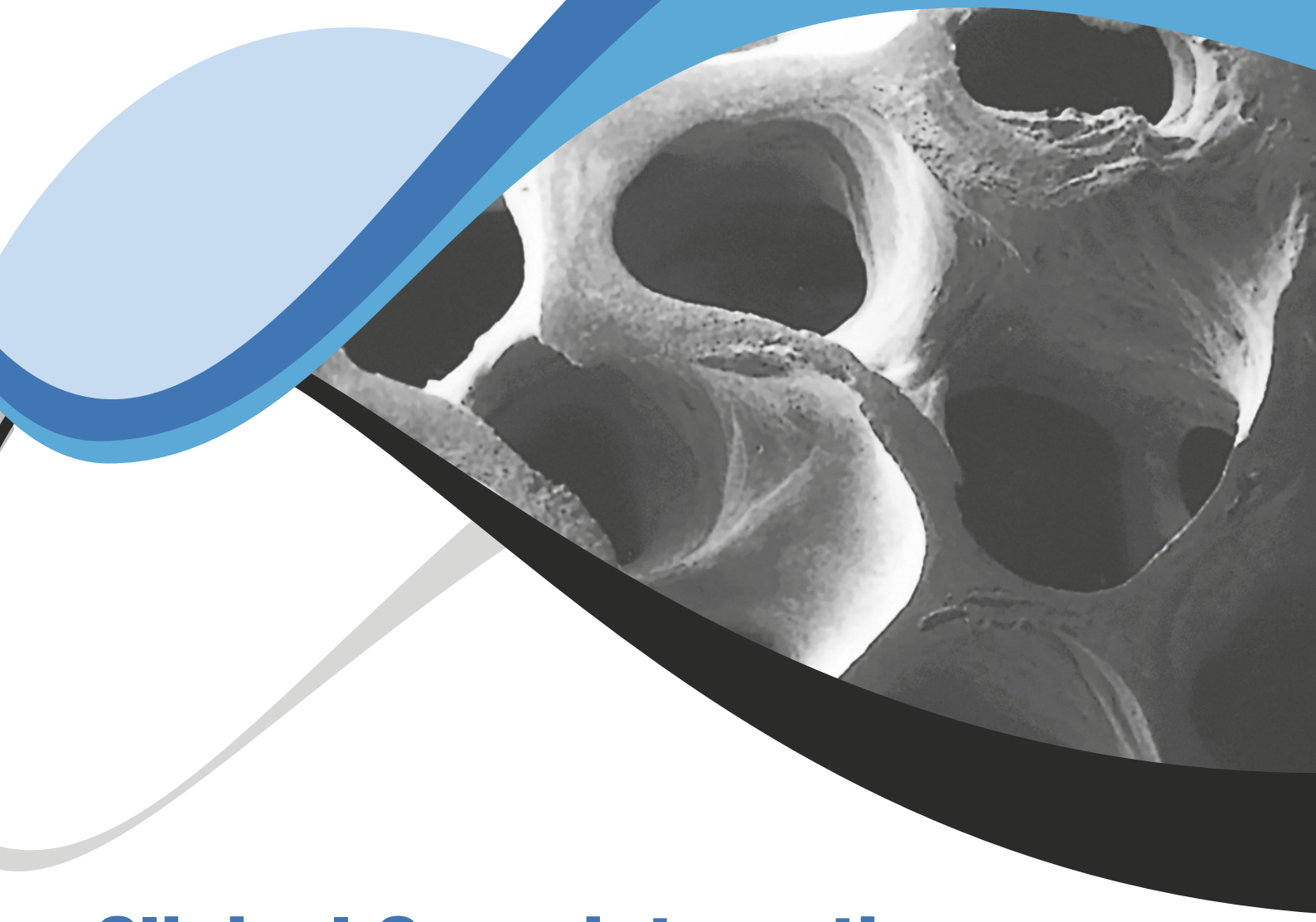
Massimo **SIMION**



Clinical Osseointegration and Bone Regeneration

QUINTESSENCE PUBLISHING

MASSIMO **SIMION**

CLINICAL OSSEOINTEGRATION AND BONE REGENERATION

To Lorenzo, who wishes to enjoy himself as a free man
in life and profession
as I have done and am doing

One book, one tree: In support of reforestation worldwide and to address the climate crisis, for every book sold Quintessence Publishing will plant a tree (https://onetreeplanted.org/).

First published as OSTEOINTEGRAZIONE CLINICA E RIGENERAZIONE OSSEA in Italian in 2022
by Quintessenza Edizioni, Italy.

How do you read a QR code?
Inside this book, you will find QR codes linked to the relevant videos. To access these videos, you will need to scan the QR code with the camera of your mobile device. Frame the QR code with the camera of your smartphone or tablet, and follow the prompts to view the video.

A CIP record for this book is available from the British Library.
ISBN 978-1-78698-141-7

QUINTESSENCE PUBLISHING
DEUTSCHLAND

Quintessenz Verlags-GmbH
Ifenpfad 2–4
12107 Berlin
Germany
www.quintessence-publishing.com

Quintessence Publishing Co Ltd
Grafton Road, New Malden
Surrey KT3 3AB
United Kingdom
www.quintessence-publishing.com

Translation: Alexandra Rendon, Bologna, Italy
Editing, Layout, Production and Reproduction: Quintessenz Verlags-GmbH, Berlin, Germany

Printed and bound in Croatia by Graficki zavod Hrvatske d.o.o.

MASSIMO **SIMION**

CLINICAL OSSEOINTEGRATION AND BONE REGENERATION

COAUTHORS:
FABIANA **ALLEVI**
MARIO **BERETTA**
FABIO **BERNARDELLO**
FEDERICO **BIGLIOLI**
PAOLO **BOZZOLI**
ELENA **CANCIANI**
CHRISTER **DAHLIN**
DANIELE **DE SANTIS**
CLAUDIA **DELLAVIA**
LUCA **FERRANTINO**
FILIPPO **FONTANA**
ELEONORA **IDOTTA**
MICHELE **MAGLIONE**
CARLO **MAIORANA**
MARCO **MORRA**
MYRON **NEVINS**
PIER FRANCESCO **NOCINI**
RICCARDO **NOCINI**
STEFANO **PIERONI**
ALBERTO **PISPERO**
ROBERTO **PISTILLI**
ALESSANDRA **SIRONI**
GIOVANNI **ZUCCHELLI**

QUINTESSENCE PUBLISHING

Berlin | Chicago | Tokyo
Barcelona | London | Milan | Paris | Prague | Seoul | Warsaw
Beijing | Istanbul | Sao Paulo | Zagreb

CONTENTS

CONTENTS

CONTENTS

CONTENTS

CONTENTS

CONTENTS

PRESENTATION

This timely text presents clinicians with significant, interesting, and new information in the field of implant dentistry. Osseointegrated implants, introduced by P.I. Brånemark, allowed for predictable dental implant therapy and changed the scope of treatment for the replacement of teeth in edentulous patients.

This comprehensive textbook is a concerted endeavor to explore the clinical efforts of all dental implant surgeons. It brings to light the improvements and progress made by clinicians over a period of more than 30 years.

The editor has engaged surgical and restorative experts for the solutions they have found for their patient's problems.

Massimo Simion and I discovered one another in 1990, when he invited me to Milan as a keynote speaker at the first Italian Academy of Osseointegration. He shared his early clinical cases on guided bone regeneration, and I invited him to join our research and clinical courses at the Institute for Advanced Dental Studies and to present his significant findings at the International Symposium in Boston. We have continued to share our research results, including at the 2022 International Symposium, where we introduced new techniques and biomaterials for augmenting the alveolar process to successfully support osseointegrated implants. Successful implant therapy depends upon proper diagnosis and treatment planning to support osseointegration.

These chapters describe the relevant anatomy and surgical techniques for the treatment of both partially and completely edentulous patients. Long-term cases provide examples of the careful decisions around diagnosis and treatment planning, as well as how to engage patient understanding and cooperation.

The text also explores the developments in implant dentistry. Immediate implant placement has expanded into immediate implant loading. Guided implant surgery opens the door to future possibilities. Finally, no leading surgeon can avoid all complications. This text covers the management of complications, including the practical resolution of peri-implantitis.

Every dental team that performs bone augmentation procedures and/or implant rehabilitation should have this text in their library and become familiar with its wisdom on patient care before, during, and after treatment. It is a fabulously complete work, and I congratulate the editor, Massimo Simion, as well as the contributors.

Myron Nevins

INTRODUCTION

My collaboration with Quintessence Publishing began in the early 1990s. In those days, world dentistry was living extraordinary years: Osseointegration techniques according to the Brånemark system were spreading like wildfire in all continents, radically reshaping the therapeutic approach to partially and completely edentulous patients, and guided tissue regeneration was rapidly extending from periodontal applications to peri-implant bone regeneration.

I was fortunate to be able to participate in the development of these two extraordinary world-changing events at a very young age. My first publication on guided bone regeneration (GBR) was published in the International Journal of Periodontics and Restorative Dentistry in 1994, and since then, I have consistently collaborated with Quintessence Publishing, both to publish scientific articles and to participate as a speaker at every Symposium on Periodontics and Restorative Dentistry in Boston, chaired by Myron Nevins.

For over 20 years, we have been discussing the idea of a book on osseointegration and bone regeneration, but only now did I finally tackle the project. My hesitation stemmed from the fact that writing such a far-reaching book would require an immense amount of work, with the danger of the writing never ending. The greatest risk was that once the book was finally finished and published, it would already be overtaken by the rapid evolution of scientific knowledge. I didn't want to write a book and, once finished, wish that I had done it differently.

Today, the speed at which knowledge is being acquired has slowed considerably compared to the first 20 pioneering years. Of course, we learn to use new digital technologies on a daily basis, which makes our work easier and more precise, but the biologic principles and surgical techniques of osseointegration and GBR are essentially equivalent to those developed between the 1980s and 1990s. In the end, I felt that writing this book was finally feasible based on the wealth of information that is now available and, presumably, its stable application for a long time, or perhaps forever.

My aim was to produce a work available to everyone—one that could be used as a textbook by students of dentistry and by those approaching osseointegrated implantology techniques for the first time but also useful for experts wanting to refine their techniques for bone regeneration and the treatment of peri-implant soft tissue in even the most complex esthetic cases. To accomplish this, I have faithfully followed the syllabus of my university course for students and the annual course that I have been holding for implant dentists for more than 20 years. All the concepts and clinical cases illustrated during these courses are presented here.

