/fT3.

SOP 1 – Screening and initial diagnosis for newly diagnosed hypertension

→ SOP 1: Screening/initial diagnosis

1. Carry out and document standardised practice-based measurements (multiple readings).
2. Initiate a week of home blood pressure monitoring (7 days, two measurements in the morning and evening); if findings are unclear, supplement with ambulatory blood pressure monitoring (ABDM).
3. Request baseline laboratory tests (Na, K, creatinine/eGFR, glucose, lipids), urinalysis + ACR, and a 12-lead ECG.
4. Conduct a physical examination, including BMI/waist circumference, pulse status and signs of end-organ damage.
5. Actively check for and document red flag criteria for secondary hypertension (see table).
6. Calculate overall cardiovascular risk (SCORE2/SCORE2-surgery) and determine the treatment stage.

5. Stepwise treatment in detail: drug classes and MRA

5.1 First-line drug classes

Drug class	Role	Advantages	Important side effects/notes
ACE inhibitors (e.g. ramipril, enalapril, lisinopril)	Core treatment at every stage	Nephroprotective in cases of albuminuria, diabetes, CHD, heart failure	Dry cough – switch to an ARB in this case.
ARBs/sartans (e.g. candesartan, valsartan, losartan, olmesartan)	Alternative to ACE inhibitors	Equivalent efficacy, better tolerated	Preferred in cases of ACE inhibitor intolerance.
Calcium channel blockers – dihydropyridines (amlodipine, lercanidipine, felodipine)	Form the basis of treatment at every stage	Effective in isolated systolic hypertension and in the elderly	Ankle oedema – combining with an RAS inhibitor or diuretic alleviates this.
Thiazide-type/thiazide-like diuretics (chlortalidone, indapamide)	Standard diuretics from stage 2–3	Long duration of action, recommended in guidelines, preferred over HCT	Hypokalaemia, hyponatraemia, hyperuricaemia.
Hydrochlorothiazide (HCT)	To be used with caution	Widely available in combination formulations	Associated with non-melanoma skin cancer and more adverse metabolic effects – no longer the first choice [2].
Beta-blockers (bisoprolol, metoprolol, carvedilol)	For specific indications	Drugs of choice for CHD, HFrEF, tachycardic arrhythmias, post-myocardial infarction	Not first-line monotherapy for isolated hypertension without these indications.
Loop diuretics (furosemide, torasemide)	Reserve/CKD	Effective even at low GFR	Indicated for eGFR <30 ml/min, oedema or volume overload; otherwise not standard practice.

! Clinical error: continuing to prescribe HCT as the standard diuretic

- HCT has historically been prescribed frequently in Germany, but there is documented evidence linking it to non-melanoma skin cancer and more adverse metabolic effects than chlortalidone/indapamide [2].
- Counter-strategy: when initiating treatment or switching therapy, give preference to chlortalidone/indapamide; consider bendroflumethiazide/amiloride as a potassium-sparing alternative.

5.2 MRA stage in resistant hypertension

The PATHWAY-2 study demonstrated, for the first time, the superiority of spironolactone over beta-blockers (bisoprolol) and alpha-blockers (doxazosin) as a fourth active substance in the treatment of resistant hypertension [4]. Indications: resistant hypertension under optimised triple therapy, as well as confirmed primary hyperaldosteronism, where curative treatment is not possible or pending definitive treatment [1,3].

Substance	Starting dose	Target dose	Special considerations
Spironolactone	12.5–25 mg 1-0-0	25–50 mg 1-0-0 (max. depending on tolerance)	Preferred MRA compound; gynaecomastia/libido problems possible.
Eplerenone	25 mg 1-0-0	25 mg 1-0-1 (50 mg/day)	Alternative in cases of spironolactone intolerance; used off-label for hypertension in Germany, authorised for heart failure.

6. Electrolyte imbalances associated with diuretics and MRAs

Treatment	Typical electrolyte disturbance	Risk groups	Monitoring
Thiazide-like diuretics	Hypokalaemia, hyponatraemia, hypomagnesaemia, hyperuricaemia	Older people, CKD, polypharmacy, low fluid reserve	Before starting treatment, 1–2 weeks after initiation or dose increase, then every 3–6 months.
Loop diuretics	Hypokalaemia, hypomagnesaemia, volume depletion	Heart failure, CKD, high doses, sequential nephron blockade	More frequent monitoring than with thiazides, particularly following combination with a thiazide.
MRA (spironolactone/eplerenone)	Hyperkalaemia, mild hyponatraemia, increased creatinine	eGFR <60 ml/min, advanced age, diabetes, RAS blockade, NSAID	Before starting treatment, after 1 and 4 weeks, then every 3 months (risk-adapted).
Potassium-sparing diuretics (amiloride/triamterene) + ACE/ARB	Hyperkalaemia	CKD, diabetes, older patients	Monitor closely, as with MRA.

Practical thresholds for guidance: Potassium 5.0–5.5 mmol/l → close monitoring, dietary adjustments, dose reduction if necessary. Potassium > 5.5 mmol/l or rapid rise → reduce or suspend MRA dose, review other potassium-raising medicines, contact nephrology if necessary [1,2].

SOP 3 – Acute situation: Hyperkalaemia during MRA/potassium-sparing therapy

→ SOP 3: Management of potassium > 5.5 mmol/l whilst on MRA

1. Repeat laboratory tests (rule out haemolysis as a confounding factor) and perform an ECG (conduction disturbances, T-wave).
2. Reduce the MRA dose or discontinue treatment; identify other potassium-raising medicines (ACE inhibitors/ARBs, NSAIDs, potassium supplements, beta-blockers).
3. Discuss a low-potassium diet; check fluid and volume status.
4. If potassium > 6.0 mmol/l, ECG abnormalities or clinical symptoms: immediate hospital admission.
5. Follow-up after 3–7 days; resume MRA only once levels have normalised and at a reduced dose.

7. Pitfalls and nuances of diagnosis and treatment

7.1 Pseudoresistant hypertension

A significant proportion of cases of supposedly resistant hypertension are attributable to pseudo-resistance: measurement errors, white-coat hypertension not confirmed by home or 24-hour BP monitoring, non-adherence (common with regimens involving more than three tablets daily) and interfering substances (NSAIDs, steroids, sympathomimetics) [1,3].

Key question	What specifically should be checked?
Is the measurement correct?	Rest period, cuff size, seated position, no conversation, multiple measurements.
Is there white-coat hypertension?	HBPM or 24-hour BP monitoring for confirmation.
Is adherence sufficient?	Dosing schedule, pill burden, side effects, collection of prescriptions, any outstanding queries.
Are there any volume-related factors?	Salt intake, NSAID, CKD, heart failure, oedema.
Is secondary hypertension a likely cause?	Hypokalaemia, young age, signs of pulmonary hypertension, suspected renal artery disease (see Section 4).

7.2 Primary hyperaldosteronism: Checklist for clinical practice

Hyperaldosteronism screening checklist

- Who to consider: treatment-resistant hypertension (≥3 antihypertensives including a diuretic), hypertension + spontaneous/diuretic-induced hypokalaemia, adrenal incidentaloma, atrial fibrillation, early onset.
- Before blood sampling: correct hypokalaemia; where possible, discontinue or switch critical medication (diuretics, ACE inhibitors, ARBs, beta-blockers, NSAIDs); 'neutral' bridging medication: verapamil, amlodipine, doxazosin, urapidil, dihydralazine.
- Pre-analytical procedures: in the morning, after 5–15 minutes' rest, in a standardised position; explicitly specify aldosterone, renin and ARQ on the laboratory request form.
- Interpretation: ARQ in the grey zone → follow-up monitoring/optimisation of pre-analytical procedures; significantly elevated ARQ plus elevated aldosterone and suppressed renin → suspected primary aldosteronism (PA); arrange a confirmatory test or referral.
- Most common error: interpreting ARQ without documenting current medication, or classifying a single borderline elevated ARQ result as 'confirmed PA' without a confirmatory test.

7.3 Renovascular hypertension: imaging – yes or no?

Imaging of the renal arteries is indicated in cases of specific clinical suspicion, not routinely for every case of hypertension [1,7].

Decision step	Criterion
1. Is there a specific clinical suspicion?	Severe/treatment-resistant hypertension, sudden onset (<30 or >55 years), creatinine increase >20% following ACE inhibitor/ARB treatment, difference in kidney size, abdominal vascular murmur, recurrent pulmonary oedema.
2. Would the finding have therapeutic implications?	Only investigate with imaging if there is a realistic option for revascularisation or a planned adjustment to the antihypertensive strategy.
3. Which method should be used first?	Duplex ultrasound of the renal arteries; if the findings are unclear or there is a high degree of suspicion, CT or MR angiography; invasive angiography only if an intervention is planned.
4. Key questions to ask before referral	Is the hypertension truly refractory to treatment (adherence, dosage and combination of treatments checked)? Have other secondary causes been considered? Would the person genuinely benefit from revascularisation?

7.4 Very elderly and frail patients

For patients aged ≥80 and frail patients, wider target ranges (140–150/<80 mmHg) tend to apply, along with slow dose titration, careful monitoring of orthostatic blood pressure drops and consistent reduction of polypharmacy, focusing on a few, well-tolerated medicines. Over-treatment (<120 mmHg systolic) should be avoided [1,3].

⚠ Red flags: consider immediate referral

- Clear suspicion of a reversible secondary cause (primary hyperaldosteronism, renovascular hypertension, pheochromocytoma, aortic isthmus stenosis).
- Treatment-resistant hypertension despite optimised triple therapy/MRA therapy and pseudoresistance having been ruled out.
- Rapidly progressive deterioration in renal function or severe electrolyte imbalance.
- Hypertensive crisis/emergency with target organ damage.
- Paroxysmal hypertensive crises with tachycardia, headache, and sweating (suspected pheochromocytoma).

8. Long-term adherence and simplification of treatment regimens

Non-adherence increases significantly with a higher number of tablets and greater dosing frequency, and is one of the most common causes of apparent treatment resistance. The ESH/ESC and NVL expressly recommend the use of fixed-dose combinations (single-pill regimens) to reduce the pill burden and improve adherence and persistence [1,3].

Practice Pearl: the KISS principle

- Ideally, 1–2 doses per day, preferably in the morning; fixed routines and weekly dosing containers make taking the medication easier.
- Two to three active ingredients at low doses usually achieve most of the blood pressure reduction with fewer side effects than a single high-dose active ingredient.
- At every follow-up appointment, ask briefly: “How well does this regimen fit into your daily routine?” – this often identifies adherence issues more quickly than checking the figures alone.

Barrier	Practical solution
Too many tablets	Switch to a fixed-dose combination (single pill), combine dosing times.
No noticeable symptoms (“silent disease”)	Brief visualisation of risk (e.g. 5-year risk at current vs. target blood pressure).
Side effects	Address these actively; switch to a different class of medication rather than ‘sweeping them under the carpet’.
Forgetting	Weekly pill box/dispenser, reminder app, firmly integrated into daily routine.
Cost	Check for generic alternatives with the same active ingredient; discuss with the chemist.

