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Medicine in Dentistry

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This book is dedicated
to our families, friends,
and mentors.

Foreword

Dear Colleagues,

Congratulations on having the courage to buy this book!

I am quite sure that you will find it very useful. I would also like to congratulate myself, because it's not every day you get the opportunity to write the foreword to a bestseller.

You are probably wondering why I am so sure that this book will be a bestseller. Well, let me take you back to 2011. When I took over as editor-in-chief of "Quintessence Dentistry", I asked the "Tröltzsch brothers" (Markus and Matthias, both of whom I had trained as students at my former university) if they would like to join my editorial team. When I was reorganizing the editorial team, I immediately thought of the two of them (who at the time were still future oral and maxillofacial surgeons) when we launched the "General Medicine" section. We were all convinced that this aspect of horizontal networking with medicine would become more and more important for dentists, and thus for our readers, from year to year in times of demographic change with its characteristic and expected aging and multimorbidity of German society. This was one of my best decisions, because the concept was a complete success: Today, it is impossible to imagine "Quintessence Dentistry" without this section.

The next logical step was to publish a book for dentists based on the newly created section. The many pharmacologic aspects, tips, and checklists alone make this book worth buying. In addition, there are many other topics - from cardiovascular risk to pulmonary and gynecologic issues - that need to be considered in dental practice.

Dentistry is medicine; that has always been clear. But the COVID-19 pandemic has made this even clearer, because the oral cavity is and remains the first line of defense against invaders, and oral immunocompetence is more important than ever. This book is the first time that everything has been brought together in a really tangible, stringent, and logical way, making it a must-read for every dentist.

Prof Dr Roland Frankenberger

Preface

How are medicine and dentistry related? Are they independent disciplines, or is dentistry simply a branch of medicine? This question is not easy to answer, and if you look at the curriculum of each degree program, you will find very little content from the other.

Medicine plays a crucial role in dentistry because dentists often see patients more frequently than other doctors. Consequently, the medical aspect becomes quite significant, particularly for early interventions. Dentists are in a great position to facilitate early diagnosis of multiple internal diseases and interdisciplinary treatment, often resulting in decreased hospitalization in older adults and improved quality of life. The widely debated effects of an aging population underscore these realities and emphasize the importance of medical expertise in routine dental practice.

Even under these circumstances, medical content remains a minor part of dental studies. The demanding core subjects like prosthodontics, operative dentistry, and periodontology as well as lab work leave limited time to explore other fields. At the same time, most specialist programs have a requirement for at least 5 years of training after medical school. Yet today's dentists are expected to possess extensive medical knowledge for both treatment purposes and increasingly for forensic reasons. But when and how are they supposed to acquire this knowledge?

Although it is possible to acquire this knowledge independently, finding literature that balances detail with practical application can be challenging. This book has been written for precisely that reason. It aims to equip you, dear reader, with the essential medical knowledge needed for day-to-day dental practice.

The invitation from Prof Dr Roland Frankenberger to oversee a portion of the German dental magazine „Quintessence Dentistry", which disseminates medical knowledge for dentists, led to an influx of technical questions from readers. These inquiries, along with numerous discussions in our courses, inspired the creation of this book and influenced its structure.

The initial chapter offers a concise review of fundamental concepts, followed by the second chapter, which focuses on pharmacologic information. The third chapter, both extensive and detailed, addresses prevalent medical issues, spanning from cardiovascular conditions to gynecology. All chapters provide essential background knowledge in a clear and succinct manner.

The concluding chapter, titled "Medical knowledge for the Dental Team", concentrates on essential information required to ensure the well-being of both dentists and their support staff.

Over the years, we have meticulously chosen our authors, and we are thrilled to have so many top-notch specialists involved in this project. An extraordinary accomplishment of theirs has been to encapsulate their expertise within a strict framework. We would like to take this moment to express our gratitude to all who contributed to this book, particularly those behind the scenes. Special thanks go to Mr Wolters and Mr Meenen of Quintessence Publishing Berlin (both now retired), whose dedication

made this project possible. Additionally, Mrs Hattenbach provided unwavering commitment while completing the book with us.

The COVID-19 pandemic highlighted the critical role of hygiene in dental practices and the potential dangers posed to dental teams by oral pathogens. Thankfully, we have overcome the primary obstacles, allowing us to look ahead with optimism. For many months, the uncertainty brought about by COVID-19 affected the outlook for our dental practices and our professional lives. This book delves into that period of uncertainty.

Each article in the book is written to stand on its own and can be read individually according to the topic of interest. We hope you find this book useful and thank you for your time in reading it. If you have any questions or comments, please do not hesitate to contact us at conference@dr-troeltzsch.de.

Yours,
Markus Tröltzsch
Philipp Kauffmann
Matthias Tröltzsch

Additional material
This book contains many checklists and additional material.

You can access them using this QR code or via the link https://video.qvnet.de/b20791/

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Contents

Cerebrovascular events—Epidemiology, symptoms, etiology, diagnosis, prognosis, and prophylaxis

Jan Samuel Schenkel, Sven Schippling, Claudio Rostetter

Introduction

Cerebrovascular events (eg, stroke, apoplexy) are among the most common diseases and the second leading cause of death worldwide.[6,22] According to an evaluation by the American Heart Association (AHA), a person suffers a stroke every 40 seconds in the USA.[17] Early detection of this acute-onset disease is crucial. In addition to the most important risk factor of high blood pressure, other factors, including diabetes mellitus, smoking, elevated blood lipid levels, and lack of exercise, also play a major role in the development of cerebrovascular events.[16]

Hemorrhagic and ischemic infarctions must be differentiated. However, this is not possible on the basis of clinical symptoms alone, so imaging of the brain or head is absolutely necessary in acute situations. Differentiation is of particular importance because it determines the type of therapy that should be carried out. In a hemorrhagic infarction, there is hemorrhage into the brain, such as in the case of hypertensive hemorrhage into the basal ganglia (loco typico). In contrast, in an ischemic infarction, either a higher degree of vascular stenosis (*hemodynamic infarction*) or acute complete vascular occlusion, such as in the case of arterial or cardiogenic embolic territorial infarction, is the triggering mechanism. The basic distinction between a hemorrhagic and an ischemic infarction is made with CT or, if available, by magnetic resonance imaging (MRI) of the skull.

A special form of infarction is the transient ischemic attack (TIA). In this case, the symptoms regress completely within 24 hours, although a complete stroke may ultimately be identified in a follow-up MRI.

In addition to peracute (immediate onset) sensory disturbances and paralysis, the classic symptoms of a stroke or TIA include the sudden monocular loss of

! Strokes occur frequently, and in many cases, result in the need for care at a later stage.

The symptoms are diffuse.

Prophylactically, patients are anti-coagulated.

vision (amaurosis fugax), severe headache or dizziness, dysarthric complaints (speech disorders), or a disturbance of oculomotor function, such as in infarcts in the posterior stromal area. Cerebrovascular events still entail a high lethality. In one study, it was found that more than 50% of affected persons died within 5 years of their first stroke (patient age at the time of the stroke ≥ 45 years).[15]

Case study

A 65-year-old patient arrived at his scheduled dental appointment for a routine checkup. When asked how he was feeling, he mentioned that his right arm felt weak since the morning and that touch feels different from the opposite side. The dentist arranged for immediate referral to a nearby hospital, where a CT scan revealed an ischemic infarction in the left hemisphere of the brain. The patient was immediately transferred to a hospital with specialized diagnostic and therapeutic neurologic expertise (stroke unit) for evaluation of lysis therapy. Thanks to the immediate referral, rapid diagnosis, and rapid start of therapy, the patient was able to return to his usual environment after neurorehabilitation was completed.

Epidemiology

In Europe, cerebrovascular events have an incidence of 101 to 240 per 100,000 men and from 63 to nearly 160 per 100,000 women over a 1-year period.[9] According to the 2006 Framingham study, the lifetime risk of suffering a stroke is estimated to be about 20% for women (one in five women) and about 17% for men (one in six men).[20]

Symptomatology

The symptoms of a cerebrovascular event can be varied. The goal is to detect a TIA or stroke as early as possible. It is particularly important to have a low threshold for further diagnostic measures.

If a patient describes a sudden onset of symptoms such as speech disturbances, feelings of weakness, loss of sensibility, visual disturbances, or dizziness, this may be an indication of a cerebrovascular event. Paralysis or sensory disturbances typically occur unilaterally. Headache, nausea, or vomiting may accompany symptoms. Speech disorders often manifest as excess speech production (*fluid aphasia*) or reduced speech production (*non-fluid aphasia*). A speech disorder (dysarthria), on the other hand, is characterized by slurred speech or even the absence of any speech production (anarthria).