As Myron Nevins says, "*No one can be an expert in all subjects. Only presumptuous ignoramuses consider themselves such.*" For this reason, I have drawn on the help of the researchers and clinicians with whom I have collaborated most closely over the past 30 years.

These voices are joined by some emerging young people of great talent, and certainly bright futures. To all of them goes my heartfelt thanks. I also want to thank all the staff at Quintessence Publishing Italia for their patience and dedication during the production of this book.

Massimo Simion

CURRICULA

MASSIMO SIMION

Degree in Medicine and Surgery at the University of Milan in 1979. Specialized in Dentistry and Dental Prosthetics at the same University in 1981. Associate Professor for the teaching of Periodontology and Implantology at the University of Milan.

Member of the Board of the European Association for Osseointegration (EAO) from 1998 to 2005, President of the EAO from 2001 to 2003 and Immediate Past-President for the years 2004–2005.

EAO Council Member 2005–2011.

Founder of the Italian Academy of Osseointegration.

Active Member and Vice-President of the Italian Society of Periodontology and Implantology (SidP) for the years 2003–2005. President 2023–2024 of the Italian Academy of Osseointegration (IAO).

Referee of the International Journal of Periodontics and Restorative Dentistry.

He has published numerous articles in scientific journals and is an international speaker on the subject of periodontology, osseointegration and bone regeneration.

CURRICULA

FABIANA ALLEVI • Degree in Medicine in 2010, specialized in Maxillofacial Surgery in 2016 at the University of Milan. In 2019, she obtained her PhD in Odontostomatological Sciences at the same university. Currently works as a Medical Consultant at the UO of Maxillofacial Surgery of ASST Santi Paolo e Carlo, Presidio San Paolo (Milan).

MARIO BERETTA • Degree in Dentistry and Dental Prosthetics from the University of Milan in 1999. Specialist in Oral Surgery. PhD in Implantology and Innovative Techniques in Implant Prosthetics at the University of Milan. Adjunct Professor at the School of Specialization in Oral Surgery at the University of Milan. Awarded national qualification as second-rank professor in 2017. Tutor at the Reference Centre for the treatment of edentulous and severe maxillary atrophies (Dir. Prof. C Maiorana). UOC Maxillofacial Surgery and Odontostomatology (Dir. Prof. AB Gianni) (IRCCS Fondazione Policlinico-Cà Granda Milano). His activity is mainly focused on Implantology, Advanced Osseointegration and Periodontology. He is author of numerous publications in national and international journals and speaker at national and international congresses.

FABIO BERNARDELLO • Graduated in Medicine and Surgery in 1990. Specialized in Pathological Anatomy and Histology in 1994 at the University of Verona. He was Adjunct Professor for the teaching of Oral Surgery (University "Alma Mater Studiorum" of Bologna and University "Aldo Moro" of Bari). Active member and currently advisor in the IPA (International Piezoelectric Surgery Academy) Board of Directors. He is author or co-author of about 30 scientific articles published in international peer-reviewed journals.

FEDERICO BIGLIOLI • Degree in Medicine and Surgery in 1992 from the University of Milan and subsequent specialization in Maxillofacial Surgery and Microsurgery at the same University. After directing the UO of Maxillofacial Surgery at the IRCCS Galeazzi in Milan (2010-2012), he assumed the role he still holds today as Director of the UO of Maxillofacial Surgery at the ASST Santi Paolo e Carlo. Currently Full Professor and Director of the School of Specialization in Maxillofacial Surgery at the University of Milan. Author of 119 scientific publications, most of them in international journals, and co-author of 11 scientific textbooks, 8 of which are in English. He deals with the various fields into which maxillofacial surgery is divided, concentrating his interest on surgery of facial and trigeminal nerve lesions, reconstructive head-neck microsurgery, and oncological surgery of the oral cavity and face.

PAOLO BOZZOLI • Degree in Dentistry and Dental Prosthetics from the University of Milan in 1991. Active SICOI member. Has obtained: Certificate in Mobile prosthetics and diagnosis and therapy of myoarthropathies (Prof. Palla University of Zurich), Diploma in Clinical Periodontology (Prof. J. Linde University of Gothenburg), Certificate in Oral Rehabilitation by means of implants (Prof. J. Linde J. Wennstrom, University of Gothenburg), Certificate in Fixed and removable prosthodontics (Prof. N.P. Lang University of Berne), Certificate in Evidence-Based-Treatment Planning (Prof. J. Linde Prof. T. Berglund), Attending member of the Continuous Education in Dentistry of the Ariminum Research & Dental Education Center with title of Senator. Master in Implantology and Oral Rehabilitation at New York University. Speaker at theoretical-practical courses in basic and advanced implantology. Speaker at national congresses. Lecturer at the Post-Graduate Course in Implantology at the University of Modena and Bologna. Lecturer at the 2nd Level Master in Digital Dentistry at the University of Insubria. Scientific Tutor at the Zimmer Institute in Winterthur Zurich. Author and co-author of scientific articles in international journals. Particularly involved in research on regenerative techniques of sinus lift and immediate loading. Trainer ZFX digital project (optical impression and digital workflow).

ELENA CANCIANI • Graduated cum laude in Medical and Pharmaceutical Biotechnology with a focus on tissue engineering and PhD in Morphological Sciences at the University of Milan (Unimi). A Research Fellow at the Department of Biomedical, Surgical and Dental Sciences (DISBIOC) of Unimi, she works at the Thin Sections Laboratory (Dir. Prof. Dellavia), conducting research in the following areas: microscopic anatomy, cell and organotypic cultures, molecular and histological analyses of dental and periodontal tissues under physiological conditions or in the presence of biomaterials and metal prostheses. He participates in research and development projects on new biomaterials, scaffolds and medical devices for the regeneration and treatment of periodontal tissues and bone tissue in particular, collaborating with numerous national and international

CURRICULA

companies and laboratories in the dental and orthopedic field. He has attended numerous national and international research institutes to learn and deepen his knowledge of the new technologies available for the preparation, observation and interpretation of the morphological data obtained. He has also participated in projects that have received funding from the Novara and Milan Community Foundations and non-profit associations. The results of his research activities have been presented at both national and international congresses and are documented by publications indexed on PubMed.

CHRISTER DAHLIN • Dr Christer Dahlin is a Professor in Guided Tissue Regeneration and Oral Surgery at Department of Biomaterials, University of Gothenburg, Sweden. He is the President-elect and a member of the Executive board for the Osteology Foundation. Professor Dahlin is considered one of the pioneers in the development of the concept of Guided Bone Regeneration. He has over thirty years of experience in this field as well as on implant treatment and related research. He has published numerous articles and textbooks on these topics. He frequently travels worldwide to lecture, conduct research and teach on this and other implant-related topics.

DANIELE DE SANTIS • Degree in Medicine and Surgery in 1989 from the University of Verona. Specialized in Odontostomatology and Maxillofacial Surgery at the University of Verona, he graduated in Dental Implantology at the Université Sophia Antipolis of Nice in France. Since 1992 to date Medical Director at the Dental and Maxillofacial Surgery Clinic (Dir. Prof. Pier Francesco Nocini) of the Verona Hospital. Member and scientific director from 2001 to date of the "University Master's Degree Programme in Oral Surgery and Implantology" at Verona University. Visiting Professor at the Christian Albrechts Universität in Kiel (Germany) in 2003 and, since 2006, Associate Professor in Dental Materials Science at the Degree Course in Dentistry and Dental Prosthetics at the same university. Instructor in a training program in oral surgery on anatomical preparation at the Medizinische Universität Graz (Austria). Court-appointed technical consultant for the Court of Verona. In 2018 he obtained the qualification as Full Professor (ASN announcement D.D. 1532/2016 concurrency sector 06/F1). Referee since 2021 of the Doctorate in Odontostomatological Sciences. He has published in the most important international journals. Author of the books: 'Treatise on pre-prosthetic surgery' (Martina, 2005) and 'New perspectives in implant prosthetics and regenerative surgery' (Quintessenza Edizioni 2010). He is co-author of 'Materials and technologies in dentistry' (Ariesdue ed., 2011). Member of the editorial board of: International Journal of Implant Dentistry, Journal of Indian Society of Periodontology and Quintessence International.

CLAUDIA DELLAVIA • Degree with honors in Dentistry and Dental Prosthetics (OPD) and PhD in Morphological Sciences at the University of Milan (Unimi). Full Professor of Human Anatomy at the Department of Biomedical, Surgical and Dental Sciences (DISBIOC) of Unimi with teaching tenure for numerous three-year degree courses at the Faculty of Medicine and Surgery, for the single-cycle degree courses in OPD and in Medicine and Surgery (international course), and for the Schools of Specialization in Dental Surgery, Pediatric Dentistry and Orthodontics. Member of the board of teachers for the PhD in Clinical Research at Unimi. She carries out her research activities in the field of macroscopic, microscopic and functional anatomy of the stomatognathic apparatus in the Thin Sections Laboratory she directs at DISBIOC. Author of more than 90 publications in international journals indexed in PubMed. She has scientific collaborations with national and international teaching and research centres belonging to Italian, European, US and African universities.

LUCA FERRANTINO • Degree in Dentistry from the University of Milan in 2009. Scholarship SIdP (Italian Society of Periodontology and Implantology) for the two-year period 2010-2011. Master in Oral Sciences at the University Complutense of Madrid in Spain in 2012, and in 2015 PhD in Bone Regeneration at the University of Milan and at the University Complutense of Madrid. Tutor in Periodontology at the University Dental Clinic of Milan 2012-2018. Since 2013 he has been following Prof. Massimo Simion in his teaching and research activities, and with him he has published several articles on implantology and regenerative techniques of hard and soft oral tissues. Since 2019, he is Adjunct Professor for the Degree Course in Dentistry at the University of Milan.

CURRICULA

FILIPPO FONTANA • FILIPPO FONTANA - Degree in Dentistry and Dental Prosthetics with 110/110 cum laude from the University of Milan. Specialized in Dental Surgery at the same university with 70/70 cum laude. In 2017 he obtained the national scientific qualification (ASN) as Associate Professor. Adjunct Professor at the School of Specialization in Oral Surgery at the University of Milan 2012-2016. Active member of the Italian Academy of Osseointegration (IAO). From 2000 to 2017 he was attending physician at the Implantology Department of the Cà Granda Foundation Dental Clinic (Dir. Prof. C Maiorana). Winner of a scholarship for further training abroad in 2002, he spent the period September 2002 – June 2003 at the Department of Oral Medicine, Infection and Immunity - Harvard School of Dental Medicine - Boston (Dir. JP Fiorellini) as a clinical assistant and researcher. He was a clinical and research consultant at the Institute for Dental Research and Education (IDRE) (Dir. Prof. M Simion). Author of national and international publications, he focuses his clinical activity on periodontology and implantology with a special emphasis on bone regeneration.