SOP 4 – Recall and follow-up

→ SOP 4: Recall/Follow-up

1. After each initiation or escalation of treatment: follow-up appointment after 2–4 weeks with assessment of the HBPM.
2. After any change in diuretic or MRA dosage: laboratory tests (Na, K, creatinine/eGFR) after 1–2 weeks.
3. Stable patients not taking diuretics or MRAs: follow-up every 3–6 months (BP, adherence, side effects).
4. Stable patients on MRA or diuretics: laboratory tests every 3–6 months, tailored to risk; more frequent monitoring in cases of CKD or diabetes.
5. Annually: Re-evaluation of the overall strategy; re-assessment for secondary causes in the event of newly emerging red flags.

9. Example regimens for stages 1–4 for three patient groups

The following regimens are deliberately kept typical and are based on NVL 2023, ESH 2023 and the KBV Medication Catalogue; they must be adapted to the product information, renal function and individual circumstances [1,2,3].

A) CKD (e.g. CKD G3a, albuminuria, diabetes)

Stage	Treatment	Monitoring
1	Ramipril 2.5 mg 1-0-0 → 5–10 mg 1-0-0 (Alternative: Candesartan 8 mg → 16–32 mg 1-0-0)	Creatinine/eGFR, potassium before starting treatment, after 1–2 and 4 weeks, then every 3 months.
2	Ramipril + amlodipine (separate or fixed-dose combination), e.g. 5/5 mg → 10/10 mg 1-0-0; CCBs preferred over thiazides in advanced CKD	As for Stage 1, plus BP control via HBPM.
3	eGFR ≥30 ml/min: + indapamide (e.g. 1.25–2.5 mg); eGFR <30 ml/min: switch to a loop diuretic (furosemide 20–40 mg 1-0-0 or 1-0-1)	Na/K/creatinine, fluid status, weight, HBPM/ABPM.
4	Spironolactone 12.5–25 mg once daily → 25–50 mg (only if eGFR ≥45 ml/min, K ≤4.5 mmol/l); if eGFR <45 ml/min, prefer optimisation of chlorthalidone + beta-blocker/alpha-blocker rather than MRA	Potassium/creatinine after 1 and 4 weeks, then every 4–12 weeks.

B) Frail older patients (aged ≥80 years, frailty, risk of falls)

Stage	Treatment	Monitoring
1	Amlodipine 2.5–5 mg 1-0-0 (alternative: candesartan 4–8 mg 1-0-0), target range preferably 130–150/70–80 mmHg	BP whilst sitting and standing, symptoms (dizziness, tiredness).
2	Candesartan 4 mg + amlodipine 2.5–5 mg 1-0-0, low-dose; use thiazides with caution (risk of falls/dehydration)	Orthostatic BP, falls.
3	Candesartan 8–16 mg + amlodipine 5 mg + indapamide 1.25 mg 1-0-0, only in cases of significantly elevated BP and good tolerance	Na/K/creatinine, orthostatic BP, falls.
4	Only selectively: spironolactone 12.5 mg 1-0-0 with caution (if renal function and potassium levels are stable) or a beta-blocker if there is an additional indication	Very close clinical monitoring (dizziness, eGFR, potassium, weight).

C) Metabolic syndrome (obesity, dyslipidaemia, prediabetes)

Stage	Treatment	Monitoring
1	Candesartan 8 mg 1-0-0 → 16–32 mg (alternative: Ramipril 2.5–5 mg → 5–10 mg); metabolically neutral: Amlodipine 5–10 mg may also be added	Glucose, lipids, weight.
2	Fixed-dose combination of candesartan and amlodipine, e.g. 8/5 mg → 16/10 mg 1-0-0 (or ramipril and amlodipine 5/5 mg → 10/10 mg)	Glucose, lipids, Na/K/creatinine.
3	RAAS inhibitor + amlodipine + indapamide 1.25–2.5 mg (more favourable metabolic profile than HCT); alternatively, olmesartan/amlodipine/HCT 20/5/12.5 mg, taking into account the metabolic effects of HCT	Glucose, lipids, uric acid, Na/K/creatinine, weight.
4	Spironolactone 25 mg 1-0-0 → if necessary, 25 mg 1-0-1 (if eGFR ≥45 ml/min, K ≤4.5 mmol/l); alternative: eplerenone 25–50 mg/day	Potassium/eGFR after 1 and 4 weeks, then every 3 months; uric acid.

10. Medication review: Interactions in cases of polypharmacy and CKD

In CKD patients with polypharmacy, the focus is primarily on RAAS inhibitors, diuretics, potassium-sparing agents and NSAIDs, as these collectively affect renal function, potassium levels and fluid balance. The combination of a RAAS inhibitor, a diuretic and an NSAID ('triple whammy') significantly increases the risk of acute kidney failure, particularly in the first 30 days of combined use [5].

Do (recommended)	Don't (avoid)
Monitor eGFR, creatinine and potassium before any relevant change in treatment and regularly in patients with CKD.	Combining ACE inhibitors with sartans or sartans with renin inhibitors – no benefits in terms of clinical outcomes, but increased risk of renal side effects and hyperkalaemia.
In CKD, use RAAS blockers as first-line treatment, particularly in cases of albuminuria, with laboratory monitoring after 1–2 weeks.	"Triple whammy" without monitoring: administering RAAS blockers + diuretics + NSAIDs simultaneously and over the long term.
Adjust diuretic to GFR: thiazide if function is preserved, loop diuretic if eGFR <30 ml/min.	Routinely continue combinations containing amiloride/triamterene in cases of CKD plus RAAS blockade.
Use spironolactone for resistant hypertension only if eGFR ≥45 ml/min and potassium <4.5 mmol/l; monitor closely.	Continue spironolactone/eplerenone in patients with eGFR <45 ml/min or elevated potassium levels without strict monitoring.
Where possible, switch to fixed-dose combinations to improve adherence in cases of polypharmacy.	Avoid prescribing an unnecessarily large number of individual tablets if equivalent fixed-dose combinations are available.
Systematically enquire about concomitant medications that increase BP (NSAIDs, steroids, SNRIs, liquorice).	Do not label blood pressure as 'treatment-resistant' without having assessed NSAIDs, fluid status, adherence and secondary hypertension.

11. Specific examples of dosages and preparations (Germany)

The selection is based on the KBV Medication Catalogue and the NVL recommendations; standard generic preparations are prioritised, whilst brand names of finished medicinal products are provided for guidance only and must always be adapted to the product information and reimbursement status [1,2,8].

Active ingredient (INN)	Starting dose	Target/maximum dose	Note
Ramipril	2.5 mg 1-0-0	5–10 mg 1-0-0	Generic versions widely available (e.g. Ramipril-ratiopharm®, Ramipril AL®).
Candesartan	8 mg 1-0-0	16–32 mg 1-0-0	For ACE inhibitor-induced cough (e.g. Atacand®, generics available).
Amlodipine	5 mg 1-0-0	10 mg 1-0-0	Dose-dependent ankle oedema (e.g. Norvasc®, generics available).
Chlortalidone	12.5 mg 1-0-0	25 mg 1-0-0	Preferred thiazide-like diuretic; less commonly available in Germany than HCT.
Indapamide	1.25 mg 1-0-0	2.5 mg 1-0-0	Alternative to chlortalidone, with a favourable metabolic profile.
Ramipril/Amlodipine (fixed-dose combination)	5/5 mg 1-0-0	10/10 mg 1-0-0	Single-pill formulation to improve adherence (e.g. Tonotec®, generics).
Candesartan/amlodipine (fixed-dose combination)	8/5 mg 1-0-0	32/10 mg 1-0-0	Single-pill, metabolically neutral.
Spironolactone	12.5–25 mg 1-0-0	25–50 mg 1-0-0	Only initiate treatment if eGFR ≥45 ml/min and K ≤4.5 mmol/l (e.g. Spironolactone-ratiopharm®, Aldactone®).
Eplerenone	25 mg 1-0-0	25 mg 1-0-1 (50 mg/day)	Alternative in cases of spironolactone intolerance (Inspra®, generics).

12. HCA module: Tasks in the hypertension clinic

Process step	Role of the healthcare assistant (HCA)	Resources	Objective
Preparing for the appointment	Collect/scan the HBPM record and medication plan before the appointment	HBPM form, medication plan	Complete set of data prior to contact with the doctor.
Clinic measurement	Carry out and document a standardised BP measurement after a period of rest	Validated upper-arm blood pressure monitor, suitable cuff	Reliable baseline values, fewer measurement errors.
Laboratory organisation	Schedule and monitor follow-up appointments for Na/K/creatinine following a change in treatment	Recall system, laboratory request template	No risk of overlooking safety-critical checks.
Training	Practise HBPM technique and timing of administration with the patient	Training sheet, demonstration device	Improved measurement quality and adherence.
Recall/Re-appointment	Actively follow up with patients with abnormal readings or overdue check-ups	Practice recall list	No missed follow-up appointments.

13. Patient perspective and conversation prompts

A brief, easy-to-understand explanation of the risks significantly increases patients' willingness to take their medication regularly, far more so than a lengthy technical explanation.

Conversation prompts for consultations

"High blood pressure doesn't cause any pain – which is why it's dangerous: it silently damages the heart, kidneys and blood vessels over many years. With your treatment, we can significantly reduce this risk."

"Rather than using a single high-dose tablet, we prefer to combine two low-dose active ingredients – this often works better and has fewer side effects."

"This combination is also available as a single tablet – that saves you from having to take several doses a day."

"How easily can you fit this plan into your daily routine? Are there any tablets you often forget to take or that cause you discomfort?"

14. Documentation modules

Text modules for the patient record card

Initial diagnosis of hypertension: BP [value] mmHg (multiple readings), mean 24-hour ambulatory blood pressure [value], baseline laboratory results normal/abnormal ([details]), ECG showing no evidence of LVH/ischaemia, ACR [value]. SCORE2 risk stratification [value]. Treatment stage [1–4] initiated.

Escalation to level [X→Y]: BP not within target range despite [previous treatment]. Pseudoresistance assessed (measurement quality/adherence/medication history) – [result]. Secondary hypertension [considered/not considered, justification]. New medication: [active ingredient, dose].

Initiation of MRA: Indication – resistant hypertension/PA. Baseline values: potassium [value], eGFR [value]. Spironolactone/eplerenone [dose] 1-0-0. Follow-up appointment arranged in 1–2 weeks.

Follow-up after initiation of diuretics/MRA: potassium [value], creatinine/eGFR [value], BP [value]. No dose adjustment required.