TIA describes a transient, vascular neurologic deficit and can be a kind of harbinger of a stroke, so further diagnostics are absolutely necessary.[4] In a TIA, the symptoms, which are indistinguishable from those of a stroke, are spontaneous and completely regress within 24 hours. However, the term is increasingly in flux and is sometimes used only when symptoms last less than 1 hour and no manifest infarcts can be detected by MRI.[7]. Even in the case of a TIA, hospitalization for further evaluation is urgently indicated because there is a significantly increased risk of a complete stroke in the hours to days that follow, and cerebral infarction occurs in 10% to 30% of those affected within the next 5 years.[4,5,12]

Etiology

Ischemic infarction

Ischemic cerebral infarctions account for the majority of strokes (70%–80%).[14,21] Ischemic infarction is caused by acute vascular occlusion or severe stenosis of a vessel responsible for cerebral perfusion. As a result of the occlusion, the brain tissue dependent on the vessel is no longer or insufficiently perfused (ie, supplied with oxygen and necessary substrates such as glucose). Nerve cells are extremely sensitive to undersupply, and irreversible damage or cell death occurs after only 5 to 6 minutes without oxygen supply.[19] Acute vascular occlusion can be either atherothrombotic or embolic. In the case of thrombotic occlusion, lipids are deposited in the vessel wall over a prolonged period of time in the course of arteriosclerosis, which eventually results in atherosclerotic plaque formation. If such a plaque ruptures, this leads to the formation of a thrombus in the wall, which can result in complete vascular occlusion.[19]

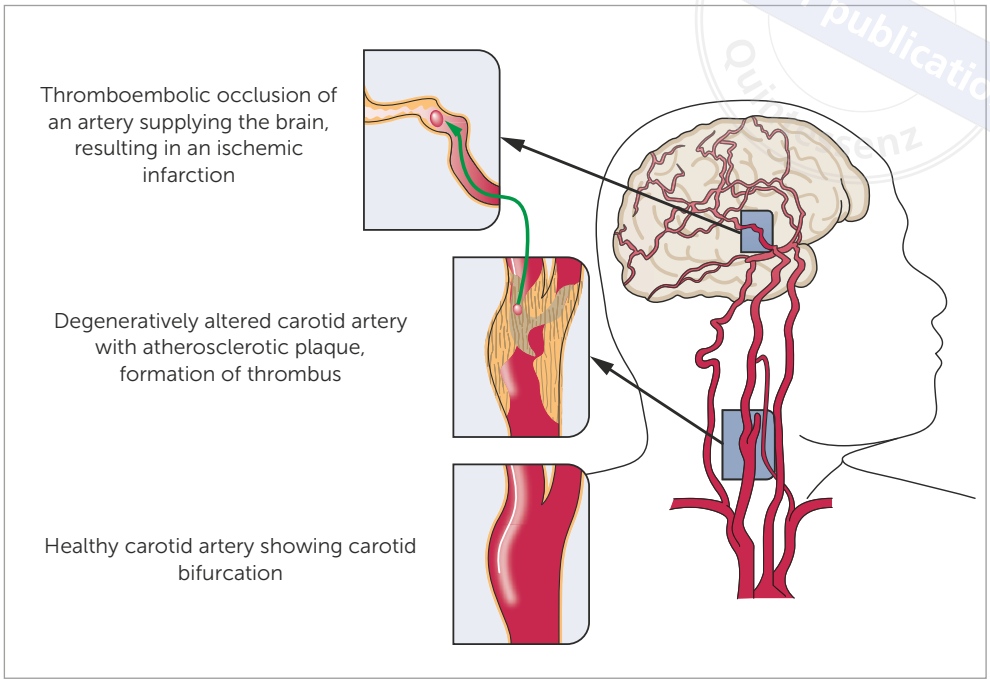

Fig 1 Schematic representation of the bifurcation of the carotid artery where atherosclerotic plaque has accumulated. This can rupture and thus cause an ischemic infarction. (Modified from: Mayo Foundation for Medical Education and Research, www.mayoclinic.org/ischemic-stroke/img20009031.)

An embolism is the acute occlusion of an arterial vessel at the base of the artery via the formation of thrombus material (eg, from the heart). Such thrombi as a source of arterial embolisms can occur, for example, in the course of atrial fibrillation. On the other hand, embolization into a downstream vessel may also occur as a result of atherosclerotic plaque build-up (eg, in the region of the carotid bifurcation). The most commonly affected vessel, whether due to embolic or thrombotic occlusion, is the middle cerebral artery, whereas occlusions of the anterior cerebral artery and posterior cerebral artery are less common (Fig 1).

Precise clinical neurologic examination and the syndromal constellation usually make it possible to identify which stream area is affected and whether it is the right or the left hemisphere. Because the motor and sensory signals cross each other on their way from the brain to the extremities, a failure of motor function and sensibility on the right side of the body localizes the insult in the left hemisphere and vice versa. This is referred to as *contralateral symptoms* because the symptoms are produced on the side of the body that is contralateral to the location of the infarction. If, on the other hand, symptoms occur on the same side as the perfusion disturbance, this is referred to as *ipsilateral symptomatology*—in this case, infarction and the symptoms are found on the same side (eg, cerebellar infarction).

Here, a brief overview is provided of the clinical symptoms that are typically encountered depending on the arterial vessel that is involved. Occlusion of the ophthalmic artery or the central retinal artery leads to the sudden unilateral loss of vision. If the anterior cerebral artery is involved, paralysis and a loss of sensation in the contralateral leg occur. Personality changes are also possible. In the case of occlusion of the middle cerebral artery, contralateral hemiparesis (reciprocal hemiplegia) and sensory disturbance in the face and arm typically occur. Aphasia or visual field loss (homonymous hemianopsia) may accompany the symptoms. However, visual field loss (homonymous hemianopsia to the opposite side) may also indicate occlusion of the posterior cerebral artery. Ipsilateral ataxia (impaired coordination of movements) and vertigo are symptoms of occlusion of the cerebellar arteries (inferior anterior cerebellar artery, inferior posterior cerebellar artery). When the brainstem arteries (basilar artery, vertebral artery) are involved, there is typically ipsilateral cranial nerve loss, vertigo and dysarthria, and contralateral sensory and motor hemiparesis.

Hemorrhagic infarction

About 10% to 15% of all strokes are caused by intracerebral hemorrhage.[21] One of the most important risk factors is arterial hypertension. In this case, a predamaged, usually small vessel ruptures. The escaping blood causes an increase in intracranial pressure, with less space available for the brain due to the volume of escaping blood. As a result of increased intracerebral pressure, compression of surrounding brain tissue occurs, leading to loss of function. The typical localization (loco typico) of hypertensive-related intracerebral hemorrhage is the basal ganglia. Characteristic symptoms include sudden headache, as well as nausea and vomiting, in addition to the aforementioned contralateral limitations of motor function or sensitivity.

Basically, it is not possible to conclude from the clinical deficit whether an ischemic or a hemorrhage-related infarction event is present, which has immediate consequences for acute diagnosis and therapy. In addition to arterial hypertension, oral anticoagulation (eg, by means of phenprocoumon [Marcumar, MEDA Pharma] or new oral anticoagulants) and the regular use of acetylsalicylic acid (eg, aspirin) are risk factors for intracerebral hemorrhage. If these patients experience fall events with subsequent neurologic deficits, a possible intracerebral hemorrhage should always be considered.

About another 5% of all strokes are caused by subarachnoid hemorrhage.[6] Typical symptoms include the peracute onset of severe headache (thunderclap headache), nausea and vomiting, and meningeal irritation (painful neck stiffness). In most cases, subarachnoid hemorrhage is caused by congenital or acquired aneurysms that carry a risk of rupture (Fig 2).

Diagnostics

In addition to the medical history, neurologic exam, and laboratory exam (blood count, coagulation, electrolytes, and kidney values), a CT or MRI scan is of particular importance in the acute phase. As mentioned, it is important to differentiate between hemorrhagic and ischemic infarctions, as this is crucial for further therapy. If a patient with a hemorrhagic infarction is subjected to lysis therapy, this may have an adverse effect, including fatal consequences (massive increase in hemorrhage with intracranial pressure). In the CT scan, cerebral hemorrhage is visible immediately, but ischemia is visible only after 2 to 3 hours. In an MRI image, ischemia can be detected early (ie, in the acute phase) in certain sequences (diffusion weighting). Thus, and also due to the more accurate soft tissue imaging, MRI is superior to CT[3,11] but by no means applicable everywhere in emergency situations, because the preparation requires a greater expenditure of time and money, and an MRI machine is not available in all hospitals over 24 hours.

In the post-acute phase, the trigger responsible for the insult should ideally be identified to prevent future strokes after initiating the appropriate secondary prophylaxis. This includes imaging of the neck and intracranial vessels (carotid stenosis, occlusion of intracranial vessels) by duplex sonography or CT angiography, a 24-hour electrocardiogram (atrial fibrillation or other embolizing arrhythmias), an echocardiography (patent foramen ovale as a possible source of paradoxical emboli, pathology of the heart valves), and the clarification of further risk factors by means of laboratory chemical analysis of the blood (coagulation, indications of vasculitis).

Therapy

In addition to ensuring sufficient oxygen supply, securing the airway, optimizing arterial blood pressure, and normalizing blood glucose and electrolyte levels, the goal after an ischemic infarction is to reopen the occluded vessel. Lysis therapy plays an important role here. Provided there are no contraindications, the thrombus can be dissolved using a plasminogen activator (eg, alteplase). Lysis therapy is used in the majority of cases only if symptoms began less than 4.5 hours prior.[10] After 4.5 hours, there is a significantly increased risk of secondary bleeding into the infarcted area. In addition, the systemic inhibition of coagulation can lead to bleeding into the brain and other regions of the body.