ELEONORA IDOTTA • Degree in Dentistry and Dental Prosthetics with 110 cum laude in 2006, from the University of Messina. Intern at the Department of Odontostomatology, AOU G. Martino, Messina. From 2007 to 2010 she carried out clinical-assistance activities at the UOS of Odontostomatology of the G. Martino Hospital of Messina with particular reference to: Odontostomatological Surgery, diagnosis and treatment of Osteonecrosis of the Maxillary Biphosphonates. From 2007 to 2010 tutor of the 2nd level Master's Degree Course in Oral Implantology and Prosthetic Rehabilitation. University of Messina. In 2010 he obtained his PhD in Implant-Prosthetic Technologies in Dentistry at the University of Messina. In 2012, she completed her specialization in Dental Surgery at the University of Milan. Author of scientific papers and a textbook on peri-implantitis with Prof. M. Simion.

MICHELE MAGLIONE • MICHELE MAGLIONE - Graduated with honors in Medicine and Surgery from the University of Milan in 1992, and specialized with honors in Odontostomatology in 1995 at the same University. Author of publications in national and international journals on the subject of implants, and speaker at national and international congresses and courses. Active member of the Italian Academy of Prosthetic Dentistry (AIOP), and member of the European Academy of Osseointegration (EAO). He practices as a dentist in Canegrate (MI) and in Milan in collaboration with Prof. Massimo Simion.

CARLO MAIORANA • Full Professor of Oral Diseases. Professor of Oral Surgery, University of Milan. Director of the School of Specialization in Oral Surgery, University of Milan. Medical Director UOC Maxillofacial Surgery and Odontostomatology Policlinic Foundation, Director of the Implantology Centre for edentulism and maxillary atrophies, IRCCS Policlinic Cà Granda Foundation. Author of 160 publications in international journals. Reviewer and Board Member of numerous international journals.

MARCO MORRA • Degree in Chemistry from the Faculty of Science at the University of Turin in 1986. His professional activity has always focused on the study, characterization and modification of the surface properties of materials. After gaining experience in research centers on the surface properties of materials used in optical communications and of polymeric and biomedical materials Co-founder of Nobil Bio Ricerche in 1994, where he delved into the surface treatment and analysis of titanium implant screws. Co-author of a couple of texts, more than one hundred publications and about twenty patents on different aspects of material surfaces, with an H-index of 48.

MYRON NEVINS • DDS, is the Editor of the International Journal of Periodontics & Restorative Dentistry and Associate Clinical Professor of Periodontology at the Harvard School of Dental Medicine. Dr. Nevins is the former Director and President of the Academy of Osseointegration Foundation Board. Dr. Nevins is a past President of the American Academy of Periodontology and a former Director and Chairman of the American Board of Periodontology where his contributions have been recognized with the Gold Medal and the Master Clinician Awards. He was previously Director at the Institute for Advanced Studies, and a former director at the American Academy of Periodontology Foundation. He is an adjunct Professor at the University of Michigan School of Dentistry. Dr. Nevins was also a Diplomate for the American Board of Periodontology. He maintains a private practice limited to Periodontics and Implantology in Swampscott, Massachusetts. Dr. Nevins is the founder and President of Perio Imp Research, Inc.

CURRICULA

PIER FRANCESCO NOCINI • Degree in Medicine and Surgery, University of Pavia in 1981. Postgraduate diploma in Odontostomatology, University of Verona in 1985. Postgraduate diploma in Maxillofacial Surgery, University of Verona in 1990. Magnificent Rector of the University of Verona, Director of the UOC Maxillofacial Surgery and Dentistry and Member of the Interdepartmental Management Board and of the AOUI Verona Health Council. 2012–2013: Member of the Commission for National Scientific Qualification of the Ministry of Education, University and Research for Competition Sector 06/E3 Neurosurgery and Maxillofacial Surgery. 2017–2019: Past-President of the Italian Society of Maxillofacial Surgery. From 2020 to present President of the College of Maxillofacial Surgery Lecturers. Has participated as a speaker at countless national and international scientific congresses. He is the author of 250 articles. He has also edited numerous books, treatise chapters and monographs with international circulation. In the course of his decades-long university career, he has held numerous teaching posts in the degree courses in Medicine and Surgery, Dentistry and Dental Prosthetics, the degree course in Dental Hygiene, specialization schools and university masters courses. He has also been invited as Visiting Professor in Degree Courses, Masters and training seminars at prestigious national and international universities.

RICCARDO NOCINI • Degree in Medicine and Surgery with 110 cum laude from the University of Verona in 2017. From 1 November 2019 to date Doctor in specialist training, School of Specialization in Otolaryngology, Azienda Ospedaliera Universitaria Integrata (AOUI) of Verona. Fields of interest: oncologic head and neck surgery, endoscopic nasal surgery, endoscopic ear surgery, reconstructive microsurgery, laser surgery of the larynx, facial plastic surgery and aesthetic medicine.Participation in the scientific organization of the courses of Anatomic Dissection of the Head and Neck (Cadaver Lab) with U.O.C of Otolaryngology (AOUI Verona). He has published in the most important international journals. Member of the editorial board of national and international scientific journals, such as: Frontiers in Dental Medicine, Journal of Applied Sciences (MDPI), Journal of Clinical Medicine (MDPI) and Section Editor for the journal Annals of Maxillofacial Surgery. Active Member Italian Society of Microsurgery (SIM).

STEFANO PIERONI • Degree in Dentistry and Specialization in Oral Surgery Odontostomatology at the University of Milan. 2010-2017 frequenter of the implantology department at the Dental Clinic in via della Commenda at the University of Milan. Since 2013 he has been working with Prof. Massimo Simion as second operator during the Advanced Surgery and Immediate Loading courses, as well as during the annual course. Author of publications in national and international journals, he focuses his clinical and research activity on topics such as bone regeneration, peri-implant soft tissue and aesthetic prosthetics. Since 2014, he has been a speaker at national and international congresses and courses. He practices his profession in Milan, devoting himself mainly to prosthetic rehabilitations on natural teeth and implants. Ordinary member of the Italian Academy of Osseointegration and the Italian Academy of Esthetic Dentistry.

ALBERTO PISPERO • Degree in Dentistry and Dental Prosthetics in 2006. Specialization in Odontostomatological Surgery in 2009 with full marks and honors at the University of Milan. Since 2019 he has been a PhD in Odontostomatological Sciences. Since 2010 medical consultant and tutor for students of the degree course in Dentistry at the Dental Clinic of ASST Santi Paolo e Carlo in Milan. Since 2017 winner of a teaching contract at the University of Milan for the Degree Course in Dentistry and Dental Prosthetics and the specialization course in Odontostomatological Surgery. Lecturer at specialization courses in Oral Pathology and Surgery. Founder with Prof. G Lodi and Dr R Franchini of the non-profit organization ACAPO (Association for the fight against cancer and oral pathologies). Active member of the International Academy of Osseointegration (IAO) and has been its secretary since 2019. Member of the International Team for Implantology (ITI), member of the Italian Society of Oral Pathology and Medicine (SIPMO), author of articles and posters presented in national and international congresses.

CURRICULA

ROBERTO PISTILLI • Degree in Medicine and Surgery from the University of Rome in 1983. Specialist in Maxillofacial Surgery since 1988; manager maxillofacial surgeon at the ACO S. Camillo in Rome from 1989 to 1994, at the ACO S. Giovanni Addolorata in Rome (1994-1999) and at the ACO S. Filippo Neri in Rome (1999-07/2015) with high professionalism (P1) and at the ACO S. Camillo in Rome from August 2015 to date with the same appointment. Director of the Annual Postgraduate Course in Oral Surgery and Implantology at ACO San Filippo Neri in Rome (2001-2015). Director of the Annual Course in Rational Dissection of the Anatomical Loggias of the Neck and Oral Cavity held at the University Miguel Hernandez de Helche, S. Juan de Alicante (Spain). Adjunct Professor at the Universities of Bologna and Genoa. Author of the book Anatomy and Surgery of the Oral Cavity (EditaliaMedica). Contributor to other textbooks in the field of Dentistry and Maxillofacial Surgery. Active Member I.A.O. Member of the Scientific Committee of the International Journal of Osseointegration. Member of the Editorial Board of the European Journal of Oral Implantology. Director CAO Rome from 01-01-2012 to 31-12-2017. Currently President of the Cultural Commission CAO Rome. President of SIRIO Roma ARCOI (Roman Academy of Oral Surgery and Implantology) from 2012 to date. President of the GISOS Society from 2019 to date. President of the Inthema Society from 2020 to present. Author of publications in national and international indexed scientific journals. Speaker at numerous courses and congresses in Italy and abroad.

ALESSANDRA SIRONI • Graduated with honors in Dental Hygiene from the University of Milan in 2011. She has attended numerous refresher courses in Italy and abroad, including: theoretical-practical course on osseointegration, regeneration and peri-implantitis held by Prof. M Simion, theoretical-practical course on the treatment of the periodontal patient, held by Dr. Ghezzi and Dr. Masiero. Theoretical module of Surgical Periodontology for Hygienists held by Prof. G Zucchelli. Clinical course in Periodontology at the University Clinics Folktandvarden in Gothenburg, Sweden, held by Prof. Berglund, Prof. Cassel and Dr. Koch. Theoretical-practical course in non-surgical periodontal therapy held by Prof. Graziani, Prof. Tomasi and Dr. Suvan. Specialization in the treatment of peri-implantitis and complex periodontal cases, has been working with Studio Simion since 2012.