15. Appendix: Practice checklist for hypertension (print format)

- Was the measurement standardised? Multiple readings taken, correct cuff used, rest period observed.
- Has HBPM/ABPM been arranged in cases of unclear or resistant readings?
- Full baseline laboratory tests (Na, K, creatinine/eGFR, glucose, lipids, ACR, ECG)?
- Has adherence been actively assessed (medication burden, side effects, costs)?
- Checked for concomitant medication that raises BP (NSAIDs, steroids, liquorice, the contraceptive pill)?
- Have red flag criteria for secondary hypertension been systematically reviewed?
- For diuretics/MRA: have potassium and eGFR been monitored before starting treatment and after 1–2 weeks?
- Checked whether a fixed-dose combination is available before prescribing multiple individual tablets?
- Has the next follow-up appointment and recall been entered into the practice system?

16. Online resources and reference sites

The following portals provide up-to-date information on the treatment of hypertension in German-speaking countries (Germany, Austria, Switzerland). All links are clickable.

GUIDELINES, REGULATORY BODIES AND RESEARCH

AWMF Register – NVL Hypertension 2023 (D) – Full National Care Guideline on Hypertension, including short and long versions.

German Hypertension League (D) – Guideline commentaries, patient information, list of validated blood pressure monitors.

European Society of Hypertension – Guidelines (International/EU) – Full text and supplementary material for the ESH Guidelines 2023.

Austrian Society for Hypertensiology (A) – Austrian guideline commentaries and continuing professional development materials.

Swiss Society of Hypertension (CH) – Swiss recommendations and continuing professional development programmes on the treatment of hypertension.

SPECIALIST OUTPATIENT CLINICS AND REFERRAL CENTRES

German Society of Nephrology (D) – Addresses of nephrology centres, information on CKD and renovascular hypertension.

German Society for Endocrinology (D) – Specialist information and centres for primary hyperaldosteronism and pheochromocytoma.

German Society for Sleep Research and Sleep Medicine (D) – Directory of accredited sleep laboratories for assessment in cases of suspected sleep apnoea.

PATIENT INFORMATION AND SELF-HELP

German Hypertension League – Patient guidelines (D) – Information on blood pressure measurement and treatment, written in layman's terms.

German Institute for Nutritional Medicine – Salt reduction (D) – Practical dietary recommendations for lifestyle intervention in hypertension.

Literature and sources

Literature and sources

Vancouver citation style: Numbers in the text [N] – Order of first appearance.

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CARDIOMETABOLIC RISK MARKERS

A3 – Lipid management in GP practice

Hypercholesterolaemia · Hypertriglyceridaemia · Practical decision-making on statin therapy

Key message

The aim of treatment is not to target a single laboratory value, but to reduce the risk of an atherosclerotic event.
LDL cholesterol is a causal and treatable risk factor. Lifestyle advice and drug are not alternatives – they are used in combination depending on the level of risk.

1. Overview and relevance

Cardiovascular diseases are the leading cause of death in Germany. Elevated LDL cholesterol levels are considered a causal, reversible risk factor for atherosclerotic events. In GP practices, the lipid profile is a standard part of health check-ups, within the framework of disease management programmes (DMPs) and in the care of high-risk patients.

Key tasks: (1) Determine risk category, (2) Set treatment target, (3) Initiate lifestyle changes and, where appropriate, medication, (4) Monitor progress and ensure tolerability.

Note: Decision-making principle

1. Confirm the measured value and rule out secondary causes.
2. Identify clear high-risk constellations (ASCVD, LDL >190, FH, DM, CKD).
3. Only if necessary: calculate SCORE2 or SCORE2-surgery.
4. Set the LDL target and the required percentage reduction.
5. Select a treatment likely to achieve the target.
6. Assess efficacy and tolerability after 4–12 weeks.
7. Before escalating treatment: check adherence, diagnosis and dosage.

2. Screening logic and risk identification

2.1 Hard indications – no score required

In the following scenarios, the risk category is immediately clear. A structured lifestyle-based waiting period is not appropriate here:

Scenario	Risk category / Consequence
Manifest ASCVD: CHD, myocardial infarction, ischaemic stroke/TIA of atherosclerotic aetiology, peripheral arterial disease (PAD), revascularisation	Very high risk – pharmacological LDL reduction without a preliminary lifestyle-based watch-and-wait period
Clear evidence of atherosclerosis on meaningful imaging	Generally very high risk; consider the quality of the findings
LDL-C >190 mg/dl after exclusion of secondary causes	High risk as a prominent single risk factor; check for FH
Familial hypercholesterolaemia (FH)	At least high risk; very high in the presence of a major risk factor or ASCVD
Diabetes mellitus	Risk category depending on duration, age, organ damage; frequently high or very high
Chronic kidney disease (CKD)	eGFR 30–59: usually high risk; eGFR <30: usually very high risk
Blood pressure >180/110 mmHg	A prominent individual risk factor; urgent blood pressure treatment required

2.2 SCORE2 for individuals without a clear indication

Age	Low to moderate	High risk	Very high risk
<50 years	<2.5%	2.5–<7.5%	≥7.5%
50–69 years	<5%	5–<10 %	≥10%
≥70 years (SCORE2 surgery)	<7.5%	7.5–<15 %	≥15 %

Germany: region with moderate cardiovascular risk (European calibration). Do not use SCORE2 in cases of established ASCVD, diabetes, CKD or FH if the category is already determined by these conditions.

3. Basic diagnostics

3.1 Minimum programme for initial lipid findings

Measure	Comment
Total cholesterol, HDL-C, TG, LDL-C	Measured without fasting is usually sufficient for screening; if TG >400 mg/dl, LDL is often unreliable → use non-HDL-C
Calculate non-HDL-C	Total cholesterol minus HDL-C; more meaningful in cases of high TG, DM, obesity
Blood pressure, smoking, BMI/waist circumference, HbA1c/fasting glucose	For SCORE2 calculation and risk category
Family history: premature ASCVD	Men under 55, women under 60
TSH in cases of suspected or known hypothyroidism or elevated LDL	Rule out secondary causes
Creatinine/eGFR	CKD risk stratification
Lp(a) once in adulthood	Particularly in cases of early-onset family history; genetically stable

3.2 Secondary causes

Findings	Common causes
High LDL	Hypothyroidism, nephrotic syndrome, cholestasis, ketogenic diet, medication (glucocorticoids, retinoids)
High TG	Alcohol, poorly controlled diabetes mellitus, obesity, hypothyroidism, pregnancy, medication (oestrogens, atypical antipsychotics)
Muscle symptoms prior to starting statins	Hypothyroidism, intense physical exertion, polymyalgia, vitamin D deficiency (clinically suspected)

3.3 LDL target levels by risk category

Risk category	LDL-C target
Low	<116 mg/dl (<3.0 mmol/l)
Moderate	<100 mg/dl (<2.6 mmol/l)
High	<70 mg/dl (<1.8 mmol/l) AND ≥50% reduction
Very high	<55 mg/dl (<1.4 mmol/l) AND ≥50% reduction
Second vascular event within 2 years despite maximum therapy	<40 mg/dl (<1.0 mmol/l) may be considered

Note: A target value is not automatically a threshold for prescribing. In cases of low/moderate risk, assess the absolute benefit in conjunction with the baseline value, risk modifiers and patient preference.

4. Further diagnostics and risk modifiers

4.1 Lp(a), non-HDL-C and ApoB

Parameters	When relevant	practice rule
Lp(a)	Once in an adult's lifetime; particularly in cases of early-onset family history	≥50 mg/dl (≥105 nmol/l): clinically significant elevation; risk modifier, not equivalent to ASCVD; statins do not lower Lp(a)
Non-HDL-C	High TG, DM, obesity, non-fasting measurement	Secondary target: ~30 mg/dl above the LDL target
ApoB	LDL-C and non-HDL do not correlate	Not a mandatory routine test for every check-up

4.2 Risk modifiers in borderline cases

If the SCORE2 result is borderline, consider the following factors:

- A family history of premature ASCVD
- Significantly elevated Lp(a)
- Obesity and lack of physical activity
- Chronic inflammatory diseases
- Early menopause or pre-eclampsia
- Psychosocial stress
- Elevated TG/ApoB
- Subclinical atherosclerosis (plaque, not IMT alone)

Coronary calcium score (CAC): only where the decision on primary prevention is genuinely unclear – not a general screening test.

5. Management according to risk level

5.1 Lifestyle advice – specific rather than general

Not ‘You need to eat more healthily’, but two to three measurable targets:

- Reduce saturated fats; replace them with unsaturated fats
- Mediterranean dietary pattern
- Increase soluble fibre and plant-based foods
- Give up smoking
- 150–300 minutes of moderate activity per week plus strength training
- If triglyceride levels are high: stop drinking alcohol, significantly reduce sugary drinks

5.2 Timetable: When to take action?

Situation	Action
Low risk, mild increase	Structured counselling; follow-up after 3–6 months
Moderate risk or marked increase	Follow-up after ~8–12 weeks; then decision on treatment
High/very high risk, LDL >190, FH or ASCVD	Lifestyle changes AND medication simultaneously; review after 4–12 weeks
Target achieved and stable	Follow-up every 6–12 months
Dose adjustment, new medication, adherence issue	Review after 4–12 weeks

5.3 Statins: selection and dosage

Target	Typical statin	Significant reduction in LDL
Moderate intensity	Atorvastatin 10–20 mg	30 to <50 %
Moderate intensity	Rosuvastatin 5–10 mg	30 to <50 %
High intensity	Atorvastatin 40–80 mg	≥50%
High intensity	Rosuvastatin 20–40 mg	≥50 %
Alternative with fewer interactions / lower potency	Pravastatin 20–40 mg, fluvastatin	<30 to ~40%

6 per cent rule: Each doubling of the dose reduces LDL by only a further ~6 per cent. Adding ezetimibe is often more effective than the next dose doubling.

5.4 Step-by-step approach in cases of inadequate statin response

Step	Action	Additional LDL reduction (approx.)
1	Maximum tolerated statin dose	30–55% (dose-dependent)
2	Add ezetimibe 10 mg/day	~20–25% additional
3	Bempedoic acid 180 mg/day (fixed-dose combination with ezetimibe if appropriate)	~15–25 %
4	PCSK9 antibody (evolocumab / alirocumab)	~50–60 %
5	Inclisiran	~50% (endpoint data to be classified differently from PCSK9 antibodies)

PCSK9 antibodies and Inclisiran: Specific prescribing requirements in Germany. Check the G-BA's Medicines Guideline and the summary of product characteristics; co-management by a specialist is usually required.