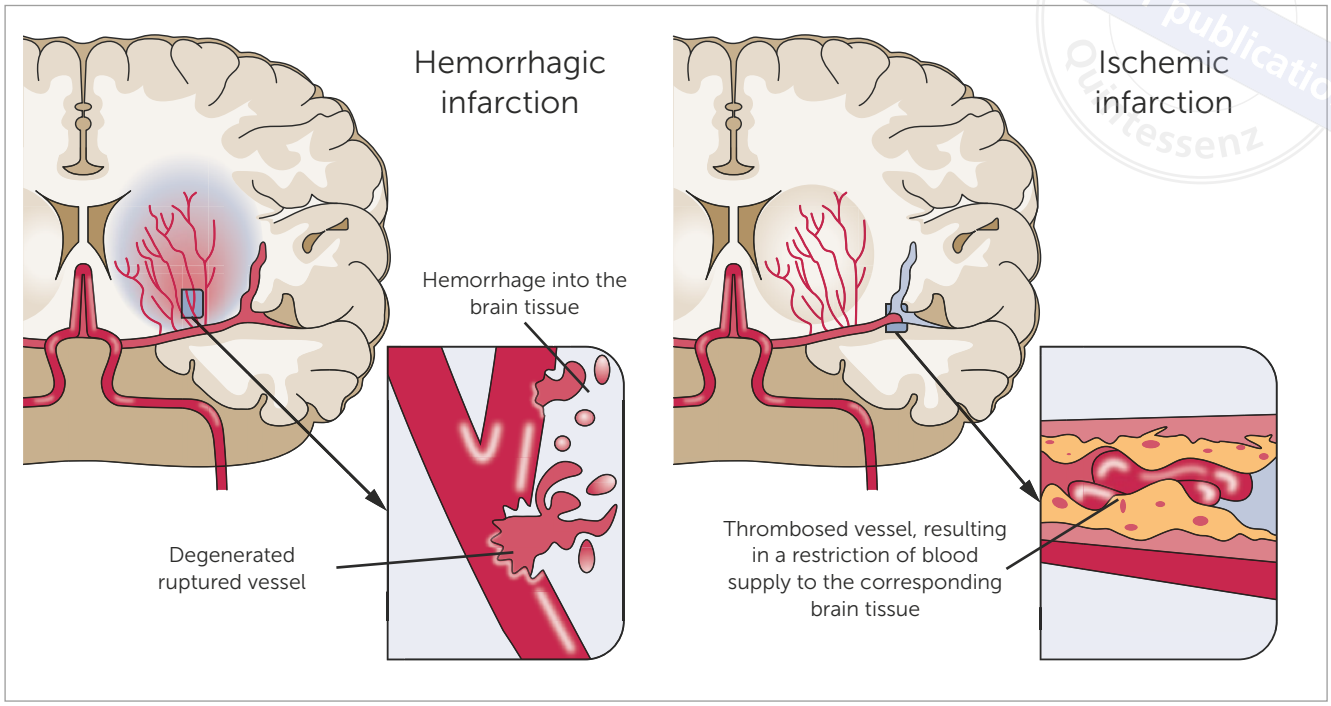

Fig 2 The left half of the image shows a hemorrhagic infarction with hemorrhage into the brain. The right half of the image, on the other hand, shows a vascular stenosis with corresponding thrombus preventing blood supply to the downstream brain tissue. (Modified from: Heart and Stroke Foundation of Canada, www.heartandstroke.ca/stroke/what-is-stroke.)

Whether or not lysis therapy is initiated, another therapeutic step is to prevent cerebral edema. If nerve cells necrosis occurs, this can lead to swelling of the brain. This results in compression of healthy parts of the brain, and as soon as the pressure in the brain exceeds a critical limit, the blood supply to non-infarcted brain areas is also placed at risk. There are various measures to prevent or treat cerebral edema. These include lowering intracranial pressure with osmotic diuretics such as mannitol.[1] Elevation of the upper body and continuous evaluation of the intracerebral pressure are also performed. Pressure can be monitored by inserting a pressure probe into the brain parenchyma. If the patient is intubated, artificial hyperventilation can be helpful, as this causes cerebral vasoconstriction, which lowers intracranial pressure.[1] Surgical decompression (opening the skull bone by drilling holes or partially lifting off the bone) may also provide additional space for the brain.

Mechanical thrombectomy can also be used in the treatment of ischemic infarcts, in addition to thrombus dissolution by lysis.[18] In this case, the thrombus is removed endovascularly from the corresponding vessel via an intraarterial catheter, thus making the vessel pervious again (Fig 3).

Lysis therapy cannot be used in the case of a hemorrhagic infarction, as this would exacerbate bleeding. However, the measures described, such as optimization of blood pressure, monitoring intracranial pressure, and surveillance of the patient, are used for therapy. In addition, neurosurgical intervention may be necessary. This has its place on the one hand in the treatment of cerebral edema, and on the other hand, it may also be necessary to evacuate a hematoma formed by the hemorrhage.

In the case of subarachnoid hemorrhages, other therapeutic options exist. Because vascular malformations (mostly aneurysms) often cause subarachnoid hemorrhage, interventional neuroradiology or neurosurgery can be helpful. Interventional radiology with a catheter can be used to find a path to the vessel that is responsible for the hemorrhage via vascular access in the groin. Once this is reached, small coils can be fired through the catheter. The coils are inserted into the aneurysm and, as it progresses, cause it to thrombose, preventing further blood flow.

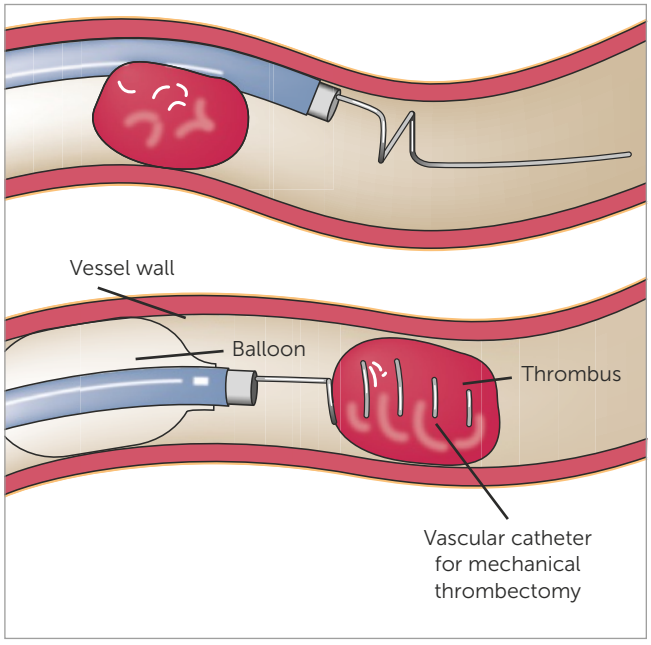

Fig 3 Mechanical thrombectomy is shown via vascular catheter. (Modified from: Stroke AHA Journal, http://stroke.ahajournals.org/content/39/4/1205.)

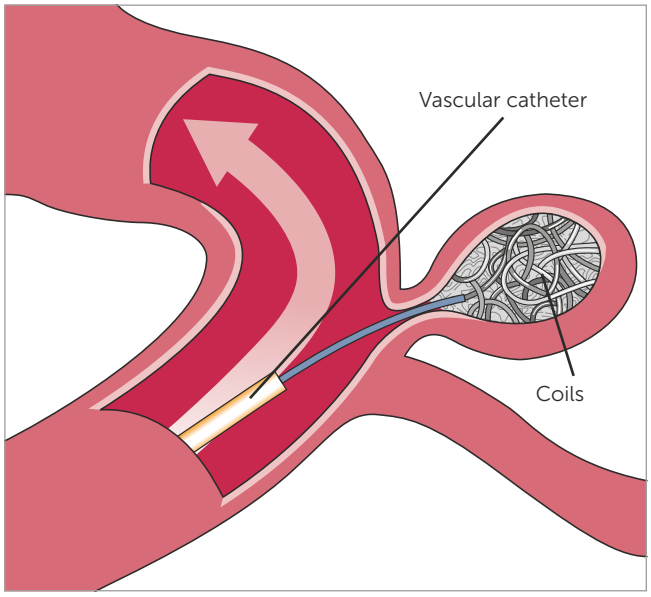

Fig 4 Coils can be inserted into the aneurysm via a vascular catheter. In this way, thrombosis of the aneurysm occurs. Blood subsequently continues to flow through the vessel (arrow), but the aneurysm is not perfused further. (Modified from: Mayfield Clinic, www.mayfieldclinic.com/PE-Coiling.htm.)

Alternatively, the affected vessel can be exposed via a neurosurgical procedure and the aneurysm can be cut off from the blood supply by a clip (Fig 4).

After the initial therapy, a further important step is early rehabilitation in a neurorehabilitation clinic, where patients receive intensive physical and occupational therapy and speech therapy as needed.