GIOVANNI ZUCCHELLI • Degree in Dentistry and Dental Prosthetics with honors in 1998. PhD in “Medical Biotechnology”, specializing in “Biomedical Technologies” at the University of Bologna. Full Professor of Periodontology at the University of Bologna. Director of the Department of Periodontology and Dental Hygiene at the University of Bologna. Coordinator of the Degree Course in Dental Hygiene at the University of Bologna. Honorary Member of the American Academy of Periodontology (AAP). President of the Italian Academy of Osseointegration (IAO). Member of the Editorial Board of the International Journal of Periodontics and Restorative Dentistry, and member of the Editorial Board of the Journal of Periodontology and International Journal Oral Implanatology. Associate Editor of the International Journal of Esthetic Dentistry. Active member of EAED (European Academy of Esthetic Dentistry), SIdP (Italian Society of Periodontology and Implantology) and IAO (Italian Academy of Osseointegration); member of EFP (European Federation of Periodontology). Author of more than 130 publications impacted in the periodontal and implant field. Winner of awards for scientific research in periodontology in Italy, Europe and the United States. Speaker at the most important national and international congresses in the field of Periodontology and Implantology. Co-author of two textbooks on periodontal plastic surgery (Ed. Martina) and Co-author of the chapter “Mucogingival Therapy-Periodontal Plastic Surgery” in the book “Clinical Periodontology and Implant Dentistry” (Lindhe J, Lang NP, Karring T [eds], Wiley-Blackwell. Author of the books “Mucogingival Aesthetic Surgery” and “Mucogingival Aesthetic Surgery on Implants” published by Quintessence Publishing Italy.

HISTOLOGICAL FEATURES OF OSSEOUS TISSUE

CHAPTER 1

Edited by Claudia Dellavia and Elena Canciani

INTRODUCTION

The alveolar bone is a vital, mineralized connective tissue of ectomesenchymal origin. It consists of a network of protein fiber bundles arranged in layers and is permeated by dense mineral deposits. The protein fibers provide strength and flexibility to the bone tissue, while the mineral deposits impart hardness and rigidity. Bone cells adapt to mechanical stresses to maintain this tissue architecture. They also assist in maintaining homeostasis in plasma calcium levels via bone remodeling processes. Alveolar bone contains more extracellular matrix (ECM) than cellular matrix. The ECM consists of an organic component (about 33%) and an inorganic component (about 67%). The inorganic component is responsible for providing mechanical support and maintaining the calcium homeostasis of the body. The outer layer of bone is covered by the periosteum, which is divided into three layers: the germinative layer, the nutritious/sensory layer, and the fibrous layer. The inner aspect of bone is covered by the endosteum, a thin connective membrane comprising a single layer of cells with osteogenic potential.

The deepest layer of the periosteum is the germinative layer, which is rich in osteoblasts and their precursors.

The intermediate nutritious layer provides vascularization for 15% to 20% of the cortical bone and is characterized by blood vessels immersed in the matrix and mixed with fibroblasts, which maintain tissue texture.

Finally, the fibrous layer is formed by bundles of collagen fibers that keep it nonelastic and fixed to the bone surface.

> *Knowledge of histology is a basic requirement for performing high-level clinical work. It helps clinicians select the correct clinical protocols and surgical techniques. Thus, this chapter and chapter 3, on the biologic principles of osteointegration, are the most important in the entire book.*
>
> *Massimo Simion*

The main functions of the periosteum are:

- to resist tractional and torsional forces applied to bone (mechanical function);
- to supply required nutrients for cell turnover of bone tissue, which is extremely vital (trophic function);
- to provide proprioceptive and nociceptive information and activate repair, modeling, and remodeling processes with its reservoir of osteoprogenitor cells (sensory function).

TYPES OF BONE TISSUE

There are two types of alveolar bone, nonlamellar bone and lamellar bone. Nonlamellar bone consists of a network of collagen fibers delimiting large cavities filled with vessels. The collagen fibers first form an osteoid matrix, which then organizes to form a woven fiber structure (woven bone). This type of immature bone is present in all cases of neodeposition.

The formative process of nonlamellar woven bone involves several steps. First, osteoblasts must process osteoid, which is a nonmineralized substance made of collagen fibers and a matrix containing glycoproteins and proteoglycans. Continuous apposition of new osteoid matrix causes the previously deposited matrix to mineralize, advancing the mineralization front. Osteoid undergoes calcification via the deposition of minerals such as calcium and phosphates, which are then transformed into hydroxyapatite.

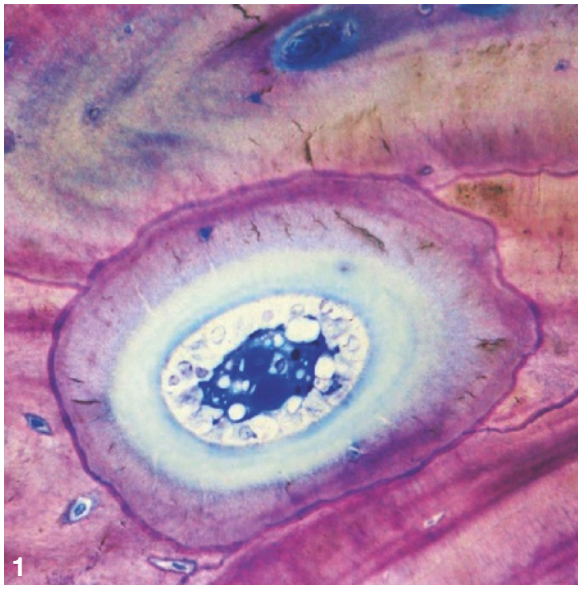
The maturation process, understood as organization and mineralization, leads osteoid to arrange itself into a weave of fibers that evolve into woven bone. At this stage, some osteoblasts become trapped in the matrix of calcified bone tissue in the form of osteocytes. These trapped osteocytes are lodged in irregularly dispersed lacunae in the calcified matrix and remain connected to one other though bone canaliculi that contain their cytoplasmic extensions.

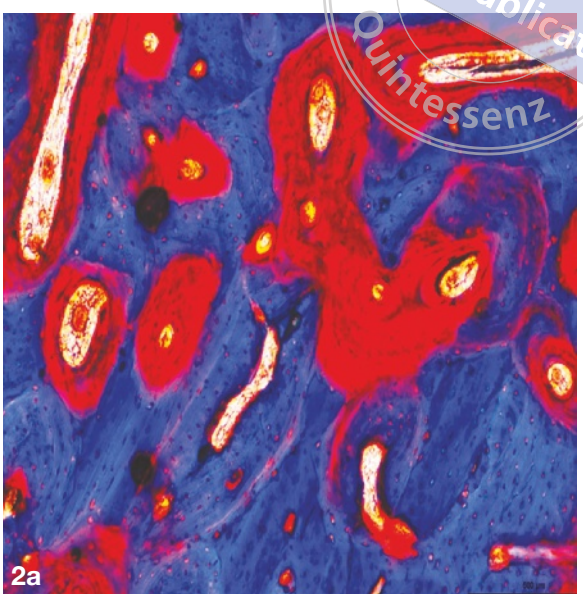
Numerous anastomoses in the canalicular system ensure a network for signaling.[1]

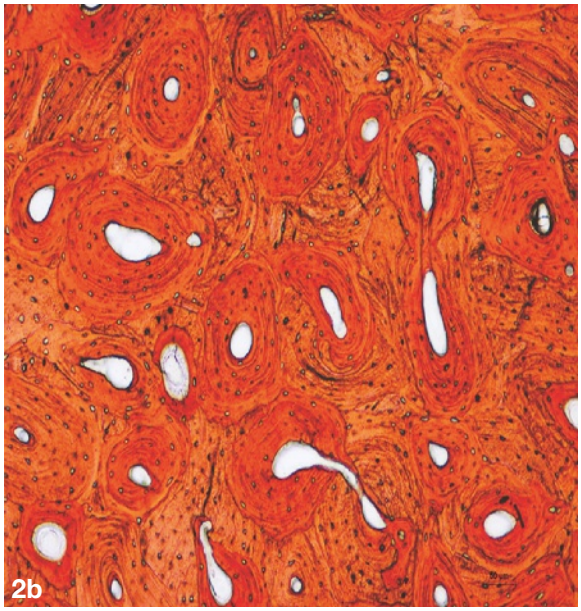
Lamellar bone represents the mature form of human bone tissue. It is organized in layers of lamellae, among which the lacunae containing the osteocytes are found. The lamellae are connected by canaliculi that pass through their entire thicknesses. Lamellar bone is less cellular than nonlamellar bone and presents as either compact bone or spongy bone.

Compact lamellar bone is characterized by osteons (or Haversian systems). Osteons are functional units with a cylindrical shape made up of groups of between 1 and 20 concentric lamellae that leave a cavity (Haversian canal) in the center, through which blood/lymphatic vessels and myelinated nerve fibers run **(Fig 1)**. Volkmann's canals are smaller-caliber neurovascular canals that cross the bone transversely or obliquely to its major axis, connecting Haversian canals to each other and opening onto the periosteal and endosteal surfaces of the bone **(Fig 2)**.

In the osteon, the innermost lamellae are those that have been most recently deposited. The osteocytes are confined to the bone lacunae and are arranged in concentric rings within the lamellae. Numerous canaliculi radiate from the lacunae in all directions. The osteocytes communicate with each other via gap junctions between the cytoplasmic extensions. The osteocyte cell body and its extensions are separated from the walls of the lacunae and canaliculi by a thin layer of osteoid. Even in mature bone, the lacunae and canaliculi form a continuous system of cavities to allow metabolic and gaseous exchanges between the blood and osteocytes.[1] Bone undergoes continuous remodeling with the destruction of old osteons, whose remnants are left behind as irregular, interstitial lamellae systems. On the outermost aspect of the compact bone, osteons are delimited by a system of concentric lamellae of dense cortical bone called circumferential or external limiting lamellae.