6. Safety rationale: red flags and clinical errors

Red flags – Immediate action required

▶ CK ≥10 × ULN: Discontinue statin immediately; rule out rhabdomyolysis; check creatinine and myoglobin in urine
▶ Severe muscle weakness + dark urine ± fever: urgent, regardless of CK level
▶ Jaundice, dark urine, marked fatigue whilst on statins: suspend treatment and investigate urgently
▶ TG ≥1,000 mg/dl or abdominal pain with very high TG levels: emergency (risk of pancreatitis)
▶ Untreated LDL ≥250 mg/dl + early cardiovascular event + xanthomas: suspected hyperlipidaemia → lipid clinic

⚠ Common clinical errors – avoid mismanagement

✗ Treating a single value without considering overall risk
✗ In cases of ASCVD or LDL >190, lifestyle management alone for months
✗ Ignoring lifetime risk in young people
✗ Interpreting high HDL as protection against high LDL
✗ Applying SCORE2 non-critically in cases of ASCVD, FH, DM or CKD
✗ Overlooking hypothyroidism or other secondary causes
✗ Routinely measuring CK → incidental findings and discontinuation of treatment
✗ Labelling every muscle problem as statin intolerance
✗ Discontinuing all statins after a single trial
✗ Simply double the statin dose instead of adding ezetimibe
✗ LDL within normal range whilst on treatment → Discontinue statins (incorrect conclusion!)
✗ Equating fish oil supplements with icosapent ethyl

7. Statin-associated muscle symptoms (SAMS)

7.1 Typical SAMS pattern

- Onset: usually within a few weeks to months of starting treatment
- Symmetrical pain / heaviness / weakness in proximal muscle groups (thighs, hips, shoulders)
- CK levels are often normal
- Improvement after discontinuation + recurrence upon re-exposure: increased likelihood

7.2 CK-guided approach

Situation	Management
Muscle symptoms, CK normal or <4 × ULN	Assess clinical severity and risk; if symptoms are tolerable, continue if appropriate; otherwise, a short break followed by re-challenge
CK 4 to <10 × ULN	Discontinue statin; check for physical exertion, TSH and interactions; monitor CK levels promptly
CK ≥10 × ULN	Discontinue statin immediately; rule out rhabdomyolysis; check creatinine and myoglobin in urine
Severe weakness, dark urine, fever	Urgent investigation regardless of CK level

7.3 Safety profile of statins – addressing common concerns

Concern	Classification
Diabetes	Slight dose-dependent increase in risk in those at metabolic risk; vascular benefits outweigh risks where there is an established indication
Liver	Clinically significant liver damage is very rare; mild, stable elevation of transaminases (<3 × ULN) = no contraindication
Memory / Cognition	Large randomised trials show no convincing causal increase
Sleep, depression, sexual function	No robust evidence of a causal link in blinded studies
Pregnancy / Breastfeeding	Refer to the product information; statins should generally be discontinued; individual advice

8. Hypertriglyceridaemia: separate decision-making pathway

8.1 Practical thresholds

Fasting TG	Priority / Approach
150–499 mg/dl	Overall cardiovascular risk, non-HDL-C/ApoB, lifestyle; LDL therapy as first-line treatment
500–879 mg/dl	Significantly elevated; increased risk of pancreatitis; prompt treatment of underlying causes, close monitoring
≥885 mg/dl (≥10 mmol/l)	Severe hypertriglyceridaemia; prevention of pancreatitis a priority; concomitant lipid-lowering treatment
≥1,000 mg/dl or abdominal pain	Very high risk; urgent investigation; in the event of symptoms: emergency

8.2 Treatment for high triglycerides

Action	Comment
Pause or stop alcohol consumption	Most important immediate measure for very high triglycerides
Reduce sugar and rapidly absorbed carbohydrates	Particularly for blood glucose levels of 150–499
Weight, exercise, diabetes management	Treat secondary causes
Statin (if indicated for ASCVD)	First-line treatment – reduces cardiovascular risk; effect on TG levels variable
Fenofibrate / omega-3 (prescription-only)	Consider for very high TG levels; fibrates primarily for the prevention of pancreatitis
Gemfibrozil + statin	Avoid where possible (risk of myopathy)
Icosapent ethyl	Selected high-risk patients with TG ~135–499 on a statin; not to be equated with over-the-counter fish oil

9. Specific patient groups

Group	Special considerations
Older people (>75 years)	Consistent secondary prevention. Primary prevention on a case-by-case basis: frailty, life expectancy, polypharmacy, eGFR. Do not discontinue statins solely on the basis of age.
Familial hypercholesterolaemia (FH)	Untreated LDL ≥190 mg/dl, early cardiovascular event, tendon xanthomas, arcus corneae <45 years → Dutch-LCNS score; consider cascade screening
Diabetes mellitus	Do not apply SCORE2 across the board. Categorise according to duration of DM, organ damage, CKD, risk factors. Often high/very high.
Chronic kidney disease	Adjust dosage according to eGFR; take particular care with rosuvastatin if eGFR <30; check for interactions
Young patients (<40 years)	10-year scores underestimate lifetime risk. Give greater weight to LDL levels, familial hypercholesterolaemia (FH), early events, Lp(a) and smoking.

10. Interactions and common pitfalls

Problem	practice rule
Clarithromycin / erythromycin, azole antifungals	Check for interactions with simvastatin/atorvastatin; discontinue or consider an alternative if necessary
Cyclosporine, HIV/HCV medicines	High risk of interaction; consult the prescribing information or database
Amiodarone, verapamil, diltiazem	Observe dose limits, particularly with simvastatin
Gemfibrozil plus a statin	Avoid where possible
Grapefruit	Relevant for simvastatin; to some extent for atorvastatin (high dose)
Untreated hypothyroidism	Risk of elevated LDL and myopathy; ensure thyroid is under control before escalating treatment
Calcium score increases whilst on statins	Not indicative of treatment failure; plaque calcification may reflect stabilisation
High HDL	Does not offset high LDL or overall risk

11. Standard Operating Procedures (SOPs)

SOP 1 – Lipid screening during health check-ups
1. Determine total cholesterol, HDL-C, TG and LDL-C (non-fasting is sufficient); if TG >400 mg/dl: use non-HDL-C
2. Record blood pressure, smoking status, BMI, HbA1c/fasting glucose and family history
3. Investigate secondary causes: TSH, creatinine/eGFR, current medication
4. Measure Lp(a) once (particularly in cases of early-onset events in the family)
5. Is there a clear indication? → Proceed immediately to treatment decision (Step 6). Otherwise, calculate SCORE2/SCORE2-surgery
6. Document risk category and LDL target; agree on lifestyle goals
7. Arrange a follow-up appointment with a clear schedule (3–12 weeks depending on risk)

SOP 2 – Initiation of treatment for elevated LDL
1. Confirm baseline value (repeat if in doubt); have secondary causes been ruled out?
2. Calculate the required percentage reduction: (current LDL – target LDL) / current LDL × 100%
3. Select a statin: atorvastatin 20 mg (moderate intensity) or atorvastatin 40–80 mg / rosuvastatin 20–40 mg (high intensity)
4. In cases of polypharmacy or drug interactions: consider pravastatin or low-dose rosuvastatin
5. Discuss the benefits and risks; discuss dosage, timing and grapefruit
6. Follow-up after 4–12 weeks: lipid profile, tolerability, adherence
7. Target not achieved? → Follow the 6 per cent rule; add ezetimibe; check adherence

SOP 3 – SAMS / Muscle problems whilst on statins
1. Document symptoms, temporal relationship and muscle status (location, symmetry)
2. Measure CK (only if symptoms are present, not routinely)
3. Rule out interactions, TSH, intense physical exertion, other conditions (PMR, vitamin D deficiency)
4. CK <4 × ULN and symptoms are tolerable: treatment may continue; CK 4–10 × ULN: suspend treatment, investigate causes, monitor
5. CK ≥10 × ULN or dark urine: Discontinue immediately; rule out rhabdomyolysis; creatinine + urinary myoglobin
6. After recovery: Re-challenge with a different statin at the lowest dose; increase gradually
7. Add ezetimibe at an early stage; only assume statin intolerance after ≥2 statins have been tried at low doses

SOP 4 – Long-term care / Follow-up
1. With stable treatment: Follow-up every 6–12 months (lipid profile, adherence, tolerability)
2. Dose adjustment or new medication: follow-up after 4–12 weeks
3. Annual TSH monitoring for patients at increased risk; eGFR/creatinine monitoring tailored to risk
4. High-risk patients: monitoring of LDL target and, if necessary, escalation (ezetimibe → bempedoic acid → PCSK9 antibody consultation)
5. Lp(a): one-off measurement; no follow-up checks unless there is a specific reason
6. Suspected familial hypercholesterolaemia: recommend cascade screening of first-degree relatives; lipid clinic/specialist consultation
7. Do not stop statin treatment on your own initiative if LDL levels are normal whilst on treatment (treatment success = target achieved!)

Practice Pearl: Small step, big impact

Instead of switching to the next statin dose, add ezetimibe:
→ Ezetimibe reduces LDL by ~20–25% – often more than doubling the statin dose (~6%).
→ Better tolerability, lower risk of muscle symptoms.
→ Evidence of clinical benefit, particularly following acute coronary syndrome (IMPROVE-IT trial).
→ In cases of statin intolerance: the lowest tolerable statin dose + ezetimibe as a bridging therapy.

13. Healthcare assistant (HCA) Module: Practice Organisation and Delegation

HCA tasks: lipid management

The following tasks can be prepared and documented by trained practice staff:

Process step	HCA task	Resources / Preparation	Objective
Screening preparation	Order lipid profile, TSH, HbA1c and eGFR; check patient master data for completeness	Laboratory form / request in the patient management system; fasting required only for triglyceride monitoring	Efficient laboratory preparation
Record baseline parameters	Record blood pressure, body weight, waist circumference and smoking status in the PMS	Measurement log; standardised questionnaire	Complete risk documentation
Reorder Management	Recall invitation for lipid screening after the start of treatment (4–12 weeks)	Recall function in the PMS; telephone list for high-risk group	Ensure follow-up monitoring
Hand out patient information	Distribute the handout ‘Cholesterol: When to change lifestyle, when to take medication?’	Standardised patient information sheet (Appendix C)	Counselling and adherence
Prescription management	Prepare statin prescriptions; flag long-term medication in the PMS	PMS long-term prescription; list of interactions for common co-medication	Continuity of care
Report any deviations	Report severe muscle pain or dark urine immediately by telephone	SAMS alert protocol; emergency pathway	Safety

14. Patient communication and conversation templates

14.1 Risk-based approach to initiating a conversation

Situation	Suggested phrasing
Initial finding: elevated LDL	“Your LDL level is elevated. That alone isn’t the deciding factor – we’ll calculate your overall risk and determine the target level we’re aiming for.”
Explaining the lifestyle phase	"Lifestyle changes always help. However, if your risk is high, we won’t delay prescribing medication for months on end."
Prescribing statins	"Statins have been proven to lower LDL and reduce the risk of heart attacks and strokes. Muscle pain can occur even without statins – if you experience any symptoms, please do not simply stop taking them, but give us a call."
Discussing SAMS	“A small proportion of muscle-related side effects are actually caused by the statin. We take this seriously, investigate it and almost always find a suitable solution.”
LDL within the normal range whilst on treatment	"A normal LDL level whilst on the medication shows that it is working – that is the aim. Stopping the medication would cause the level to rise again."