Prognosis and prophylaxis

Prognosis

Cerebrovascular events have a high lethality. According to a 2011 American Heart Association evaluation, 1 year after first-time stroke, 28% of men and 32% of women died, compared with 52% of men and 56% of women 5 years after the first stroke. Data are for patients 45 years and older at the time of stroke.[15]

In addition to the localization, the size of the area affected by the ischemia is decisive for the prognosis. The earlier a stroke can be diagnosed and treated, the better the chances of survival and the greater the likelihood that a patient will be able to return to his or her usual daily routine. If a stroke is suspected, immediate admission to a medical center with a stroke unit is indicated so as not to delay therapy. Early rehabilitation plays a particularly important role and should ideally take place in a clinic specializing in neurorehabilitation. The greatest progress is made in the first months after a stroke.[13]

It is of particular importance to prevent further cerebrovascular events, which corresponds to secondary prophylaxis. Stroke patients are at high risk of having another stroke. The probability was reported in a Swedish study to be 13.5% at 1 year, nearly 40% at 5 years, and approaching 55% at 10 years.[8] Another study found a 13-fold increased risk of stroke in TIA patients compared with a healthy control group.[5] The 5-year risk of stroke after TIA was as high as 30%.[5]

Primary prophylaxis

Because, despite all available therapeutic measures, strokes still have a serious prognosis with a high lethality, primary prophylaxis (ie, preventing an infarction from occurring in the first place) is of central

importance. Reducing the risk of stroke primarily involves a healthy lifestyle and avoiding the known risk factors such as, lack of exercise, unhealthy diet, cigarette smoking, and excessive alcohol consumption. Arterial hypertension is one of the most important risk factors for a cerebrovascular event, so it should be treated with medication. Elevated cholesterol levels can be reduced by an appropriate diet or with drugs (eg, statins). Patients with atrial fibrillation must be orally anticoagulated. Otherwise, there is a high risk of cardiac embolism.

Secondary prophylaxis

If a patient has already suffered a stroke or TIA, secondary prophylaxis is required. In addition to the measures described in the previous section, a platelet aggregation inhibitor with acetylsalicylic acid (eg, aspirin) or with clopidogrel (eg, Plavix [Sanofi] in the case of stenoses of brain-supplying arteries) is indicated. Patients with a cardiogenic source of embolism and/or potentially embolizing arrhythmias are usually anticoagulated. Patients who have 70% stenosis in the carotid artery are candidates for carotid endarterectomy.[2] This means that the buildup in the carotid artery is excised or removed in a vascular surgery procedure.

Summary

Cerebrovascular events are frequent and associated with high mortality or later need for care. Primary prophylaxis is therefore of great importance. If a stroke or TIA is clinically suspected, immediate hospitalization for further evaluation is essential, as this can prevent patients from dying or becoming more severely disabled. Sudden weakness, signs of paralysis, sensory disturbances, dizziness, extremely severe headaches, or disturbances of the visual field can also occur in the dental practice and must be clarified immediately. Decisive for further therapy is the differentiation between ischemic and hemorrhagic infarctions, which is performed by means of CT imaging or ideally by MRI.

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Metabolic bone diseases and osteoporosis

Matthias Tröltzsch, Stefanie Kriegelstein, Ursula Hanf, Markus Tröltzsch

Introduction and pathophysiology

Like all tissues, bone is subject to changes with age.[10,12] Genetic predisposition, hormonal changes, biochemical and cellular processes, and reduced functional load (immobility) can lead to an imbalance between bone formation and bone resorption and consequently to a reduction in bone mass.[10,12,16] The changes in bone remodeling[19] are due, among other things, to decreased regeneration of osteogenic cells, increased cell death of osteocytes, protracted healing processes, and differential activity of osteoblasts and osteoclasts.[6,12,16] These processes are referred to as *osteoporosis* and affect both cortical and cancellous bone. Ultimately, osteoporosis leads to a reduction in the mechanical load-bearing capacity of the bones and skeletal system.

Clinically, a distinction is made between primary and secondary forms of osteoporosis. Primary osteoporosis predominantly affects women and usually develops after menopause as a consequence of estrogen deficiency.[17] Sex hormones (estrogens, androgens) appear to have an osteoprotective effect via complicated mechanisms of action.[5,12] In the presence of reduced concentrations of these hormones, osteoclast activity is increased and bone substance is rarefied.[10,12,19] However, recent findings suggest that primary osteoporosis is a multifactorial process, with genetic factors probably exhibiting strong influence.[15] Secondary forms of osteoporosis can be attributed to an underlying disease (Table 1), the treatment of which can also have a positive influence on bone quality.

! The reduction of bone mass and bone stability is called osteoporosis.

A healthy lifestyle and physical activity have a preventive effect against osteoporosis, as do sufficient vitamin D levels.

When osteoporosis occurs, pharmacologic therapy is important to avoid severe consequences.

Table 1 Causes of secondary forms of osteoporosis[4,15]

Metabolic diseases	Other diseases
Type 1 diabetes mellitus	Malabsorption syndromes
Cushing syndrome and associated disorders (pathologically elevated levels of cortisol in the blood)	Anorexia
Hyperparathyroidism (elevated concentrations of parathyroid hormone of various etiologies)	Renal disease (renal osteopathy: lowered calcium levels with the induction of secondary hyperparathyroidism)
Hyperthyroidism	Oncologic diseases
Hypogonadism (decreased production of sex hormones)	

Epidemiology

Osteoporosis is a very common disease that affect over 200 million people.[3,8,11] The disease is significantly more common in women than in men, and the prevalence increases with age.[8] In the mid-2000s, more than 300,000 osteoporosis-associated fracture events were recorded in Germany, generating total costs of more than 5 billion dollars. Each osteoporosis-associated fracture event leads to mobility limitations for patients and is not infrequently associated with loss of independence, increased care requirements, and increased mortality.[11,15,18] The economic cost of the disease is significant and expected to increase. It is therefore all the more astonishing that only a small number of osteoporosis patients receive appropriate treatment, despite the existence of therapeutic indications.[14,17]

Clinical presentation

Osteoporosis may be clinically "silent" over a long period of time.[3] Even morphologic changes in vertebral bodies that are typical of osteoporosis may occur as incidental findings during radiologic diagnosis without clinical correlation (Fig 1). Typical morphologic changes of vertebral bodies result from different force effects on the spine, depending

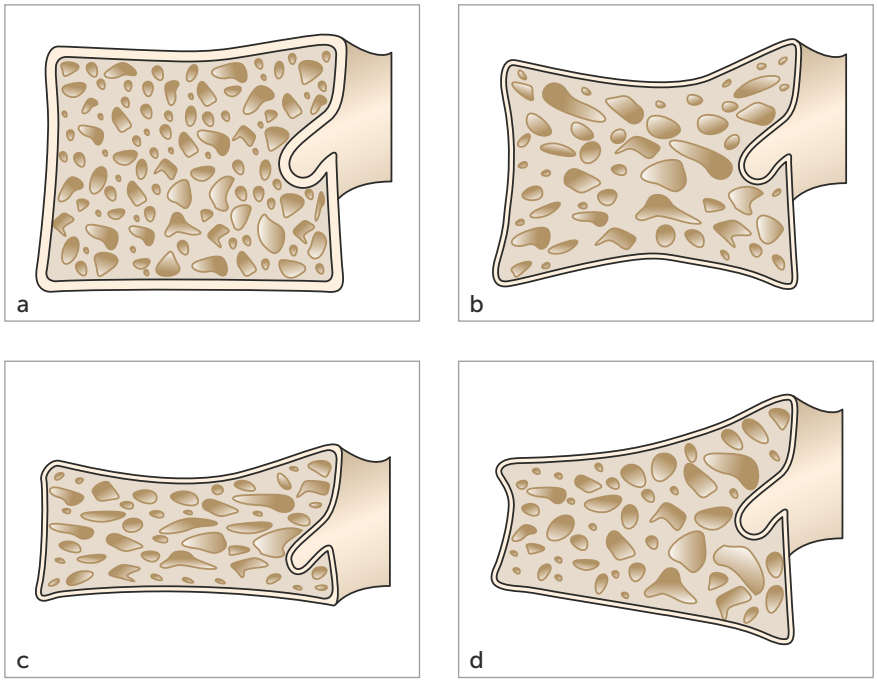

Fig 1 Vertebrae of different morphology—deformation and fractures as late consequences of osteoporosis. *(a)* Normal vertebra with dense cancellous bone. *(b)* Compressed osteoporotic vertebra with depressed cover plates ("fish vertebra"). *(c)* Osteoporotic sintered vertebra ("flat vertebra"). *(d)* Osteoporotic sintered vertebra ("wedge vortex").

Fig 2 Typical forward-bent gait in an osteoporosis patient.