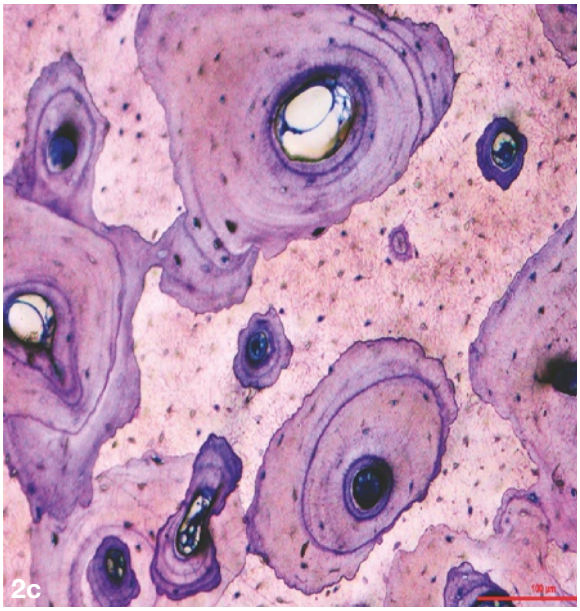


Fig 1 *The lamellae are not yet well delineated around the neurovascular canal of Havers, where osteoblastic cells, vessels, and nerve fibers can be seen. Toluidine blue and Pyronin yellow staining; ×400 magnification.* ***Fig 2*** *Cortical bone in three colors to highlight different aspects of mineralization (×200 magnification).* (a) *The bone stained with Goldner's trichrome modified for hard tissue shows less-mineralized and more recently deposited lamellae in red and bone with a high degree of mineralization in blue.* (b) *Red Alizarin staining makes it possible to observe the entire mineralized matrix without defining the levels of mineralization, which are evident with the Toluidine blue and Pyronine yellow staining.* (c) *Toluidine blue and Pyronine yellow staining highlights the shape of the newly formed osteons undergoing remodeling and immersed in a matrix with a high level of mineralization.*

Spongy lamellar bone is composed of thin laminae called trabeculae, which are formed by irregular bone lamellae that branch and anastomose into a 3D network containing bone marrow within its lattice (medullary cavities).

Osteocytes are contained in the lacunae, and their extensions are contained in the canaliculi that open into the medullary cavities[1] **(Fig 3)**.

THE COMPOSITION OF BONE TISSUE

Bone tissue is a connective tissue characterized by an abundant ECM that consists of fibers, an amorphous substance of glycoprotein origin, and an inorganic component in which numerous cells are dispersed.

ECM

The inorganic portion of the ECM is made up of mostly hydroxyapatite crystals, calcium carbonate, calcium fluoride, and magnesium phosphate. The hydroxyapatite crystals are deposited among the Type I collagen fibers of the organic portion, which are themselves immersed in an amorphous substance consisting mainly of noncollagenous proteins and adhesion proteins **(Fig 4)**. The noncollagenous proteins include phosphoproteins, osteocalcin, matrix Gla protein (MGP), lipids, lipoproteins, and alkaline phosphatase. The adhesion proteins include osteopontin, osteonectin, fibronectin, thrombospondin, and sialoproteins.[2]

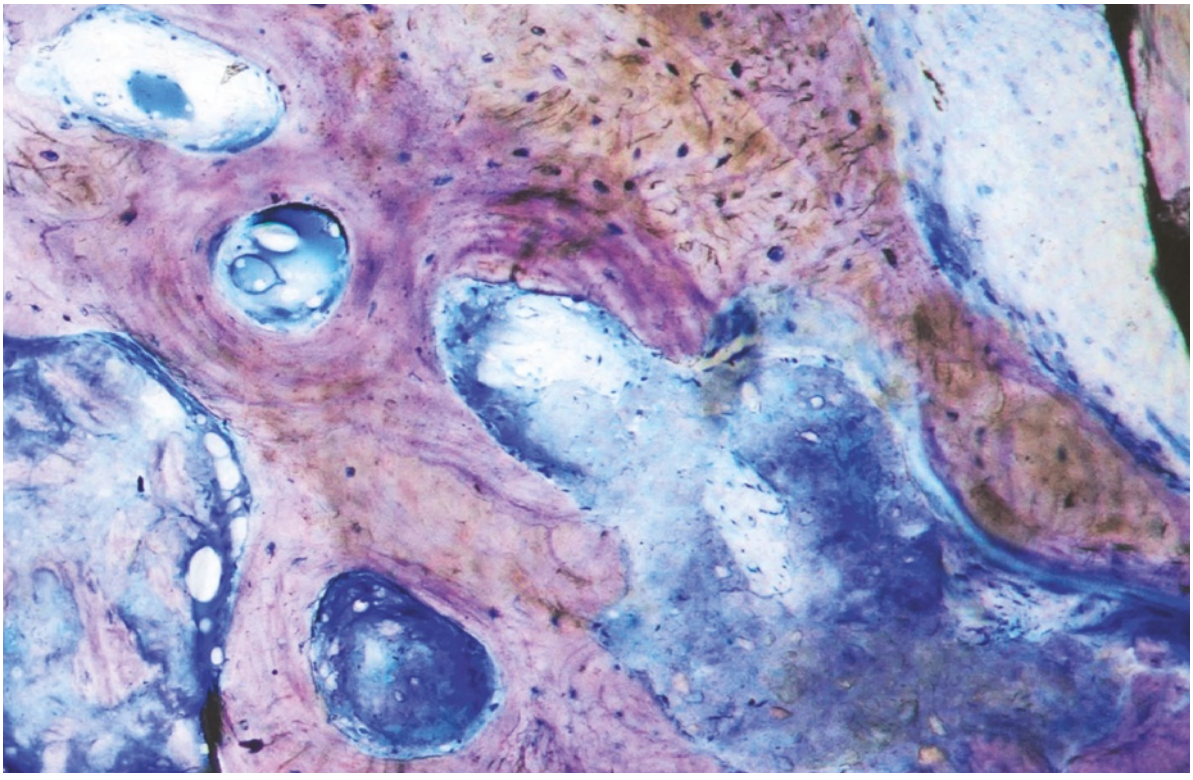


Fig 3 *Spongy alveolar bone. The thick bone trabeculae are surrounded by matrix of varying degrees of mineralization that is undergoing remodeling. Areas of newly deposited osteoid matrix are depicted in blue, areas of mineralizing tissue (woven bone) are seen in purple, and areas with a high degree of mineralization are shown in brown. Toluidine blue and Pyronin yellow staining; ×400 magnification.*

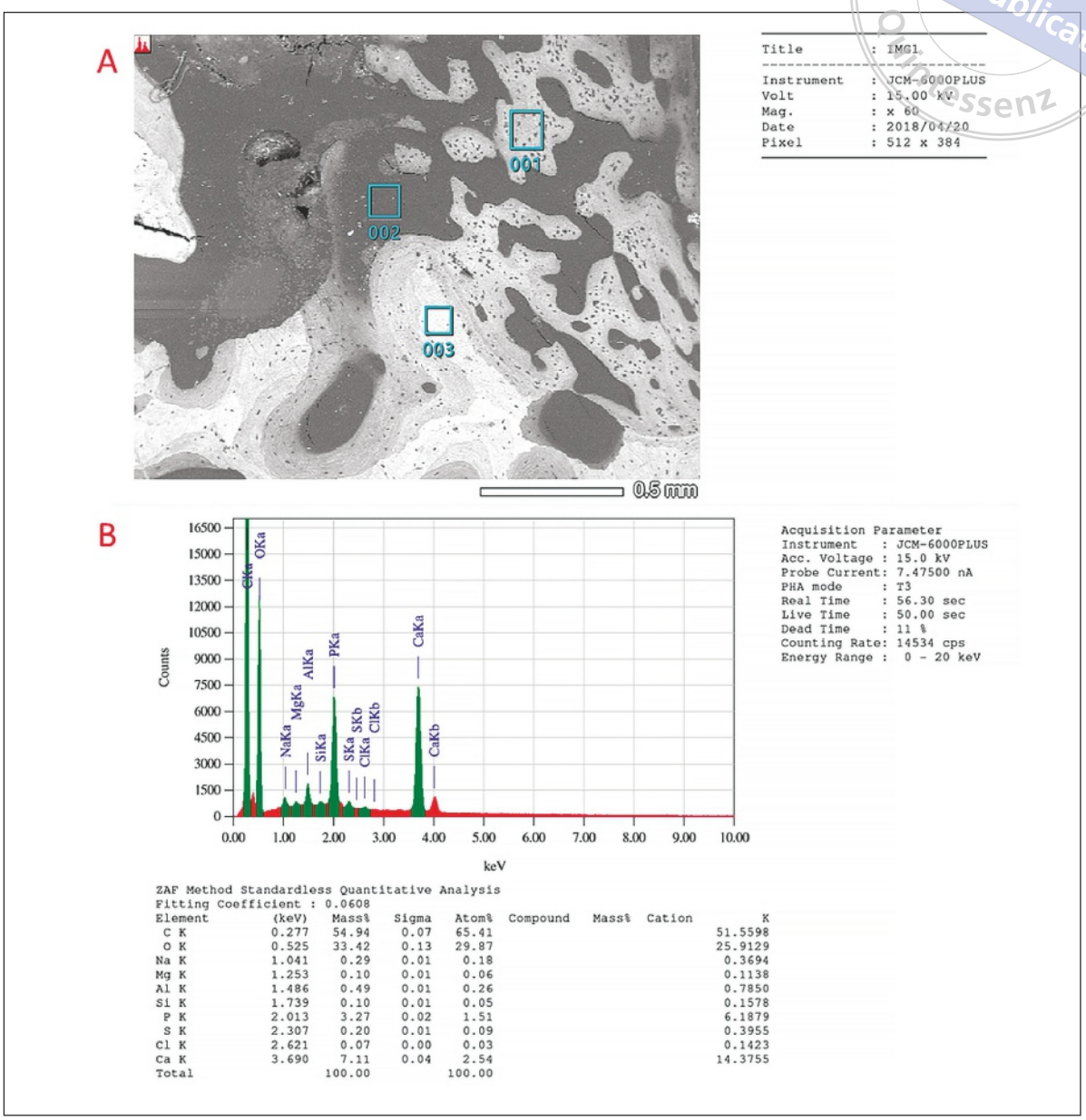


Fig 4 (A) *A scanning in a backscattered scan electron microscope imaging (BEI) can be used to observe the density of the matrix based on the atomic number of the elements it is composed of in different shades of gray. The lighter area is more mineralized than the darker one.* (B) *In relation to image a, the areas in the blue box can be further analyzed by defining the element analysis using a detector to identify the characteristic X-rays of each element present in the sample under analysis. This information is then processed by dedicated software that generates the spectrum and indicates the percentage mass of the most-represented chemical elements. Certain elements can then be analyzed to make comparisons between samples, for example, by calculating the ratio between the amount of calcium and phosphate contained in different samples to better understand the degree of mineralization of regenerated tissue.*

Noncollagenous proteins

The role of noncollagenous proteins is still being studied, but they seem to be involved in the mineralization processes of bone tissue.

Phosphoproteins are proteins conjugated to phosphoric acid residues.