15. Case study

Case study – 57-year-old female patient

Results: LDL 182 mg/dl, total cholesterol 248 mg/dl, HDL 66 mg/dl
Pre-existing conditions: hypertension (treated), hypothyroidism (known), cardiac arrhythmia, positive family history
No known ASCVD.
Approach according to Manual 4 logic:
1. Confirm lipid levels + check TSH and thyroid control (secondary elevation of LDL possible)
2. Complete risk assessment: smoking status, systolic BP, HbA1c/diabetes, eGFR, clarify family history
3. Calculate SCORE2 for Germany (no reliable estimate without complete data!)
4. Measure Lp(a) once; assess probability of familial hypercholesterolaemia (Dutch-LCNS score)
5. In cases of high/very high risk: start statin therapy (likely atorvastatin 20 mg or rosuvastatin 10 mg)
6. LDL 182 mg/dl + target <70 mg/dl → required reduction ~62% → high-intensity therapy immediately if necessary, or add ezetimibe
7. Review after 6–8 weeks; adjust treatment to meet the target
Note: Neither cardiac arrhythmia nor female sex are automatically equivalent to ASCVD.

16. Documentation modules (PMS text modules)

Abbreviations	Text module
INITIAL LIPID	Lipid profile: LDL ___ mg/dl, non-HDL-C ___ mg/dl, TG ___ mg/dl, HDL ___ mg/dl. No/existing ASCVD. Secondary causes assessed: TSH ___, HbA1c ___, eGFR ___. SCORE2 ___%. Risk category ___. LDL target ___ mg/dl.
LIPID-START	Statin therapy: Indications, benefits and side effects discussed. Joint decision to prescribe ___ mg/day. Target: LDL <___ mg/dl, reduction ≥___ %. Follow-up in ___ weeks. Warning signs discussed (muscle pain, dark urine, jaundice).
LIPID-LIFE	Currently no clear indication for medication. Agreed lifestyle targets: ___. Follow-up appointment in ___ weeks/months. If findings remain unchanged, a new risk-based decision on treatment will be made.
LIPID-SAMS	Symptoms of ___ (symmetrical/asymmetrical) present for ___ since ___. CK ___ × ULN, creatinine ___, TSH ___. Interactions/stress factors assessed. Treatment paused until ___; re-challenge with ___ at a dose of ___; follow-up in ___.
LIPID-INTOL	Statin intolerance: intolerance to 1st ___ dose ___ and 2nd ___ dose ___. Symptoms resolved during treatment break. Maximum tolerated statin dose ___. Supplemented with ___; lipidology consultation discussed.
LIPID-TG	Fasting TG ___ mg/dl. Secondary causes: alcohol ___, HbA1c ___, TSH ___, BMI ___, medication ___. No symptoms of pancreatitis. Advice on alcohol, sugar and weight. Follow-up in ___. Emergency advice: abdominal pain → seek immediate medical attention.

Appendix A – Quick overview: GP's overall algorithm

Step	Action
1	Abnormal lipid profile → Check plausibility and previous results
2	TG ≥500 mg/dl → confirm on an empty stomach; prioritise risk of pancreatitis
3	ASCVD / FH / LDL >190 / CKD / diabetes? → Determine risk category immediately
4	Otherwise: calculate SCORE2 / SCORE2-surgery
5	Document target LDL and required reduction
6	Lifestyle: Agree on 2–3 specific, measurable targets
7	High risk: Start statins immediately. Low/moderate risk: defined monitoring period
8	Follow-up after 4–12 weeks
9	Target not met → review adherence, dosage and add ezetimibe
10	If necessary: bempedoic acid or specialist PCSK9/inclisiran strategy
11	Symptoms → SAMS algorithm; do not automatically discontinue treatment permanently
12	Stable → Check-up every 6–12 months

Appendix B – Patient information leaflet: Understanding cholesterol

For patients: Cholesterol – when is lifestyle management sufficient, and when does medication help?

Cholesterol is essential for life. Too much LDL cholesterol can build up in the walls of blood vessels and increase the risk of heart attack, stroke and circulatory problems.

Not every elevated reading means you need to take tablets straight away. We take into account: your age, blood pressure, smoking status, diabetes, kidney function, LDL level, family history and, where relevant, lipoprotein(a).

What you can do yourself:

- ▶ Don’t smoke
- ▶ Exercise regularly (at least 150 minutes per week)
- ▶ Follow a Mediterranean diet; reduce your intake of saturated fats
- ▶ If your triglyceride levels are high: significantly reduce your alcohol and sugar intake
- ▶ Lose excess weight gradually

Why statins?
Statins have been proven to lower LDL and reduce the risk of heart attacks and strokes. Most people tolerate them well.
Muscle pain is common even without statins. If you experience any symptoms: do not stop taking the medication on your own – please ring us!

Report immediately if you experience:

- ▶ Severe muscle weakness
- ▶ Dark-coloured urine
- ▶ Yellowing of the skin
- ▶ Severe upper abdominal pain in patients with known high triglyceride levels

Monitoring: 4–12 weeks after starting treatment or changing the dose. Every 6–12 months once treatment is stable.

Appendix C – Online resources

Online resources (D / A / CH)
Germany (D) → ESC HeartScore – SCORE2/SCORE2-surgery calculator → ESC CVD Risk App (risk calculation) → ESC/EAS Guidelines on Dyslipidaemia 2025 – Focused Update → G-BA Medicines Guideline (prescription restrictions on PCSK9 inhibitors) → DEGAM – Guidelines and recommendations for GP practice
Austria (A) → ÖGK – Lipid therapy guidelines and care models → Austrian Society of Cardiology – Guidelines
Switzerland (CH) → SGIM – Lipid management in internal medicine / general practice → HIN Mediserver – Swiss drug information and interactions

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ENDOCRINOLOGY & DIABETOLOGY (THYROID/OBESITY)

A4 – Elevated glucose and HbA1c levels in GP practice

From an incidental finding in a general urological examination to a definitive diagnosis of diabetes

Prediabetes – HOMA-IR – PCOS – Gestational diabetes – Type 2 diabetes – Red flags

1. Why this chapter is necessary

Slightly elevated glucose levels are among the most common incidental findings in health check-ups (for those over 35) and routine laboratory diagnostics. Levels of 105, 110 or 115 mg/dl in fasting blood tests occur regularly – and present the GP with a typical diagnostic challenge: Has the patient eaten recently after all? Is the patient prediabetic? Is an HbA1c test required immediately, or can we wait three months?

This chapter answers the key question for GPs: a glucose or HbA1c level is abnormal – what specific, safe and sensible action should I take now?

In addition to the ‘cappuccino effect’ (pre-analytical factors), the chapter addresses insulin resistance and HOMA-IR, family risk for type 2 and type 1 diabetes, PCOS as a cardiometabolic risk factor, follow-up care for gestational diabetes, initial diagnosis of type 2 diabetes, pharmacotherapy tailored to the patient’s profile, as well as red flags and the limitations of GP practice.

NOTE

First ensure fasting status and the measurement context, then classify the result against the threshold, then make targeted use of HbA1c / oGTT, and finally plan confirmation and follow-up intervals.

2. The 60-second preliminary check

Before making any decision regarding an elevated glucose or HbA1c level, go through the following eight screening questions:

Table 1: 60-second preliminary check

Check question	Why is this important?	Consequence
Acute symptoms?	Polyuria, polydipsia, weight loss, vomiting, abdominal pain, altered consciousness	If symptoms are present: Red Flag pathway, not the routine pathway.
Truly fasting?	Cappuccino, milk, juice, sugar and snacks distort the fasting value	Do not record as a fasting result; repeat if necessary.
Venous laboratory tests?	Capillary samples, practice devices and CGM do not replace a confirmed laboratory diagnosis	Venous confirmation required when making a diagnostic decision.
Is HbA1c valid?	Anaemia, CKD, pregnancy, blood loss or haemoglobin variants may skew results	In the event of discordance: use glucose / oGTT / trend analysis.
Risk profile?	BMI, waist circumference, blood pressure, lipids, fatty liver, family history	Monitoring should be risk-adapted.
Special group?	PCOS, history-taking for GDM, desire to conceive, steroids, ethnic risk factors, young/slender	Previous or other diagnostic tests; if necessary, oral glucose tolerance test (oGTT) / specialist assessment.
Medications?	Steroids, SGLT2 inhibitors, insulin, sulphonylureas and psychotropic drugs influence risk and test results	Take account of daily profiles, sick-day rules and hypoglycaemia.
Red flags?	Ketones, SGLT2 inhibitors + vomiting, pregnancy, very high readings, foot infection	Treat as a routine case or refer to hospital.

3. Pre-analytical considerations: Was the result valid?

For a diagnostic fasting glucose test, the person should have fasted for 8–12 hours and consumed only water. Cappuccino, latte, juice, sugar, sweets or a snack mean the result is not a reliable fasting value – regardless of the absolute level.

Table 2: Pre-analytical pitfalls and next steps

Situation	Assessment	Next step	Documentation
Glucose 105 after a cappuccino	Not valid when not fasting	Repeat on an empty stomach or check HbA1c if at risk	This value does not indicate prediabetes.
Glucose 112 – definitely taken on an empty stomach	IFG / risk category	HbA1c, risk profile, trend	Abnormal fasting glucose; no diagnosis of diabetes.
Elevated glucose levels due to infection	Stress-induced hyperglycaemia possible	Repeat after recovery; if symptoms occur, carry out an acute check	Document the acute clinical context.
Elevated glucose levels whilst on steroids	Postprandial levels often higher	Check daily profile, dose and duration	Fasting level may be underestimated.

PMS MODULE: Unclear fasting status

Glucose level borderline/elevated if fasting status at the time of blood sampling is not confirmed. No diagnostically useful fasting value. Pre-analytical procedures explained; repeat fasting test or HbA1c planned according to risk profile.

4. Diagnostic cut-off values and confirmatory diagnostics

In the absence of acute symptoms, a diagnosis of diabetes should be confirmed by a second abnormal measurement or a second abnormal parameter. In the presence of classic symptoms (polyuria, polydipsia, weight loss) and a random glucose level ≥200 mg/dl, the diagnosis is likely – nevertheless, further laboratory investigations (ketones, HbA1c, type classification) must not be omitted.

Table 3: Diagnostic cut-off values (adults)

Parameter	Normal	Prediabetes / risk range	Diabetes range	Caution
Fasting glucose (venous plasma)	<100 mg/dl	100–125 mg/dl (IFG)	≥126 mg/dl	Only if fasting status is confirmed.
HbA1c	<5.7 %	5.7–6.4 %	≥6.5 %	Valid only in the absence of relevant confounding factors.
Random glucose	context-dependent	140–199 mg/dl – non-specific	≥200 mg/dl + symptoms: strongly suggestive	Symptoms determine the urgency.
2-hour oGTT value	<140 mg/dl	140–199 mg/dl (IGT)	≥200 mg/dl	Particularly in cases of discordance, PCOS or history-taking for GDM.