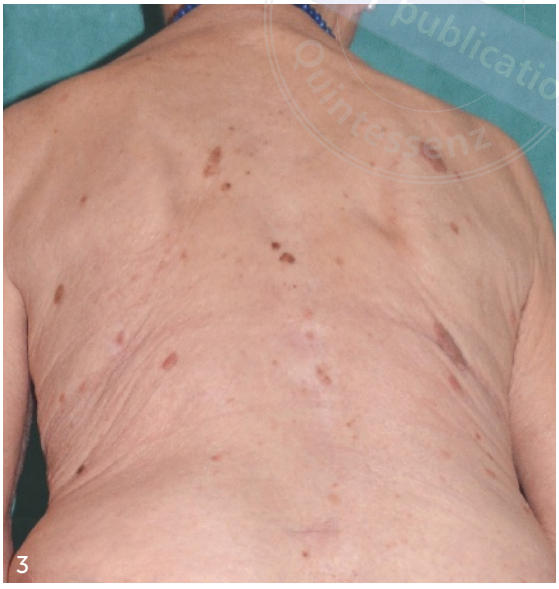

Fig 3 "Fir tree phenomenon" of the back skin. Due to the sintering of the vertebrae caused by osteoporosis, there is an excess of skin in the region of the back, and the vertebrae become curved and deformed.

on the localization.[3,9] However, a clinically manifest fracture may occur in the case of trivial trauma or entirely without significant external force effect.[3,9,17] Osteoporosis-associated fracture events are associated with the typical clinical presentation of bony injuries (pain, functional limitations) and require individually targeted conservative or surgical treatments.[3,4,14,20] Vertebral bodies, the femoral neck, the humerus, and the wrist region are particularly frequently affected by osteoporotic fracture events.[3,14]

Due to morphologic changes in the vertebral body with osteoporosis, kyphosis in the thoracic spine increases ("hump formation"), and there is a loss of height of the axial skeleton.[3] The anatomical changes in the spine also explain other physical changes, such as the stooped gait (Fig 2), the formation of skin folds on the back of osteoporosis patients ("fir tree phenomenon"; Fig 3), and protrusion of the abdomen despite weight reduction[3] (Fig 4).

Diagnostics

Due to the severe reduction in quality of life and the potential increase in mortality after osteoporosis-associated fractures, avoiding fracture events is of primary concern.[3,4,14,17] Diagnostic protocols to determine the ideal initiation of osteoporosis treatment are available. In addition to patient age, these protocols take numerous other risk factors into account and enable the estimation of the 10-year fracture risk.[4,14,17] If this risk exceeds 20% in an individual case, the initiation of basic diagnostics is indicated.[4]

Basic diagnostics include clinical, laboratory, and radiologic examinations[3,4,9,14,17] (Table 2). Radiologic procedures are used to detect currently existing morphologic osteoporosis-associated skeletal changes (Fig 5) and to determine bone density.[9] Bone density is measured primarily by means of dual-energy x-ray absorptiometry (DXA).[7,9] Bone density is measured at several sites in the lumbar spine and proximal femur,[3,7,9] and the lowest value determined is compared with reference collectives. Two values are of importance here[9]:

1. T-value: Comparison of the measured bone density value with bone density values of a comparison collective of young adults
2. Z-value: Comparison of the measured bone density value with bone density values of a comparison collective of adults of the same age

The relevant value used for diagnostics is given in standard deviations from the mean. This results in a dimensionless number that provides information on bone density [9,14] (Table 3). The summary of the results

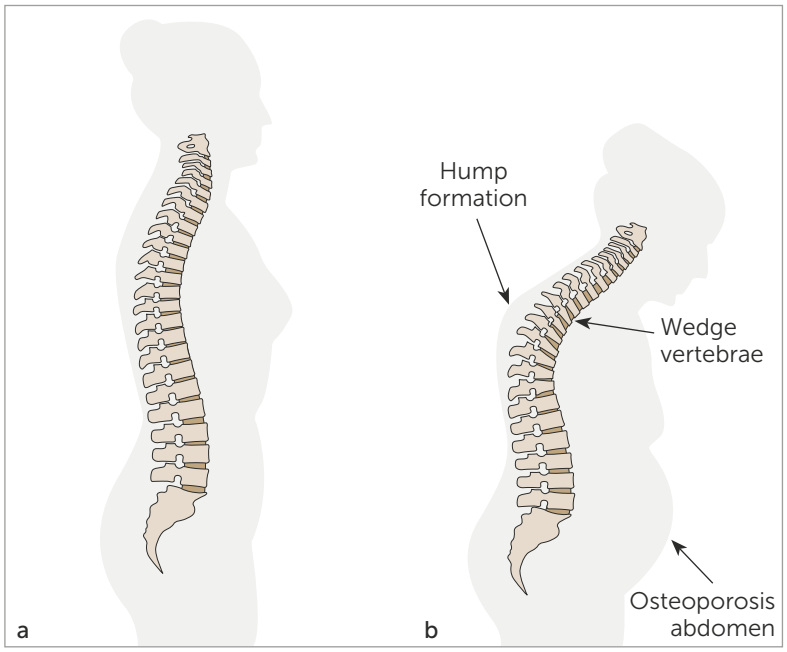

Fig 4 Morphology of the axial skeleton. *(a)* Normal physiologic spine shape with regular morphology of the vertebral bodies. *(b)* Shape of the spine in advanced osteoporosis with increased kyphosis in the thoracic region.

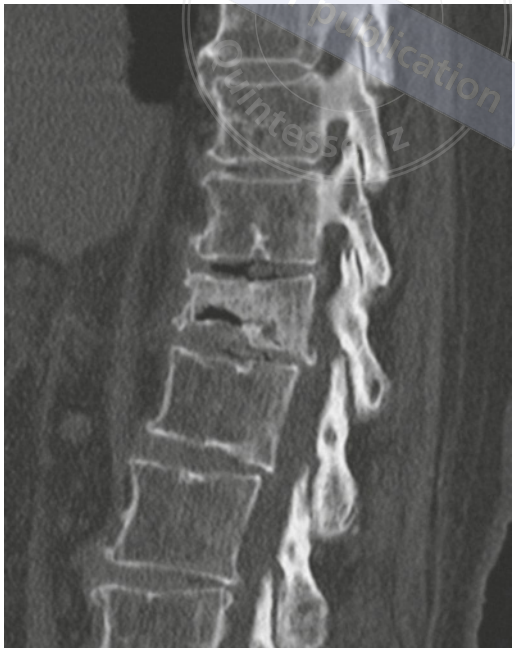

Fig 5 Fresh vertebral fracture of an osteoporotic wedge vertebra (sagittal CT section).

of the basic diagnostics may vary depending on the age of the patient, concomitant diseases, the presence of fractures, or specific drug intake and lead to the initiation of osteoporosis drug therapy.[3,4,14] It should be mentioned that in the presence of typical osteoporosis-associated fractures (clinical and/or radiologic), the indication for osteoporosis drug treatment may exist under certain circumstances even without bone densitometry.[14] Thus, even with T-values greater than –2.5, there may be a need to initiate specific therapy according to the new recommendations.[4] The range of indications was chosen so that the benefits of treatment clearly outweigh the possible side effects.[17]

Table 2 Components of the anamnesis and the clinical and laboratory examinations as part of the basic diagnosis of osteoporosis (selection)[3,4,15]

Medical history and clinical examination procedures	Laboratory chemistry tests
Detailed patient interview and collection of information on ongoing medication use, underlying diseases, cognitive and physical performance, fall and fracture events that have occurred, and/or constitutional changes	Calcium and phosphate levels
Determination of height, weight, body mass index (BMI)	Kidney function values
Examination of the skeletal system and musculature (mobility, pain, strength)	Detection of bone metabolism (eg, alkaline phosphatase)
Investigation of the tendency to fall by means of suitable tests	Blood count and inflammation values
	Thyroid function
	If necessary, further metabolism-relevant examinations (eg, determination of sex hormone levels, vitamin D_3, etc)

Therapy

For the primary prevention of osteoporosis, physical activity, healthy lifestyle (balanced diet, abstinence from nicotine), the avoidance of being underweight (body mass index > 20), and sufficient intake of calcium and vitamin D are generally recommended.[1,4] It is advisable to give preference to sufficient calcium intake from food (calcium-containing foods and mineral water) over supplementation, because overdose can lead to undesirable side effects (cardiovascular problems, kidney stones).[1,17] In the case of inadequate sun exposure, the prescription of vitamin D preparations should be considered.[1] Especially in northern latitudes, vitamin D deficiency is a widespread phenomenon.[1] A decrease in the incidence of fractures in patient collectives with regular vitamin D levels emphasizes the importance of supplementation.[14,17]

Various drugs are available for the treatment of manifest osteoporosis[13,17,18] (Table 4). The goals of pharmacotherapy are to increase bone density, prevent further bone loss, and reduce fracture rates.[3,17] It is well documented that the patient population treated with bisphosphonates and denosumab have significantly lower fracture rates compared with non-treated patients.[2,13,18] Teriparatide, which is approved only for special indications, appears to provide a significant improvement in bone quality.[2] Further comparative studies to more accurately assess the efficacy of the aforementioned pharmaceuticals are currently underway.

Table 3 Expected bone density at different T values[3,9,17]

T-value	Bone density
> −1	Normal
−1 to > −2.5	Osteopenic
≤ −2.5	Osteoporotic

In the case of manifest osteoporotic fractures, trauma treatment may be necessary.[14,15] This mainly concerns fractures of the extremities (eg, femoral neck, upper arm). In the spine, surgical stabilization can also be performed in extreme cases of vertebral body compression fractures or therapy-resistant pain[14,15] (Fig 6).