They have the characteristic of binding calcium and thus act as mineral nucleators.[3] Proteoglycans, on the other hand, inhibit calcification by masking sites on collagen fiber, which reduces chemical interactions and limits the sequestration of calcium ions and calcium phosphate complexes.[4]

Osteocalcin is produced by osteoblasts and is the most abundant noncollagenous protein in bone, accounting for about 20% of the noncollagenous matrix proteins.[5,6] It contains three calcium-binding Gla (gamma-carboxyglutamic acid) residues and is dependent on vitamin K. Its physiologic role is to regulate mineralization by facilitating the formation of mineralized nodules.[7] Like alkaline phosphatase, osteocalcin is used clinically as a marker of osteoblastic activity, and serum osteocalcin is used as a marker of bone turnover.[8]

Adhesion proteins

Adhesion proteins are located on the cell surface and have the function of creating bonds with other cells and/or the ECM.

Osteopontin is a relatively abundant noncollagenous sialoprotein produced by osteoblasts. According to the literature, it functions as a chemoattractant for osteoclasts.

It binds with these osteoclasts thanks to the RGD (arginylglycylaspartic acid) sequence that increases intracellular calcium by activating the phospholipase C pathway in the osteoclast. Osteopontin also has binding sites for hydroxyapatite crystals. It is regulated by vitamin D, which promotes its secretion and therefore the formation of inorganic bone matrix.[2,9]

Osteonectin is an acidic glycoprotein that supports bone remodeling and maintains bone mass in vertebrates. It is synthesized by osteoclasts, as well as by fibroblasts, tendon cells, and odontoblasts. Osteonectin binds to collagen and hydroxyapatite and assists in the subsequent formation of the ECM and the promotion of the nucleation of mineral groups.[2,5]

Fibronectin is a ubiquitous cell adhesion protein produced by cells in the bone both locally and nonlocally. Nonlocal fibronectin is transported via the circulatory stream. Although not fully understood, fibronectin's function in bone seems to be coordinating the migration, interactions, and differentiation of osteoblast precursors.[10]

The role of thrombospondin in bone tissue is still unknown. This protein is known to have calcium-binding sites and the RGD sequence.[4]

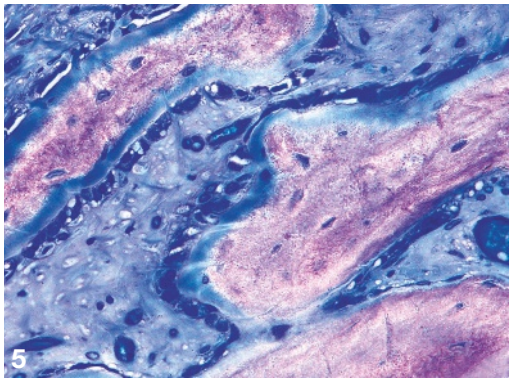
Bone sialoprotein (BSP), like osteopontin, is a sialoprotein found exclusively in the skeleton. In addition to promoting cell adhesion by activating osteoclasts, it is thought to be a hydroxyapatite nucleator, although its sensitivity to vitamin D is still unknown.[6]

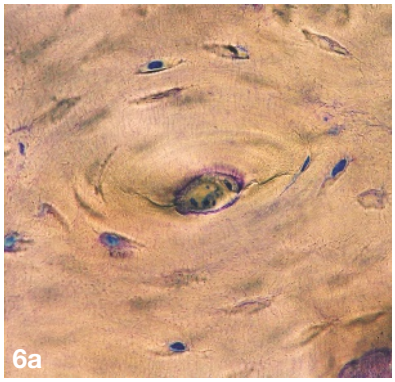
The cellular elements of bone

The cells that make up bone are osteoblasts, osteocytes, and osteoclasts.

Osteoblasts are the main cells responsible for bone formation. They synthesize the components of the extracellular organic matrix (especially collagen Type I, proteoglycans, and glycoproteins) and control matrix mineralization. Osteoblasts are round, mononucleated cells derived from osteoprogenitor cells residing in the periosteum and endosteum. They are located on bone surfaces that show active matrix deposition.

Osteoblasts can eventually differentiate into either lining cells or osteocytes. Osteoblast morphology varies in the active deposition phase, during which they present as either large cubic or tapered cells. In the inactive phase, they appear as thinned fusiform cells[1] **(Fig 5)**.





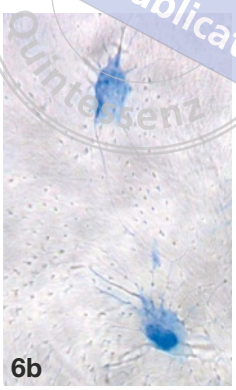


Fig 5 *Osteoblasts cords in the vicinity of newly formed bone trabeculae in an active deposition phase. Toluidine blue and Pyronin yellow staining; ×400 magnification.* ***Fig 6*** *Osteocytes lodged in osteocyte lacunae.* (a) *Several osteocytes can be seen residing in compact bone tissue with a high degree of mineralization. Toluidine blue and Pyronin yellow staining; ×600 magnification.* (b) *Two osteocytes characterized by cytoplasmic processes that reside in the osteocyte canaliculi. Toluidine blue and Pyronin yellow staining; ×1,000 magnification.*

Osteoblasts produce an enzyme called alkaline phosphatase, which is very important in the early stages of mineralization. Alkaline phosphatase is considered an early marker of osteogenesis because it is produced by differentiated osteoblasts in the first days of the bone matrix deposition process. In in vitro studies, the activity of this important marker is usually studied 7 and 14 days after the seeding of cells ready to deposit new mineralized matrix.[11]

Osteocytes are star-shaped cells that remain in the mineralized bone matrix during the matrix deposition phase, residing in the osteocyte lacunae. They are osteoblasts that have entered a state of quiescence, and once trapped in the already formed bone, they can participate in bone remodeling **(Fig 6a)**. They are unable to carry out mitotic division and remain inside the lacunae, where they renew and maintain the ECM by communicating with each other through cytoplasmic extensions that run through channels between the lacunae (osteocytic canaliculi)[1] **(Fig 6b)**.

Osteoclasts are very large, phagocytic, multinucleated cells of monocyte-macrophage lineage that can develop and adhere to the bone matrix and subsequently secrete lytic enzymes capable of degrading and breaking down the organic and mineral components of bone. The matrix degradation process results in the formation of specialized extracellular compartments known as Howship's lacunae **(Fig 7)**. One of the enzymes that osteoclasts produce is TRAP (tartrate-resistant acid phosphatase), which plays a very important role in many biologic processes, including collagen synthesis and degradation, macrophage recruitment, and the regulation of bone mineralization.

TRAP has been observed in the ECM at remodeling fronts, and in fact, it is customary to use anti-TRAP stain to identify osteoclasts in areas of bone undergoing resorption.[13]

The correct balance of osteoblastic deposition activity and osteoclastic resorption activity regulates bone remodeling by maintaining calcium-phosphate homeostasis and tissue morphology.[1,11] Osteocytes are mechanosensors that are capable of perceiving loads applied to the bone and subsequently coordinating bone remodeling by activating or inhibiting the activity of effector cells.[14] Thus, mechanical forces can induce changes in bone microarchitecture and density, which is of clinical relevance for the design of implant-supported restorations.[15,16]

Another characteristic of osteocytes is that they modify their biologic and biochemical activities according to homeostatic principles, secreting cytokines and growth factors that regulate tissue repair during fracture healing processes.[17] Osteocytes activate bone modeling and remodeling based on the need to adapt to tension and/or damage.[17]

Activation seems to depend on signaling resulting from forces imparted on the bone that radiate into the lacuna-canalicular system containing interstitial fluid.[17,18]

The alveolar bone has a faster remodeling rate at both cortical (minor changes) and trabecular (major changes) levels compared to other bones because it is subjected to continuous stimulation from various directions. The mandible typically exhibits continuous and rapid changes in the orientation, thickness, connectivity, and spacing of its trabeculae.[17]

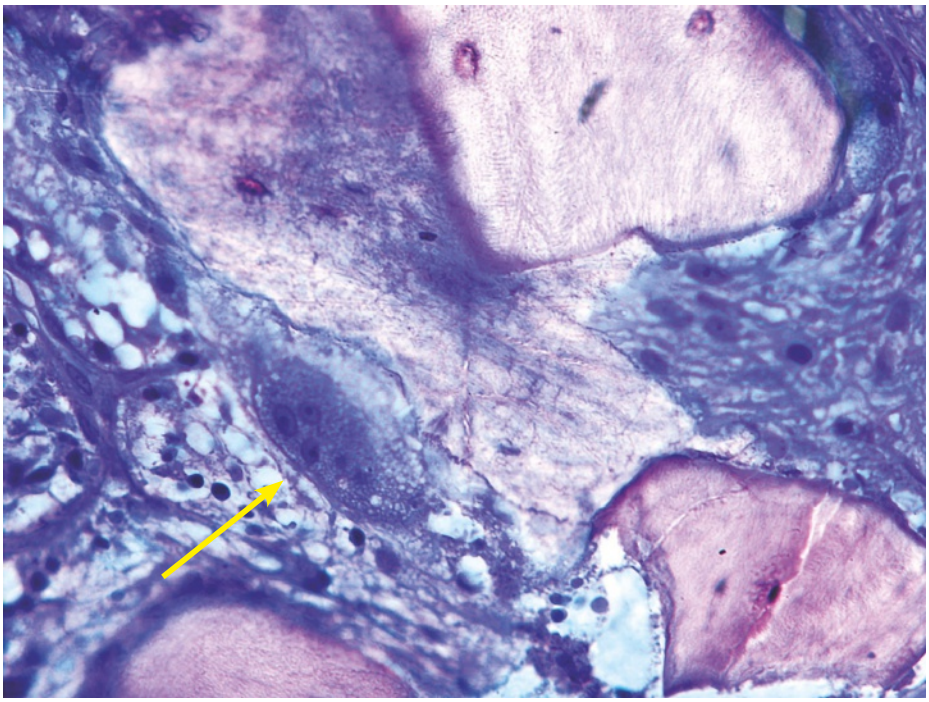


Fig 7 *An osteoclast in an active resorption process. The cell is lodged on the edge of a bony trabecula in a Howship's lacuna that has just been formed by lytic enzymes released to degrade the bone matrix. Toluidine blue and Pyronin yellow staining; ×600 magnification.*

MICROSCOPIC STRUCTURE OF THE ALVEOLAR BONE

The alveolar bone is the part of the alveolar process that rests on top of the basal component of the mandible and maxilla. It shares a close relationship with the dentition, and its shape, position, and volume change in response to the functional life of the teeth. In both the maxilla and mandible, the diploe structure involves two compact bone plates that enclose a spongy or trabecular part **(Fig 8)**. Two parts of the alveolar bone can be distinguished: the alveolar bone "proper," which comprises the wall of the alveolus where the teeth roots are housed, and the "supporting" alveolar bone, which surrounds the proper alveolar bone. The inner surface of the alveolus consists of a layer of specialized compact bone tissue called bundle bone. Bundle bone is characterized by the insertion of periodontal ligament (PDL) collagen fibers, known as Sharpey's fibers. Radiographically, this layer presents as a radiopaque band about 1.2 mm thick, and it is clinically referred to as lamina dura. At the crestal level, the lamina dura continues with the compact lingual bone plate, whereas on the vestibular side, the bundle bone forms the bone crest in continuity with the vestibular compact bone plate more apically.