NOTE

Do not diagnose diabetes based on a single unreliable reading, a ‘cappuccino’ reading or an isolated HOMA-IR result. Two abnormal readings or two different methods are required to confirm the diagnosis.

SOP 1: Incidental glucose finding from a health check-up

1. 60-second preliminary check: Symptoms? Fasting status? Risk profile? Special patient group?
2. Clarify pre-analytical factors: Was the sample truly taken whilst fasting? Timing, diet, medication?
3. If the result was not taken whilst fasting: document this, plan a repeat test whilst fasting or request an HbA1c test depending on risk.
4. If the fasting result is 100–125 mg/dl: request HbA1c, assess risk profile, provide lifestyle advice, follow-up in 3–6 months.
5. If the result is ≥126 mg/dl or ≥200 mg/dl + symptoms: confirmatory diagnostics, same-day acute assessment, check for ketones.
6. Document findings, classification, next steps and follow-up appointment in the patient record system (PMS).

5. HbA1c: benefits, limitations and pitfalls

HbA1c is useful because it reflects the average blood glucose level over the past few weeks – without the need for fasting and without daily variability. However, it is not a perfect measure. If the HbA1c level does not correlate with glucose levels or clinical findings, confounding factors must be investigated.

Table 4: HbA1c pitfalls

Situation	Possible effect	Alternative / Consequence
Iron-deficiency anaemia	HbA1c may be falsely elevated	Treat iron deficiency; use glucose / oGTT / follow-up.
Haemolysis, blood loss, transfusion	HbA1c may be falsely low or implausible	Glucose profiles, oGTT; take time intervals into account.
Advanced CKD / EPO	HbA1c interpretation limited	Glucose profile; refer to a specialist laboratory if necessary.
Pregnancy	HbA1c has limited diagnostic value	Pregnancy-specific diagnostics (oGTT weeks 24–28).
Haemoglobin variants (e.g. HbS, HbC)	Assay-dependent false results	Contact the laboratory; use an alternative method.

Patient statement regarding implausible HbA1c results: *"Sometimes the long-term value is distorted by anaemia, kidney problems or other factors. In such cases, we do not rely on it blindly, but verify the result with other tests."*

6. Prediabetes: take it seriously without dramatising it

Prediabetes refers to a risk category, not an inevitable fate. It is not DMP diabetes, but a sensible point in time for structured prevention. Studies show that lifestyle changes can significantly delay or prevent progression to overt diabetes.

Table 5: Pre-diabetes risk levels and monitoring intervals

Constellation	Classification	Measures	Monitoring
Glucose 100–109, questionable fasting result	Uncertain result	Repeat on an empty stomach; HbA1c depending on risk	1–3 months or as a routine test
Glucose 110–125, definitely fasting	IFG / suspected prediabetes	HbA1c, risk, lifestyle	3–6 months
HbA1c 5.7–5.9 %	Previously considered a risk category	Lifestyle, weight, blood pressure, lipids	approx. 6 months

Constellation	Classification	Measures	Monitoring
HbA1c 6.0–6.4 %	Higher risk	More intensive counselling, oGTT if necessary	approx. 3 months
IGT on oGTT	Postprandial impairment	Lifestyle changes, high-risk prevention where appropriate	3–6 months
PCOS / GDM + borderline values	Special group	oGTT (generous criteria), specialist referral	promptly / 3–6 months

‘The Talk’ in 5 minutes

Minute 1: Explain the findings – not diabetes, but within the risk range.

Minute 2: Significance – metabolism is under strain, but this can be changed.

Minute 3: Choose one area to focus on (sugar-free drinks, exercise, evening meals, strength training, weight).

Minute 4: Set a specific goal – not just ‘living more healthily’.

Minute 5: Schedule a follow-up appointment.

SOP 2: Confirmation of prediabetes and lifestyle advice

1. Classify the findings: IFG, IGT or HbA1c risk range? A combination?
2. Assess risk profile: BMI, waist circumference, blood pressure, lipids, fatty liver, family history.
3. Check pre-analytical factors: Was the reading truly taken on an empty stomach?
4. Lifestyle counselling with a specific goal: focus on one area at a time, not everything at once.
5. Metformin for prevention: only in exceptional cases; not a standard recommendation without a basis in clinical guidelines.
6. Schedule a follow-up appointment, add the PMS module, and arrange a follow-up visit.

PMS MODULE: Prediabetes

Explanation of prediabetes / risk profile: no confirmed diabetes, but an increased, modifiable risk. Lifestyle counselling with a specific goal has been carried out. Agreed: [Goal]. Follow-up appointment in [Interval].

7. HOMA-IR and insulin resistance

HOMA-IR is a calculated value derived from fasting insulin (µU/ml) × fasting glucose (mg/dl) / 405. It can estimate insulin resistance, but is not a standard test for diagnosing diabetes and is no substitute for HbA1c, fasting glucose or an oral glucose tolerance test (oGTT). Elevated HOMA values are common in cases of obesity, fatty liver and metabolic syndrome – without this in itself constituting an independent indication for treatment.

Statutory health insurance reimbursement: Fasting insulin is not a mandatory service covered by statutory health insurance as part of a health check-up. Where clinically indicated (e.g. PCOS, metabolic syndrome, desire to conceive), it can be arranged as an IGeL service or via special billing – check regional medical association regulations.

Table 6: HOMA-IR in GP practices

Situation	Is HOMA-IR appropriate?	More important / better	Key message
HbA1c + glucose normal, no risk profile	usually no	Routine, general lifestyle	The test is unlikely to change anything.
Obesity / fatty liver / metabolic syndrome	Explanatory if necessary	Weight, waist circumference, lipids, liver, blood pressure	We treat the risk, not just the calculated figure.
PCOS	only as a supplementary measure	oGTT in cases of risk / desire to conceive	A normal HOMA score does not rule out risk.
Confirmed type 2 diabetes	No	DMP, HbA1c, kidneys, heart	The diagnosis has already been made.
Patient's request / self-paying	following consultation	Document benefits / limitations	We only carry out tests if they influence the decision.

NOTE

Requirements for HOMA-IR: standardised morning fasting blood sample, no acute infection, no acute steroid influence, no exogenous insulin.

8. Family history and young patients

Family history is not a foregone conclusion, but a risk indicator. The age at which relatives developed the condition is more important than the mere fact of having diabetes: grandparents who only develop type 2 diabetes at the age of 80 convey a different message than parents or siblings who are already affected by the age of 50.

Table 7: Family history and screening recommendations

Family patterns	Relevance	Screening approach	Comment
Parent with type 2 diabetes	significantly	Regularly, particularly where other risk factors are present	Increased risk, but modifiable.
Both parents have type 2	high	Monitor earlier and more closely	Do not wait until readings are clearly pathological.
Siblings with type 2	relevant	active screening	Genes and living environment are often similar.
Grandparents Diabetes in old age	moderate to low	Routine if otherwise low risk	Don’t overreact.
Several relatives developed the condition at a young age	High / unusual	Check for MODY / FH / metabolic disorders	Age at onset is crucial.
Type 1 in parents / siblings	Different mechanism	If symptoms are present, apply a low threshold for Type 1 diagnosis	Do not equate Type 1 with Type 2.

In young, slim patients with hyperglycaemia, weight loss or ketones, Type 2 diabetes must not be assumed automatically. Type 1, LADA, MODY or secondary diabetes must be actively considered.

9. PCOS, gestational diabetes and special groups

PCOS as a cardiometabolic risk

PCOS is not merely a gynaecological or cosmetic issue. It is a cardiometabolic risk constellation associated with an increased risk of insulin resistance, type 2 diabetes and cardiovascular disease. The Rotterdam criteria apply for diagnosis (2 out of 3: oligo- or anovulation, clinical or biochemical hyperandrogenism, polycystic ovaries). A normal HbA1c level does not rule out an increased risk of hyperglycaemia – particularly in the presence of obesity.

Gestational diabetes (GDM)

Gestational diabetes often resolves after childbirth – but indicates long-term metabolic vulnerability. The lifetime risk of type 2 diabetes following GDM is significantly increased and warrants regular monitoring.

Table 8: Special groups – diagnosis and screening intervals

Special group	Basic screening	Additional screening	Interval / Action
PCOS, low risk	HbA1c, fasting glucose, lipids, blood pressure, waist circumference	oGTT if at risk / planning a pregnancy	1–3 years, risk-adjusted
PCOS + obesity or elevated HbA1c	Basic + liver function tests	oGTT (generous criteria)	3–12 months
PCOS + desire to conceive	HbA1c, fasting glucose	oGTT, gynaecology / endocrinology	promptly
GDM history-taking	HbA1c, fasting glucose	oGTT depending on timing / risk	Regularly, often annually
Steroid therapy	Glucose / HbA1c depending on duration	Postprandial daily profile	Depending on dose and symptoms
Fatty liver / MASLD	Glucose, HbA1c, lipids, liver	oGTT in cases of discordance	6–12 months
Young / slim + symptoms	Glucose, ketones, HbA1c	C-peptide, autoantibodies	Immediately / promptly

NOTE

In cases of PCOS and history-taking for GDM, a normal HbA1c is reassuring but not always sufficient. The clinical risk determines the next diagnostic steps.

10. Confirmed type 2 diabetes: the first 12 weeks

Once the diagnosis has been confirmed, treatment should not consist solely of a diabetes management programme, but rather structured cardiometabolic risk management. The Diabetes Management Programme (DMP) is not merely a formality, but a safety net for the kidneys, eyes, feet and heart.

Basic diagnostic tests: HbA1c / glucose, creatinine / eGFR, UACR (!), lipids, liver function tests, full blood count, electrolytes as appropriate.

Examination: weight, BMI, waist circumference, blood pressure, foot assessment, pulses / sensation as per risk.

Table 9: Structure of the first 12 weeks following diagnosis

Time	Tasks	Who	Documentation
Diagnosis consultation	Confirm diagnosis, check for red flags, explain diagnosis, offer DMP, basic laboratory tests, treatment plan	Doctor	Document the diagnosis.
1–2 weeks	Lab results review, eGFR / UACR, questions, tolerability, medication	Doctor / Healthcare Assistant (HCA)	Document laboratory results and treatment.
4 weeks	Lifestyle goals, blood pressure, weight, side effects, adherence	Doctor	Lifestyle goals in PMS.
8–12 weeks	HbA1c trend, treatment adjustment, review DMP structure	Doctor / Healthcare Assistant (HCA)	HbA1c trend + treatment pathway.
Annually	Feet, kidneys (eGFR + UACR), lipids, vaccination status, eyes depending on findings	Doctor + healthcare assistant (HCA)	DMP documentation, follow-up.

NOTE

UACR (urinary albumin-to-creatinine ratio) is an early marker of diabetic nephropathy and cardiovascular risk. Don’t forget: measure UACR at the time of initial diagnosis and annually as part of the DMP.