Patients with symptomatic osteoporosis and/or osteoporosis requiring therapy must be included in a recall program to ensure that new fractures or functional limitations can be quickly identified and treated.[4] The initial duration of drug therapy should not be less than 12 to 24 months.[4] Thereafter, further continuation of treatment depends on the individual fracture risk, which should be reevaluated periodically during the follow-up program.[4,17] It is recom-

Table 4 Pharmaceuticals approved for the treatment of osteoporosis[2,4,13,17]

Pharmaceutical	Mode of action	Adverse drug reactions
Bisphosphonates	Inhibition of osteoclast function via intracellular mechanisms of action	Osteonecrosis of the jaw, gastrointestinal problems, acute phase reaction, atypical femur fractures
Denosumab	Antibody against RANKL, inhibition of osteoclast function and differentiation by inhibition of RANKL action	Osteonecrosis of the jaw, nonspecific infections, joint pain
Raloxifene (selective estrogen receptor modulator)	Tissue-specific unfolding of agonistic and antagonistic estrogen effects (possibly lowers breast cancer risk)	Sinusitis, flushing, nonspecific pain symptoms, liver elevation
Strontium ranelate	Inhibition of bone resorption and increase of bone density	Gastrointestinal side effects (rare): DRESS (drug rash with eosinophilia and systemic symptoms) syndrome
Teriparatide (recombinant parathyroid hormone [PTH])	Osteoanabolic effects with intermittent application	Nonspecific side effects; application time limited due to unclear association with malignancies in animal studies

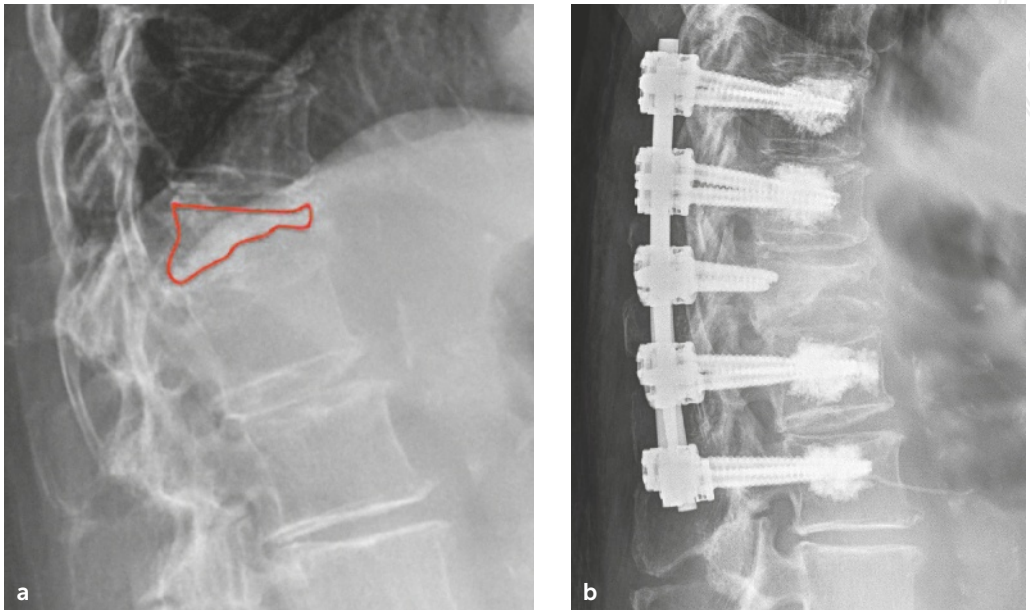

Fig 6 *(a)* Fracture of a thoracic vertebra with wedge vertebrae formation (marked red, lateral spine). *(b)* Dorsal stabilization of the vertebral fracture in Fig 6a.

mended that decisions regarding possible discontinuation of therapy take into account that the efficacy of all the aforementioned pharmaceuticals, with the exception of bisphosphonates, is likely to decline rapidly after discontinuation due to their limited half-lives, and a "rebound" of symptoms must therefore be expected.[4,10]

Conclusion

Bone not only performs mechanical functions in the human body but also plays a crucial role as a metabolic organ. Like all organ systems, bone is not protected from pathologic influences, wear and tear, and aging processes. In addition to osteoporosis, there are numerous other metabolic bone diseases, some of which are congenital (see Table 1), but their diagnosis and treatment are reserved for specialists. With the exception of bony injuries and age-related changes in bone structure and mechanics, pathologic processes of the bone are fortunately rare and are usually secondary consequences of other diseases. With a healthy lifestyle and sufficient physical activity, nothing stands in the way of a functional skeleton into old age.

Summary

The skeletal system is affected by the aging process. Various influences and factors lead to a reduction in bone mass and stability. These processes are referred to as *osteoporosis*. Clinically, the course is often initially asymptomatic, but relatively small amounts of force can lead to bone fractures with a significant impact on the independence and quality of life of the affected patients. In view of the several million osteoporosis patients in Germany alone, this is a medical and economic problem that should not be underestimated. Osteoporosis can be unmasked with targeted diagnostics so that successful treatment and the avoidance of late complications are possible. The aim of this section is to present the pathophysiology, epidemiology, clinical presentation, and therapy of osteoporosis, as well as other diseases of bone metabolism.

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CHECKLIST

Metabolic bone diseases and osteoporosis

Keep this list easily accessible or attach it to your quality management documents.

Osteoporosis	Decrease in bone density, rarefaction of cortical bone and cancellous bone with decreased biomechanical strength of the bone, and increased risk of fracture
Clinical presentation	Often asymptomatic initially but later involves reduction in height, hunchback formation, stooped gait, "fir tree phenomenon" in the back, and protruding belly
Diagnostics	Basic diagnostics if indicated: medical history, clinical examination, radiography, bone densitometry, laboratory diagnostics
Therapy	Basic therapy: adequate calcium and vitamin D_3 intake, physical activity, increase in gait stability, physiotherapy; pharmacotherapy: mainly bisphosphonates and denosumab
Complications	Fractures, pain, loss of independence, increased mortality
Particularly affected areas	Spine, proximal femur, humerus, distal radius

Lung infection: Basic pulmonary physiology and pathophysiology and the diagnosis and therapy of pneumonia

Matthias Tröltzsch, Markus Tröltzsch, Stefanie Kriegelstein

! Pneumonia is one of the most common infectious diseases.

Recognizing pneumonia symptoms in patients and employees and promptly referring them for medical treatment is the dentist's responsibility.

Introduction and epidemiology

With about 500,000 new cases per year, pneumonia is one of the most common infectious diseases in Germany.[10,11] Because about 200,000 of these pneumonia patients require inpatient treatment during the course of the disease, pneumonia is also of enormous economic importance.[11] Although pneumonia can occur at any age, elderly patients are particularly frequently affected.[10] Approximately half of all persons requiring either outpatient or inpatient treatment for pneumonia are older than 65 years.[1] Pneumonia is the infectious disease associated with the highest mortality.[3] Mortality increases with increasing age of onset.[11] Pneumonia acquired in the hospital is the second most common nosocomial infectious disease after urinary tract infections, with a very high complication rate.[6]

Anatomical and physiologic basics

The physiologic tasks of the lungs include the exchange of gases between blood and respiratory air, as well as the regulation of the acid-base balance of the body.[8] One of the largest organs in the human body, the lungs—protected by the ribs—are located in the thorax together with the mediastinal organs and the heart (Fig 1). The lung is a bilateral organ with three lobes on the right and two lobes on the left. The lung lobes are divided into segments.[8] The functional unit of the lung is the alveolus (Fig 2). The alveoli are located at the end of the bronchial tree, through which the air flows first via the trachea, then via large and small lobes and segmental bronchi into smaller and smaller bronchioles and finally into the fine alveoli.[7,8] The wafer-thin alveolar walls are located in close proximity to the finest capillaries.[8] This enables gas exchange between the blood vessels and pulmonary alveoli via diffusion.[9]

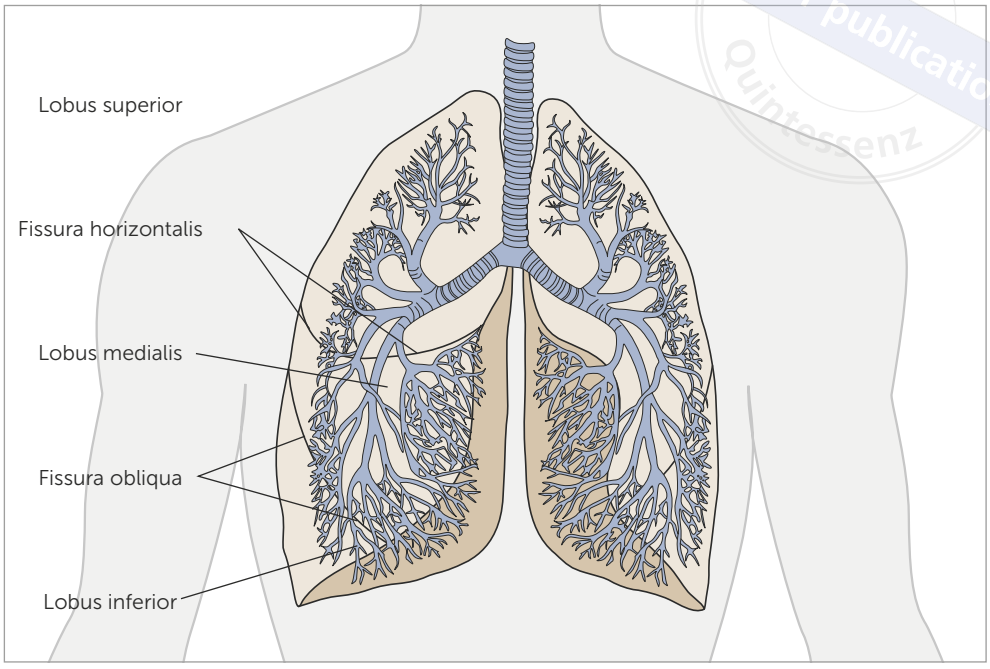

Fig 1 Schematic anatomical representation of the bronchial tree and the lung lobes in the thorax (modified after Rohen[8]).