The trabecular pattern of the alveolar bone varies according to location within the arches. In portions of the arches that undergo less masticatory load, the trabecular composition is thin and dense, whereas in areas subjected to more stress, trabeculae are thick and more widely spaced, arranged according to the direction of the forces.[19] The pattern of trabeculae in jaw bones differs from that

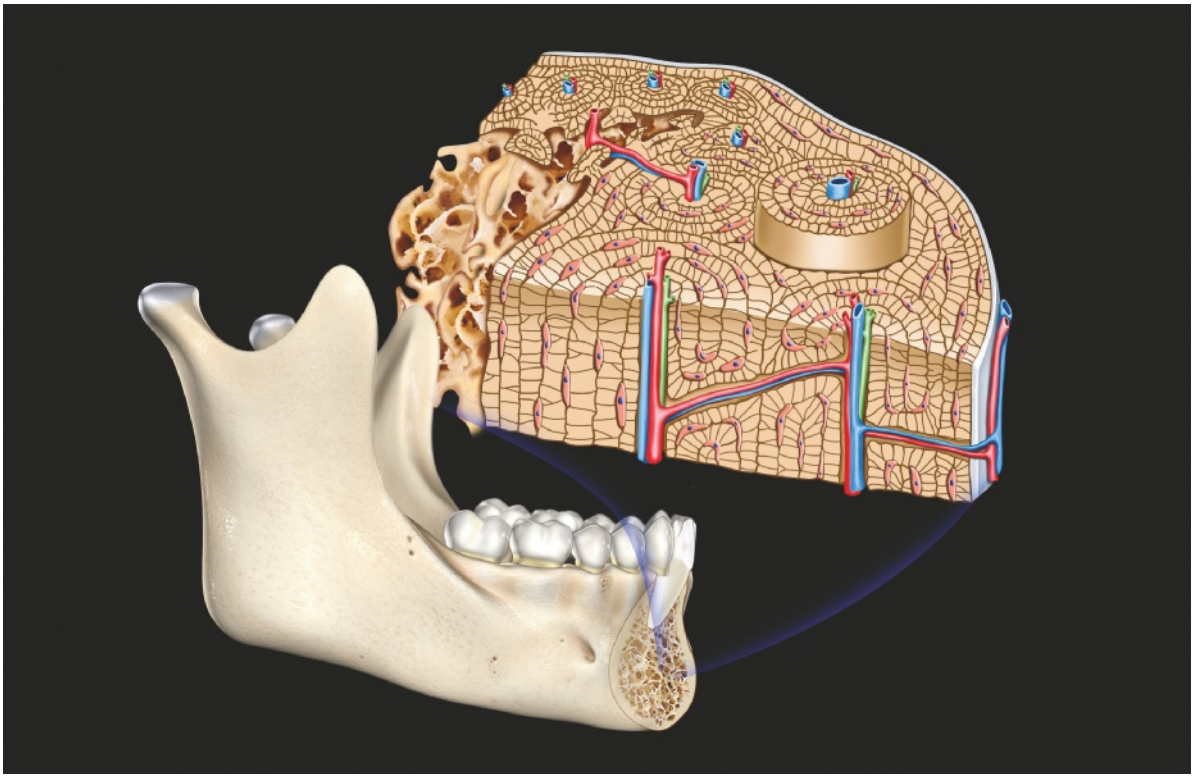


Fig 8 *Structure of the mandibular bone.*

in long bones, as found in micro CT studies.[20] In the femur, for example, the trabeculae of the bone resemble scattered rods in the medullary spaces, whereas in the alveolar bone, the trabeculae are characterized by a greater thickness and width, resulting in reduced medullary space. Histomorphometric analyses have also shown that mineral density, bone volume fractions, and trabecular thickness in the alveolar bone are also higher than in the femur.

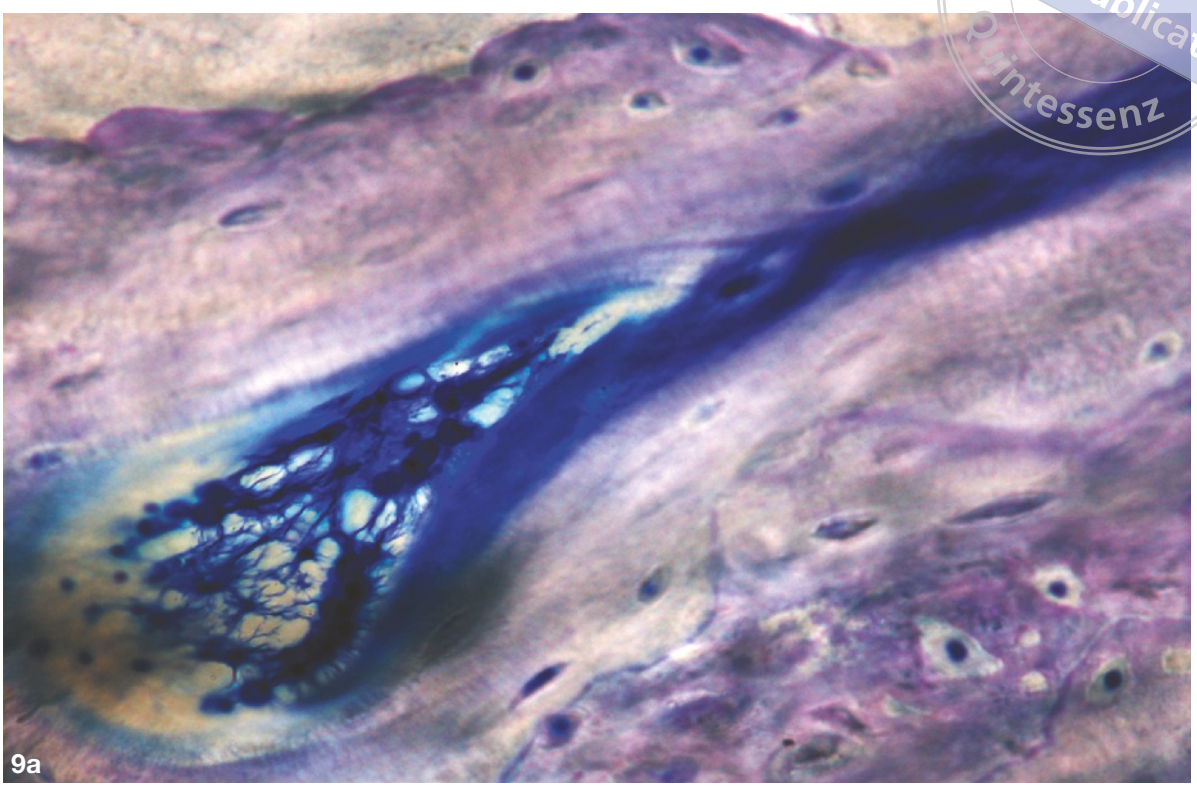
Compared to the maxilla, the mandible appears to undergo more pronounced bone remodeling and has higher mineral density and better bone quality, which is understood by Lekholm and Zarb to be represented by the ratio of cortical to trabecular bone tissue.[21,22] Microstructural changes in the jaw bones can be caused by edentulism, changes in masticatory loading, hormonal conditions, and age. It has been observed that the mandible and anterior maxilla show microstructural changes that are more pronounced in patients of advanced age, resulting in bone that is predominantly cortical in composition. Furthermore, von Wowern and Stoltze showed that mandibular cortical porosity increases with age without interfering with trabecular bone mass, which was not found to correlate with aging.[23]

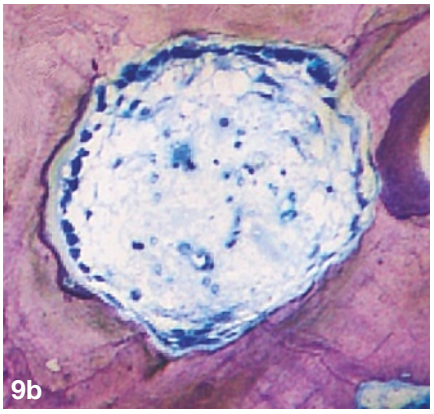
BONE SHAPING AND REMODELING

Once the bone is formed, the new mineralized tissue begins to be remodeled by resorption and deposition processes. These phenomena of resorption and tissue matrix deposition occur simultaneously and lead to the cyclic replacement and regeneration of the biologic components of the tissue, according to systemic and local functional needs (dental load and eruptive phenomena). The combination of these processes allows the bone to change morphologically in accordance with physical stimulations and to reach configurations that are appropriate for supporting the loads placed on it.[24] The metabolic mediation conducted by bone tissue (ie, functioning as a calcium and phosphate reserve) is controlled and connected to remodeling phenomena, as well as being driven by hormonal mechanisms.

Bone modeling is considered a distinct process from bone remodeling.[19] Bone modeling is the process of modifying/constituting the initial bone architecture. It has been suggested that external stimuli (ie, load variations) on the bone tissue trigger neoapposition phenomena and subsequent bone modeling in response to changes in the support required from the bone segment. Remodeling, on the other hand, refers to a change that occurs within the mineralized bone structure, without concomitant change in the architecture of the segment in which it occurs. Bone remodeling includes bone matrix renewal processes, changes to meet metabolic demands, and the replacement of immature primary bone with lamellar bone during bone formation. Bone remodeling is made possible by the constant presence of bone multicellular units (BMUs) in the tissue. A BMU is composed of an osteoclastic front on the bone surface undergoing resorption (ie, the resorption front), a compartment containing blood vessels and pericytes, and a layer of osteoblasts at the newly formed organic matrix (ie, the apposition front). Pericytes are undifferentiated mesenchymal cells that partially surround the endothelial cells of capillaries and venules. They can differentiate into osteoblasts given the appropriate stimulation by osteoinductive and/or osteopromoting growth factors.