SOP 3: Initial diagnosis of type 2 diabetes and enrolment in the DMP

1. Confirmatory diagnostics complete: two abnormal results or two methods.
2. Check for red flags: ketones? Young / slim? Symptoms? SGLT2? Pregnancy?
3. Communicate the diagnosis: explain, play down the severity, answer questions.
4. Offer the Type 2 Diabetes Management Programme (DMP): explain the benefits (structure, follow-up, quality assurance).
5. Arrange baseline laboratory tests: HbA1c, eGFR, UACR, lipids, liver function tests, full blood count.
6. Discuss treatment plan: lifestyle + medication according to profile; set HbA1c target.
7. Plan referrals: ophthalmologist, diabetes specialist in case of red flags, nephropathy pathway.
8. Follow-up appointment in 1–2 weeks: review lab results, address any questions, assess tolerance.

PMS MODULE: Confirmed type 2 diabetes

Type 2 diabetes diagnosed following confirmed diagnosis ([values]). Diagnosis, treatability, DMP, basic diagnostics and treatment options explained in a way the patient can understand. Follow-up appointment arranged.

11. Pharmacotherapy tailored to the patient's profile

The choice of medication does not begin with the list of substances, but with the patient's profile: heart, kidneys, weight, risk of hypoglycaemia, frailty, pregnancy / desire to conceive, preference and eGFR.

Table 10: Choice of medication based on patient profile

Profile	Priority	Check first	Caveats
New-onset type 2, moderate HbA1c, no high-risk complications	HbA1c, tolerability	Lifestyle + metformin	Check eGFR / gastrointestinal tolerance.
CHD / ASCVD / PAD / stroke	Cardiovascular endpoints	GLP-1 receptor agonist or SGLT2 inhibitor with endpoint benefit	Check indication / reimbursement.
Heart failure	Hospitalisation / prognosis	SGLT2 inhibitors	Fluid status, eGFR, sick days.
CKD / albuminuria	Renal protection	SGLT2, RAAS / BP / lipids	Don't forget UACR.
Obesity	Weight + glucose	GLP-1 receptor agonist / GIP-GLP-1 receptor agonist	Gastrointestinal side effects, availability.
Frail / elderly / risk of hypoglycaemia	Safety	DPP-4, simple regimens, de-intensification	Sulfonylureas / insulin critical.
Very high HbA1c / symptomatic	Rapid control	Consider insulin / diabetology	Type 1 / LADA / check for ketones.

Sick-day guidelines

Patients with diabetes need clear instructions on what to do on days when they are unwell (vomiting, diarrhoea, fasting, surgery). These guidelines should be provided in writing.

Table 11: Sick-day guidelines by medication

Medication	Illness scenario	Action	Warning signs
Metformin	Vomiting, dehydration, severe infection, hypoxia, surgery / chemotherapy	Pause treatment / seek medical advice	Dehydration, risk to kidney function.
SGLT2 inhibitors	Vomiting, diarrhoea, fasting, surgery, severe infection, ketogenic diet	Suspend treatment, monitor ketones (!)	Vomiting + abdominal pain → emergency.
GLP-1 / GIP-GLP-1 receptor agonists	Severe vomiting, dehydration	Seek medical advice	Persistent abdominal pain / vomiting.

Medication	Illness scenario	Action	Warning signs
Sulfonylurea	Poor appetite, CKD, alcohol	Risk of hypoglycaemia; adjust treatment if necessary	Sweating, trembling, confusion.
Insulin	Acute illness	Do not discontinue treatment across the board; plan in advance	Hyperglycaemia / ketones or hypoglycaemia.

Practice Pearl

Do not combine GLP-1 receptor agonists (GLP-1-RA) and SGLT2 inhibitors (do not combine GLP-1-RA with DPP-4 inhibitors either – no additive benefit, subject to statutory health insurance reimbursement assessment). Actively address de-intensification in cases of frailty: safety takes precedence over HbA1c target.

12. Red flags and limitations of GP practice

Table 12: Red flag matrix

Findings	Suspicion	Urgency	Action
Glucose ≥200 mg/dl + polyuria / polydipsia	Manifest hyperglycaemia	orange	Ketones, basic laboratory tests, same day.
Young / slim + weight loss + hyperglycaemia	Type 1 / LADA	orange / red	Ketones, C-peptide / antibody, diabetology / clinic.
Vomiting / abdominal pain + ketones	DKA	RED	Clinical presentation / Immediate emergency care.
SGLT2 + vomiting / abdominal pain	Euglycaemic DKA possible	RED	Suspend SGLT2, ketones / seek immediate medical attention.
Elderly, very high glucose, confused / dehydrated	HHS (hyperosmolar coma)	RED	Ambulance / hospital.
Pregnancy + hyperglycaemia	Foetal-maternal risk	RED	Immediate consultation.
Hypoglycaemia in frail patients	Risk of falls / loss of consciousness	orange	Reduce the intensity of treatment.
Foot wound + redness / fever	Diabetic foot infection	orange / red	Foot / wound / hospital interface.

RED FLAG – ACT IMMEDIATELY

⚠ Vomiting + ketones + diabetes = DKA until proven otherwise → go to hospital immediately.

⚠ SGLT2 + vomiting + abdominal pain = euglycaemic DKA even with glucose 160 → pause SGLT2, check for ketones, go to hospital.

⚠ Young / slim + weight loss + hyperglycaemia = never automatically classify as type 2.

⚠ Pregnancy + elevated blood sugar = act immediately, do not wait.

SOP 4: Red flag escalation and acute management

1. 60-second preliminary check: Symptoms? Ketones? SGLT2? Pregnancy? Age + dehydration?
2. Measure ketones (urine or blood): positive → escalation (hospital or same-day referral to a diabetes clinic).
3. Assess vital signs, level of consciousness, hydration, blood pressure and glucose (venous).
4. Immediately discontinue SGLT2 inhibitors if ketoacidosis is suspected.
5. Pregnancy + hyperglycaemia: immediate consultation with gynaecology/obstetrics.
6. Diabetic foot with redness / fever: wound care on the same day / specialist pathway.
7. Hypoglycaemia with impaired consciousness: intravenous glucose / glucagon, emergency services.
8. Document findings, measures and escalation in the patient record.

13. Common misjudgements

Table 13: Top errors and corrections

Error	Risk	Correction
Treating non-fasting blood as a fasting value	Misdiagnosis	Document pre-analytical procedures.
Diagnosing diabetes based on a single test result	Overdiagnosis	Confirmatory diagnostics.
Ignoring a glucose reading of 110	Prevention missed	Risk profile + progression.
Rely on HbA1c despite anaemia	Incorrect classification	Check for HbA1c pitfalls.
Use HOMA-IR as a diagnostic tool	A parallel approach alongside standard diagnostics	Prioritise standard diagnostics.
PCOS without a metabolic check-up	Risk overlooked	Follow the PCOS pathway.
Failure to perform history-taking for GDM	High-risk case missed	Ask specifically and record in history-taking.
Young, slim patient with diabetes = Type 2	Risk of DKA / LADA	Ketones, C-peptide, antibody.
SGLT2 without ‘sick-day’ rules	Ketoacidosis	Education at the start.
Sulfonylureas in frailty	Hypoglycaemia / falls	Avoid / reduce intensity.
Do not measure UACR	Renal protection missed	eGFR + UACR at initial diagnosis and annually.
Patient feels ashamed	Loss of adherence	Do not give moralising advice.

AVOID CLINICAL ERRORS

– Never diagnose diabetes based on a single reading – always seek confirmation.
– Never record a ‘cappuccino’ reading as a fasting reading.
– Never prescribe SGLT2 inhibitors without following the ‘sick-day’ guidelines.
– In cases of frailty: prioritise safety over HbA1c targets; critically review the use of sulphonylureas and insulin.
– UACR is mandatory – not optional.

14. Case-based training – 10 key cases

CASE 1: Glucose 105 after a cappuccino

Hyperglycaemia in a 48-year-old woman, BMI 27, no family history of diabetes. Glucose 105 mg/dl.

Findings	Assessment	Action
Glucose 105, questionable fasting status	Cannot be interpreted as a fasting value. No prediabetes classification.	Repeat as a fasting test or measure HbA1c according to risk profile.

PMS MODULE: Case 1

Glucose level borderline, pre-analytical status unclear (cappuccino not avoided). No valid fasting result available. Pre-analytical status explained; fasting test in 4 weeks or HbA1c planned.

CASE 2: Glucose 118, HbA1c 6.0%, BMI 31

52-year-old man, definitely fasting, known to have fatty liver, father has type 2 diabetes.

Findings	Assessment	Action
Glucose 118 (fasting), HbA1c 6.0%, BMI 31	Prediabetes likely (IFG + HbA1c in the risk range).	Blood pressure, lipids, waist circumference. Lifestyle goals. Follow-up in 3 months.

PMS MODULE: Case 2

Prediabetes explained. Lifestyle target agreed: [target]. Follow-up in 3 months.

CASE 3: HOMA-IR 2.8, HbA1c 5.5%, LDL 227 mg/dl

Patient arrives having done their own research on 'insulin resistance' and wishes to be tested. LDL levels are abnormal in the latest lab results.

Findings	Assessment	Action
HOMA-IR 2.8, HbA1c normal, LDL 227	HOMA is a risk indicator, but LDL is the priority. No diagnosis of diabetes.	Explain HOMA: an explanatory measure, not a substitute for diagnosis. Prioritise LDL treatment.

PMS MODULE: Case 3

Pre-analytical aspects of insulin resistance and HOMA-IR cut-off values explained. No diabetes diagnosis. LDL risk discussed, treatment initiated.

CASE 4: PCOS + desire to conceive + HbA1c 5.6%

29-year-old woman, diagnosed with PCOS, wishing to conceive, BMI 29.

Findings	Assessment	Action
HbA1c 5.6%, PCOS, desire to conceive	Normal HbA1c, but PCOS + desire to conceive → consider oGTT, gynaecology/endocrinology.	Arrange oGTT, referral to gynaecology/endocrinology, lifestyle advice.

PMS MODULE: Case 4

PCOS metabolic assessment: HbA1c and fasting glucose. Desire to conceive: consider oGTT. Interdisciplinary consultation arranged.

CASE 5: History-taking for GDM without follow-up

38-year-old woman, GDM 3 years ago, no follow-up since then, BMI 26.

Findings	Assessment	Action
GDM 3 years ago, HbA1c 5.8%	Explain the prediabetes risk category and the long-term risk of GDM.	HbA1c / fasting glucose annually, oral glucose tolerance test (oGTT) if necessary. Lifestyle. Follow-up appointment.

PMS MODULE: Case 5

Actively perform history-taking for GDM. Explain increased long-term risk. Agree on regular check-ups.

CASE 6: Weight loss + polyuria + glucose 220

24-year-old man, slim, 6 kg weight loss in 4 weeks, polyuria, tired.

Findings	Assessment	Management
Glucose 220, weight loss, young / slim	Type 1 / LADA / possible decompensation. Never automatically classify as type 2.	Check for ketones immediately! Basic laboratory tests, C-peptide, antibody. Refer to a diabetes specialist / hospital on the same day.