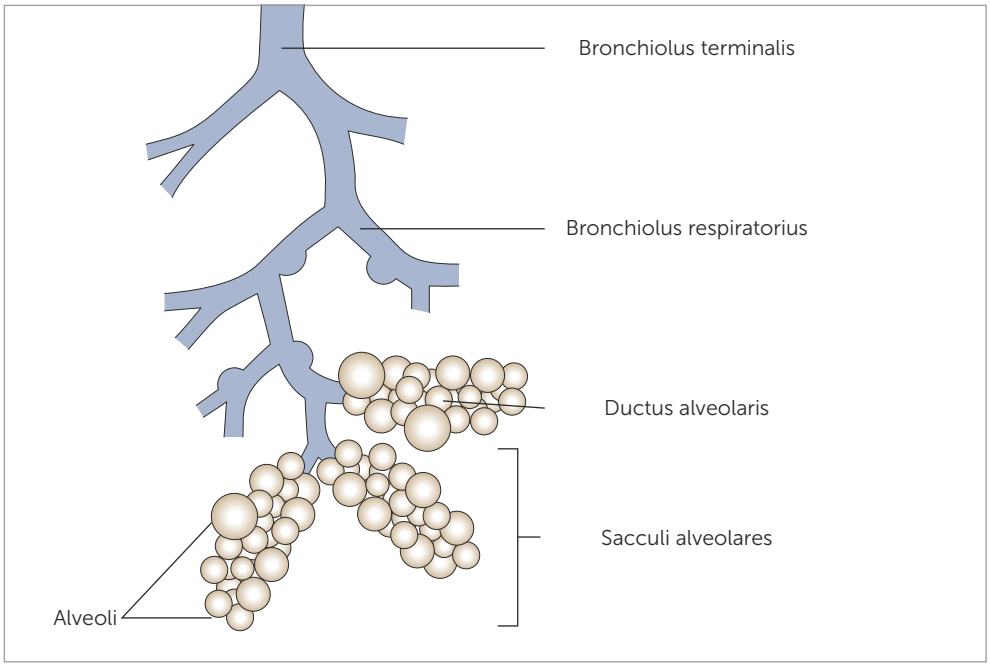

Fig 2 The smallest functional unit of the lung is formed by the alveoli, in which gas exchange takes place. They are located at the end of the bronchial tree in the pulmonary acini. (From Rohen.[8])

The lungs undergo morphologic and histologic changes throughout life. For example, the weight and elastic restoring force of the lungs decrease, resulting in reduced lung function.[5] This causes reduced physical activity and, together with changing dietary habits and altered immunity that comes with age, increases the susceptibility of the elderly to respiratory infections.[5] In addition, cigarette smoking induces consequential structural damage to the lungs. Females are much more affected by these influences than males.[5]

Pathophysiology

Pathogens find their way into the lungs via various routes. Aspiration of pathogens from the mouth and pharynx is of particular importance.[10] Pathogens can proliferate in the pulmonary alveoli and induce an infection with an inflammatory reaction.[3] The resulting inflammatory exudate stands in the way of gas exchange in the alveoli by prolonging the diffusion distance or making it insurmountable.[9] The larger the lung portions affected by the diffusion disturbance, the more severe the effects on the oxygen partial pressure in the peripheral blood.[9] Incipient hypoxia initially leads physiologically to an increase in respiratory rate as a compensatory mechanism. In addition, the Euler-Liljestrand mechanism reduces the perfusion of hypoxic lung areas, with the aim of diverting the blood to areas with better gas exchange.[9] When a critical number of the lung alveoli are damaged by fluid accumulation, the aforementioned compensatory mechanisms no longer have sufficient effect, and hypoxia occurs with direct clinical effects (eg, cyanosis).[3]

Classification and pathogen spectrum

Today, a fundamental distinction is made between community acquired and nosocomial-acquired pneumonia.[1,3,4,10,11] This differentiation is based on the pathogen spectrum and the courses of the diseases.[10] Community acquired pneumonia is mostly caused by Gram-positive pathogens (most commonly *Streptococcus pneumoniae*); On the other hand, Gram-negative pathogens play a role in nosocomial-acquired pneumonia as part of aerobic-anaerobic mixed infections.[11] From a clinical point of view, pneumonia can still be classified according to typical and atypical courses.[3] The typical course is characterized by a sudden onset of illness with high fever and putrid sputum, whereas atypical courses (pathogens: *Legionella*, *Mycoplasma*, etc) often manifest themselves over a protracted course with only mild fever and headache and muscle pain.[3] Pneumonia caused by viruses also plays a role. Viral pneumonia can be complicated by bacterial superinfection.[10]

Diagnostics

The first step in the diagnosis of pneumonia is acquiring the patient's medical history. This provides information about the type of pneumonia (outpatient or nosocomially acquired). This important information provides decisive clues as to the therapeutic steps that will be necessary later. If pneumonia occurs without a temporal connection to a hospital stay or within a maximum of 48 hours after admission to a hospital, it is referred to as *community acquired pneumonia*.[3] The distinction between community acquired and nosocomial-acquired pneumonia is sometimes blurry.[1] In addition, a precise medical history can identify any risk factors that are associated with a more severe course of pneumonia. These include chronic internal diseases (chronic cardiac and renal insufficiency, structural lung diseases, liver cirrhosis), tumor diseases, alcohol abuse, and underlying neurologic diseases.[4,10]

The clinical examination and the medical assessment of the patient are also of great importance. In addition to the general medical examination (Table 1), percussion (Fig 3) and auscultation (Fig 4) of the thorax are performed. Muffled tapping sounds and rattling, and possibly also reduced or even absent breath sounds over certain parts of the lungs, may be signs of inflammatory exudates located there.[2]

Another important step is the imaging examination. For the exact diagnosis of pneumonia, a radiograph of the thorax (preferably in two planes) must be obtained.[3,7] The detection of infiltrates in the thoracic radiograph (Fig 5) is a main criterion for the diagnosis of pneumonia.[3,7] In case of doubt, a CT scan of the thorax should be considered. In addition, an ECG, laboratory tests (eg, to determine the leukocyte count, the level of C-reactive protein, and, if necessary, the procalcitonin concentration and the partial pressure of oxygen), and possibly the collection of sputum, bronchial secretions, and blood cultures are recommended.[3,4] Only if there is a well-founded suspicion of specific pathogens (eg, travel history!) must further serologic and urine tests be performed, if necessary.[3,11]

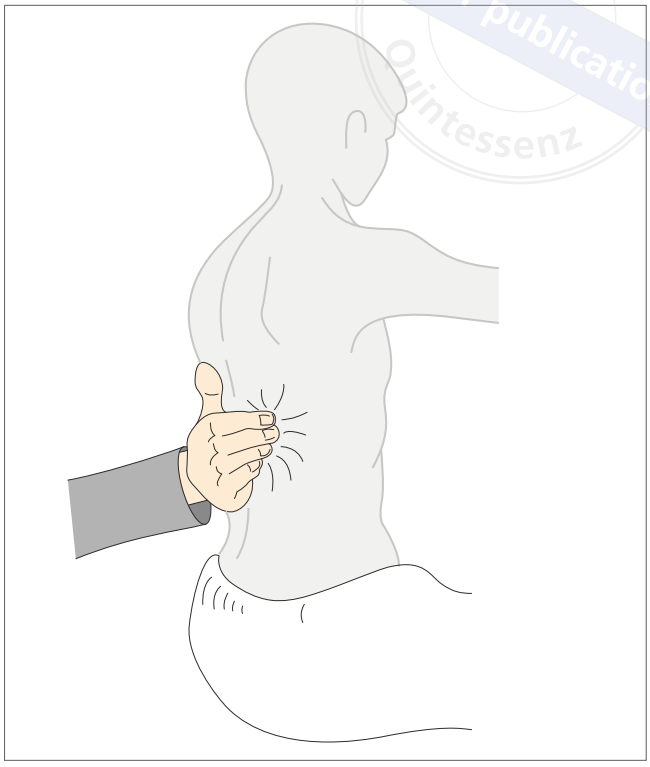

Fig 3 Schematic representation of a percussion examination of the lung, which should always be performed as a side-by-side comparison. (Modified from Dalicho.[2])