PMS MODULE: Case 6

Acute hyperglycaemia with suspected Type 1. Ketones positive. Immediate clinical referral arranged.

CASE 7: Type 2 diabetes + eGFR 48 + albuminuria

67-year-old man, type 2 diabetes for 8 years, eGFR 48, UACR 180 mg/g.

Findings	Assessment	Action
eGFR 48, UACR 180 mg/g, type 2 diabetes	Cardiovascular. High-risk pathway: check SGLT2 + RAAS + BP + lipids.	SGLT2 inhibitor indication, RAAS blocker, blood pressure < 130/80, LDL target < 55.

PMS MODULE: Case 7

Diabetic nephropathy G3b A2. Cardiorenal risk pathway initiated. UACR and eGFR monitored annually.

CASE 8: Frail + HbA1c 7.8% + hypoglycaemic episodes

81-year-old woman, care level 2, sulphonylurea, 3 falls in the last year.

Findings	Classification	Action
HbA1c 7.8%, sulphonylurea, falls	Safety margin from target value. Sulphonylurea as a risk factor for falls.	Replace or discontinue sulphonylurea. Raise the HbA1c target (7.5–8.5%). De-intensification.

PMS MODULE: Case 8

De-intensification discussed and initiated. Safety prioritised over HbA1c target.

CASE 9: Prednisolone + glucose 280 in the evening, fasting 108

59-year-old man, prednisolone 40 mg/day, evening hyperglycaemia, fasting glucose 108.

Findings	Assessment	Action
Prednisolone, fasting glucose 108, 280 mg/dl in the evening	Steroid-induced postprandial hyperglycaemia. Fasting level underestimated.	Daily profile (postprandial!), insulin at lunch/dinner as per plan if necessary.

PMS MODULE: Case 9

Steroid-induced hyperglycaemia explained. Daily profile requested. Treatment adjustment based on progress.

CASE 10: SGLT2 + vomiting + abdominal pain + glucose 165

54-year-old man, empagliflozin 10 mg, has had vomiting and abdominal pain since yesterday.

Findings	Assessment	Management
SGLT2 inhibitor, vomiting, glucose 165 (not significantly elevated)	Euglycaemic ketoacidosis possible – glucose may be normal!	Discontinue SGLT2 immediately. Measure ketones. Assess clinical presentation if results are positive.

PMS MODULE: Case 10

Risk of euglycaemic DKA identified. SGLT2 paused. Ketones positive → clinical management initiated.

15. Practice tools: healthcare assistant (HCA), follow-up appointments, PMS

Healthcare assistant (HCA) module: Processes and tasks

HCA task matrix

Process step	HCA task	Resources / Guidelines	Objective
Laboratory preparation GU	Check fasting status, hand out information sheet	Fasting information sheet	Ensure correct pre-analytical procedures.
Receipt of laboratory results	Abnormal glucose / HbA1c → doctor's triage	Triage list 'immediate / elective'	Timely medical assessment.
Healthcare assistant (HCA) triage: red flag	In cases of vomiting, ketone detection, pregnancy + high blood sugar: immediate	Red Flags checklist	Escalation without delay.
DMP enrolment	Prepare form, record HbA1c / blood glucose / weight, enter recall	DMP checklist	Complete enrolment.
Annual follow-up for people with diabetes	Generate reminder: UACR, lipids, foot, HbA1c, vaccination, eye	Follow-up calendar / PMS list	Don't forget any organs.
Hand out sick-day information sheet	When starting SGLT2 / GLP-1 treatment, provide the handout and explain it	Sick-day leaflet	Patient safety in the event of illness.

Healthcare assistant triage – contact a doctor immediately

Situation	Action
Vomiting, abdominal pain, confusion or rapid breathing	Inform the doctor immediately.
Severe thirst, polyuria, weight loss (acute)	Contact a doctor immediately.
Positive for ketones (urine dipstick test)	Contact your doctor immediately.
SGLT2 inhibitors + ill / fasting / planned surgery	Inform your doctor immediately.
Pregnancy + elevated blood sugar levels	Inform your doctor immediately.
Foot wound with redness / fever	Inform your doctor immediately.
Hypoglycaemia / low blood sugar	Inform your doctor immediately; take oral glucose if necessary.

Follow-up intervals

Table 14: Follow-up schedule

Situation	Recommended follow-up
Glucose 100–109, fasting status uncertain	Every 1–3 months or routinely depending on risk
Glucose 110–125	3–6 months
HbA1c 5.7–5.9 %	approx. 6 months
HbA1c 6.0–6.4 %	approx. 3 months
PCOS + planning a pregnancy	promptly
History of gestational diabetes without follow-up care	promptly / 3 months
New-onset type 2 diabetes	1–2 weeks for laboratory review, then 8–12 weeks
SGLT2 / GLP-1 initiation	2–6 weeks if at risk, otherwise 4–12 weeks
Hypoglycaemia or red flag	On the same day / promptly

PMS Module Library (Quick Reference)

PMS MODULE: Unclear fasting status

Glucose level borderline where fasting status is unconfirmed. Pre-analytical considerations explained. Fasting follow-up / HbA1c planned.

PMS MODULE: IFG (100–125 mg/dl)

Fasting glucose [X] mg/dl (abnormal fasting glucose). No diabetes. HbA1c [Y] %. Risk explained, lifestyle target agreed. Follow-up appointment [date].

PMS MODULE: Suspected type 1 / LADA

Due to young age / slender build / weight loss / symptoms / ketones, suspicion of type 1 / LADA. No routine type 2 pathway. Ketones / baseline laboratory tests and diabetic / clinical assessment arranged.

PMS MODULE: SGLT2 Sick-Day Guidance

Sick-day guidelines explained: treatment should be paused in the event of vomiting, diarrhoea, fasting, surgery, severe infection or dehydration. Warning signs of ketoacidosis and emergency procedures for ketones discussed. Handout provided.

PMS MODULE: De-escalation in cases of frailty

De-escalation discussed: safety prioritised over HbA1c target. [Medication] reduced / discontinued. New HbA1c target [X–Y %]. Follow-up monitoring agreed.

16. Online resources and reference sites (clickable)

All links were checked in June 2026. Guidelines and organisational websites should be checked regularly to ensure they are up to date.

1. Guidelines, regulatory bodies and research (Germany / Austria / Switzerland)

NVL Type 2 Diabetes – AWMF (Germany) – National Care Guideline for Type 2 Diabetes; authoritative guideline for GP practice

DDG – German Diabetes Society – professional association; practical recommendations, guidelines, continuing professional development, current recommendations

DEGAM – German Society for General Practice – User version of the NVL Diabetes 2025 and DEGAM GP guidelines available free of charge

ÖDG Guidelines – Austrian Diabetes Society – Austrian guidelines for diabetes mellitus; updated annually

DGE PCOS S2k Guideline 2025 – AWMF – Current S2k Guideline on PCOS 2025 from the German Society for Endocrinology

2. Outpatient clinics, specialist clinics and referrals

DDG Patient Information, Advice & Support – Search for DDG-accredited treatment centres and specialist diabetes practices

ÖDG – Austrian Diabetes Society – Austrian professional association; search for a network of specialist outpatient clinics and expert contacts

Swiss Diabetes Society (SDG) – Patient organisation and professional network in Switzerland; counselling and training services

3. Patient organisations, self-help and networking

diabetesDE – German Diabetes Aid – Largest patient organisation in Germany; advice, self-help groups, information material

Diabetes Self-Help Overview (diabetesDE) – Nationwide map and list of regional self-help groups for patients

diabetes.or.at – Austrian self-help portal – Official self-help portal of the Austrian Diabetes Association; guides and groups

diabinfo.de – Independent diabetes information portal – Patient information, contact points and self-help groups; DDG / diabetesDE initiative

17. References and evidence base

For final application, threshold values, DMP requirements, summary of product characteristics, eGFR thresholds, STIKO recommendations and regional billing issues should be regularly checked against current German guidelines, NVL / DDG practice recommendations, summary of product characteristics and G-BA / KV regulations.

This chapter sets out practice-oriented guidelines and is not a substitute for an individual review of guidelines in complex situations, during pregnancy, in cases of severe exacerbation or where the form of diabetes is unclear.

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Appendix A – Patient handouts

PATIENT HANDOUT: My blood sugar was slightly elevated

A slightly elevated blood sugar level does not automatically mean you have diabetes. The key factors are whether you were truly fasting and whether any other test results are abnormal. Contact your doctor immediately if you experience: severe thirst, frequent urination, weight loss, vomiting, abdominal pain or severe weakness.

PATIENT HANDOUT: Prediabetes – What you need to know and what you can do

Your readings are not within the diabetes range, but they indicate an increased risk. You can actively manage this by: choosing sugar-free drinks, walking for 10–15 minutes after meals, getting 150 minutes of exercise per week, doing strength training, eating a high-fibre diet, and setting a realistic weight goal.

PATIENT INFORMATION SHEET: Insulin Resistance and HOMA-IR

HOMA-IR is a calculated value based on fasting insulin and fasting glucose. It can provide guidance, but is less standardised than HbA1c, fasting glucose or an oral glucose tolerance test, and does not determine treatment on its own.

PATIENT INFORMATION SHEET: PCOS and blood sugar

PCOS can affect metabolism. HbA1c and fasting glucose should be checked regularly. If you are planning to conceive or have risk factors, an oral glucose tolerance test may be advisable. Exercise and achieving a healthy weight also help with hormonal metabolism.

PATIENT INFORMATION SHEET: Previous gestational diabetes – long-term risk

Gestational diabetes often resolves after childbirth, but is associated with an increased risk of developing diabetes later in life. Regular monitoring (HbA1c, and an oral glucose tolerance test (oGTT) if necessary) therefore remains advisable – even years after childbirth.

PATIENT INFORMATION SHEET: Newly Diagnosed Type 2 Diabetes

We check not only blood sugar levels, but also kidney function, urine (albumin!), blood lipids, blood pressure, feet, eyes, vaccination status, medication and lifestyle. The aim is to protect the heart, kidneys, eyes, nerves and blood vessels. Diabetes is treatable – it is not a punishment, nor is it a life sentence.

PATIENT LEAFLET: Warning signs of diabetes – when to seek immediate medical attention

Contact the surgery immediately if you experience: vomiting, abdominal pain, confusion, severe weakness, rapid breathing, a smell of acetone, very high blood sugar levels, positive ketones (urine test strips), hypoglycaemia (sweating, trembling, confusion) or a foot wound with redness / fever.

PATIENT HANDOUT: Sick-day guidelines for people with diabetes

SGLT2 inhibitors (e.g. empagliflozin, dapagliflozin): SUSPEND treatment in the event of vomiting, diarrhoea, fasting, surgery or a severe infection, and contact your GP's surgery. Metformin: SUSPEND treatment in the event of vomiting or dehydration. Insulin: never stop taking it without medical advice, but monitor blood sugar levels closely if you are unwell.

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