Table 1 Measures of the general medical clinical examination performed on patients with suspected pneumonia

Investigation step	Necessary instruments	Information gain
Inspection of the face and thorax	–	Information about any cyanosis, dyspnea, or orthopnea
Measurement of body temperature	Clinical thermometer	Hyperthermia as a sign of a possible immune reaction; hypothermia as a possible sign of an incipient sepsis
Blood pressure measurement	Blood pressure cuff	Hypotension in infectious disease may indicate the onset of sepsis
Inspection of skin	–	Search for exanthema or redness/ hemorrhage as possible evidence of septic streaks
Neurologic examinations/reflex status	Reflex hammer	In advanced stages, infectious diseases may cause limitations in neurologic function
Palpation of the abdomen	Bimanually	Seldom is the cause of the reduced general condition clear at the initial patient presentation, so possible causes can be sought or ruled out
Auscultation of the heart	Stethoscope	Information on heart rhythm and function
Measurement of oxygen saturation	Pulse oximeter	Information on the degree of disturbance of gas exchange—possible indication for immediate inpatient treatment in the event of poor values

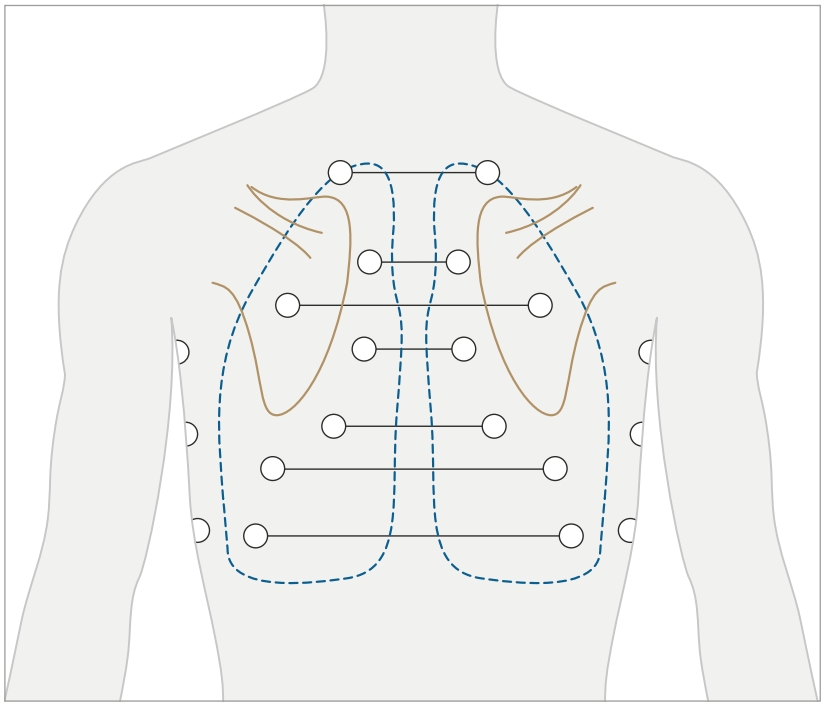

Fig 4 Schematic representation of the auscultation points of the lung, which must be selected in such a way that, as far as possible, all parts of the lung (posterior and anterior) are recorded in a lateral comparison. (Modified from Dalicho.[2])

Table 2 Limits and divisions of the CRB-65 index

Clinical parameter	Limits
C = Confusion	0 = not present; 1 = present
R = Respiratory rate	0 = < 30/min; 1 = ≥ 30/min
B = Blood pressure	0 = ≥ 90/60 mmHg; 1 = < 90/60 mmHg
(Age)	0 = < 65 years; 1 = ≥ 65 years

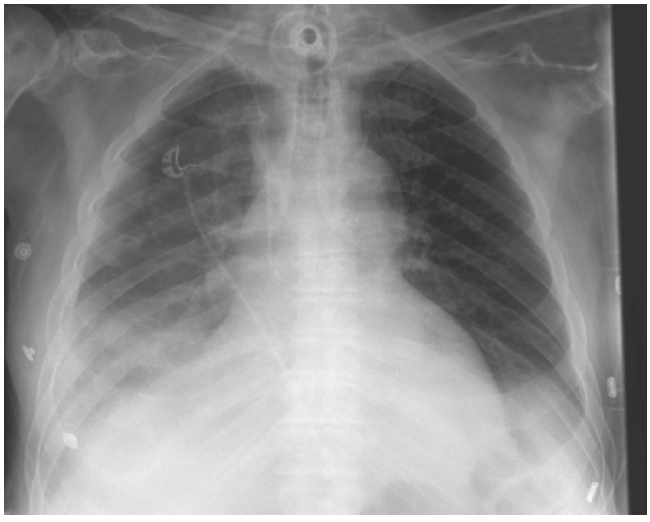

Fig 5 Radiography of the thorax (anteroposterior beam): Pneumonic infiltrates can be seen in the area of the right lower lobe of the lung.

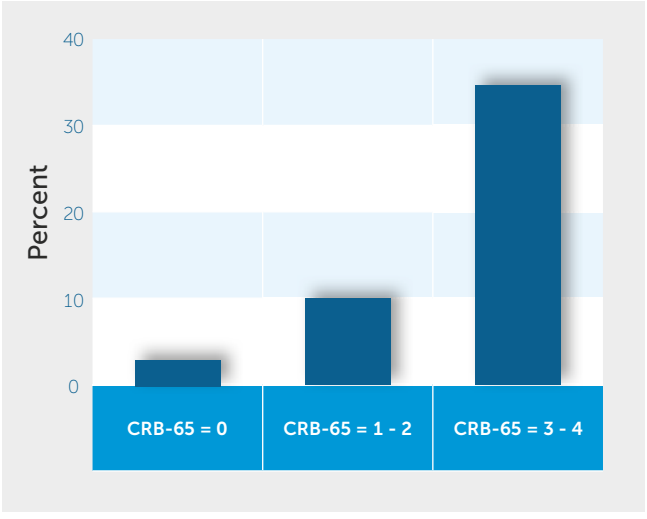

Fig 6 Mortality from pneumonia as a function of CRB-65 value in percent.

The mentioned examinations are used to determine an important index that is validated for the prognosis of pneumonia and provides information on the therapeutic procedure—the CRB-65 index (Table 2; Fig 6). At a CRB-65 value of 0, outpatient treatment of pneumonia can be performed, whereas at a value of 1, inpatient admission and monitoring should be considered.

Therapy

The therapy for pneumonia is essentially based on the CRB-65 value. In any case, antibiotics should be administered at an early stage. The drugs of first choice for community acquired pneumonia are aminopenicillins (eg, amoxicillin in a weight-adjusted dose of 1-1-1).[4] Resistance of the underlying pathogens currently plays only a minor role in community acquired pneumonia. The duration of antibiotic therapy is based on the clinical manifestations (reduction of fever, increase in oxygen saturation, stability of circulatory parameters, slowing of respiratory rate) but should not be less than 5 days.[4,10] Patients suffering from pneumonia require close clinical monitoring even during outpatient therapy. In case of deterioration, hospitalization may be appropriate.

The therapy of nosocomial pneumonia is extremely complex (sometimes requiring intensive medical measures) and is not discussed in detail here.

Prevention

The influenza vaccination offered annually provides effective protection against pneumonia, which can follow influenza infections as a superinfection.[4,10] In addition, pneumococcal vaccination appears to effectively reduce the incidence of pneumonia associated with *S pneumoniae* (the most common causative agent of community acquired pneumonia). It is regularly recommended by the Permanent Vaccination Commission of the Robert Koch Institute for children.[4,10] Adults at increased risk of exposure to pneumococci (nurses, physicians, persons with social occupations) also benefit from this vaccination.

Conclusion

Pneumonia is a very common but serious infectious disease. Pneumonia is technically a subtype of pneumonitis (an inflammatory change in the lung tissues) but with the involvement of pathogens and accumulations of inflammatory purulent exudate in the pulmonary alveoli. The term *pneumonitis* also includes other inflammatory changes of the lung tissues that are not bacterial or viral.

Despite its prevalence in elderly patients, people of any age can contract pneumonia. Clinically, pneumonia usually begins nonspecifically with fever and general malaise but occasionally also presents with headache and muscle pain. Thorough clinical and radiologic diagnosis and early antibiotic therapy are the main pillars in the treatment of community acquired pneumonia. In many cases, an unfavorable course can be averted by early consultation with a physician, a precise medical history, and clinical and instrumental diagnostics. The aim of this section was to explain not only the essential anatomical principles of the lungs and the bronchial system but also the pathophysiology, classification, diagnosis, and therapy of the common clinical presentation of pneumonia.

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CHECKLIST

Pneumonia

- Pneumonia is one of the most common infectious diseases in Germany.
- People of older age are affected more often than younger people.
- In addition to the medical history and a detailed general and organ-related clinical diagnosis, radiographs of the thorax are among the basic diagnostic tools.
- Antibiotics should be administered at an early stage for therapy.