Cutting cones are the main mechanism of bone modeling in the healing phase, particularly following fractures. They are characterized by an osteoclastic resorption front followed by a string of osteoblastic cells that lay down new bone matrix **(Fig 9a)**. The cutting cone passes through the fracture site, which is healed by the formation of numerous secondary osteons **(Figs 9b to 9d)**. This process is very slow and can take months or years.[25,26]





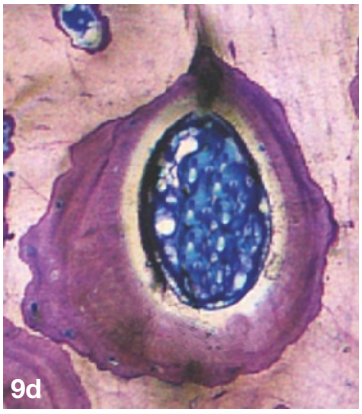


Fig 9 (a) *A cutting cone. The process of bone resorption sometimes takes place in channels dug longitudinally into the tissue, which results in the formation of cavities called cutting cones. Toluidine blue and Pyronin yellow staining; ×600 magnification.* (b to d) *Stages of new matrix deposition by the osteoblast chordae until the formation of a secondary osteon. Toluidine blue and Pyronin yellow staining; ×400 magnification.*

Recent studies[16] describe the viscoelastic properties of bone that vary as the tension to which the tissue is subjected changes. Following an increase in the load affecting it, Young's modulus of elasticity increases, expressing the propensity of a tissue to lengthen or shorten as a result of a loading force. To renew itself, repair itself, and adapt to continuous functional demands, stimuli, and loads, bone tissue undergoes constant remodeling, which enables it to preserve its strength or increase its rigidity.

Biochemical aspects

The remodeling process is cyclic and involves a complex series of phenomena that are finely regulated by systemic factors, such as hormones involved in both the regulation of skeletal growth and calcium metabolism (parathormone [PTH], growth hormone, leptin, and calcitonin) and local factors, such as cytokines, prostaglandins, and certain proteins, that are responsible for the interaction between osteoblasts and osteoclasts.[12,27,28] Communication between osteoblasts and osteoclasts can occur directly via cell-to-cell contacts through ligand-receptor interactions or indirectly via the secretion of soluble molecules such as cytokines, hormones, and growth factors.[27–29]

Biomechanical aspects

When microdamage accumulates in the interstitial bone tissue, microfractures present as discontinuities in the calcium-rich matrix, reflecting cracks and fractures in the mineral component that can evolve into a macrofracture due to continuous cyclic fatigue.[29] When microfractures occur at a slow rate, the bone has the opportunity to repair itself, a response that is referred to as targeted remodeling. Osteons can act as barriers to prevent the coalescence of microfractures and prevent breakage. In fact, dispersed microfractures have a higher energy absorption potential than coalescing ones. The mechanical behavior of compact bone is related to its hierarchical microstructure in terms of content, fiber direction, and the arrangement of osteons along the load axis. The more mineralized and less collagen-rich primary osteons develop more dispersed microfractures than secondary osteons derived from bone remodeling. Strength, energy-absorbing capacity, and modulus of elasticity appear to decrease with increasing percentage of the total bone surface occupied by osteons.[30]

There are many physiologic, pathologic, and pharmacologic systemic conditions that can affect cortical/trabecular thickness, mineralization, collagen content (bone tissue structure), and bone metabolism, thus modifying mechanical properties such as strength, rigidity, and fragility. With aging, bone trabeculae become thinner and are associated with significantly more highly mineralized trabeculae,[31] ultimately resulting in osteoporosis, which is characterized by reduced strength and increased bone fragility due to reduced osteoblastic activity, bone matrix formation, and mineralization.

FORMATION OF THE JAW BONES

Bone growth, function, and structure are biologically dependent on each other. The bone's growth program involves not only the bone tissue itself but also the soft tissues that cover it, such as muscles, the mucous membrane, and the vascular and nerve formations. The bones of the skull develop in relation to the central and peripheral nervous system. In fact, during development and growth, the dura mater regulates the sutures of the cranial vault and apparently also the synchondroses of the base, thanks to the continuous expression of regulatory genes.

When the encephalon expands, it compresses the dura mater, which comes into direct contact with the cranial bones, thus stimulating the pathway of signals for the development of the skull.

The bones of the splanchnocranium and some of the neurocranium originate from the embryonic connective tissue derived from the neural crests, while the remainder of the neurocranium originates from the mesodermal connective tissue of the somites in analogy to that of the long bones.

The embryonic derivation of the jaw bones is different than that of most bones of the skeleton, and the results are reflected in differing osteoclastogenic potentials, meaning the bones are affected differently by diseases of the skeletal system.[32] For example, the alteration of physiologic microarchitecture and systemic bone loss observed in osteoporotic experimental animals are site-specific, with the reduction in bone mineral density generally being lower in the cranial bones and jaw bones than in the long bones, lumbar vertebrae, and ileum.[33]

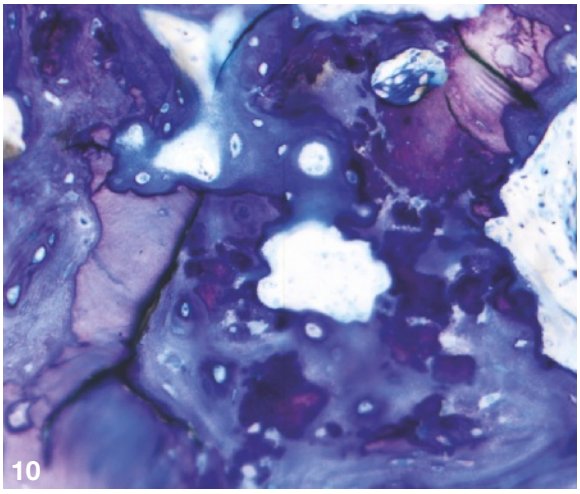
The mode of ossification of the different skeletal components of the skull also has some specificities. Bone formation in the skull occurs via two main mechanisms: intramembranous (direct) ossification and endochondral (indirect) ossification.

Intramembranous ossification leads to bone formation without intermediate processes. This is accomplished through deposition from primitive connective tissue mesenchymal cells with rich vascular support at the site of future bone deposition. After cellular differentiation, osteoblasts initiate the interstitial deposition of collagen-rich osteoid matrix, developing ossification centers characterized by small bundles of packed collagen fibers that gradually mineralize through the deposition of calcium salts in the intercellular spaces and adjacent osteoid **(Fig 10)**. All the flat bones that make up the vault of the skull and most of the maxillofacial complex are membranous bones, as are some parts of the bones that make up the base of the skull, for example, the squama and tympanic portion of the temporal bone. Intramembranous ossification genetically predetermines which bones will be subject to growth by traction during embryonic development.

A particular form of direct ossification is mantle ossification, typical of the mandible, which is characterized by the direct deposition of bone within the mesenchyme surrounding Meckel's cartilage (first pharyngeal arch) as a biomechanical scaffold. The body of the mandible and most of the mandibular ramus originate from this type of ossification. The remaining part of the ramus, including the condylar and coronoid processes, develop from secondary cartilage cores that undergo indirect ossification. At the end of ossification, Meckel's cartilage is completely resorbed, while the connective tissue that interposes at the median level between the two cartilages (right and left) differentiates into chin fibrocartilage (symphysis).[34,35]

Endochondral (indirect) ossification leads to the formation of bone from a primitive hyaline cartilage model. From the cartilaginous template, there is cell hypertrophy leading to calcification of the intracellular matrix, with subsequent degeneration of the chondrocytes. The cartilage matrix is then invaded by blood vessels and osteoblasts, reabsorbed, and replaced with osteoid **(Fig 11)**. This mechanism is responsible for the formation of all the long bones of the skeleton and can also be found in some bones of the skull base, such as the occipital bone (not intraparietal), temporal bone (petrous and mastoid portions, hearing ossicles, and styloid process), sphenoid (body, small wings, large wings), and ethmoid.

The endochondral ossification genetically predetermines bones that will be subject to high pressures during embryonic development.[36]



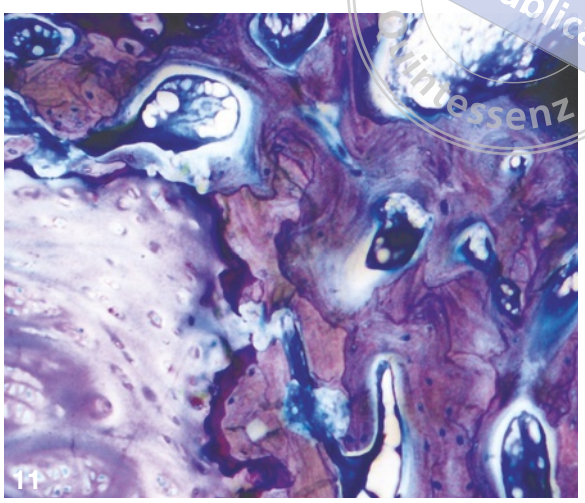


Fig 10 *Process of intramembranous bone formation. In purple, mineralized islands surrounded by connective matrix are observed. Toluidine blue and Pyronin yellow staining; ×400 magnification.* ***Fig 11*** *Process of endochondral bone formation. Cartilage can be seen on the left of the image, which is progressively replaced by bone tissue (right). Toluidine blue and Pyronin yellow staining; ×400 magnification.*

REFERENCES

1. Dellavia C, Gagliano N, Orlando F. I colori e le forme della ricerca in odontoiatria. Atlante di istologia orale. Quintessenza Italy, 2011.
2. Al-Qtaitat AI, Aldalaen SM. A review of noncollagenous proteins; their role in bone. Am J Life Sci 2014;2: 351–355.
3. Mundy GR. Bone Remodeling and its Disorders. Martin Dunitz, 1995.
4. Nudelman F, Lausch AJ, Sommerdijk NAJM, Sone ED. In vitro models of collagen biomineralization. J Struct Biol 2013;183:258–269.
5. George A, Veis A. Phosphorylated proteins and control over apatite nucleation, crystal growth, and inhibition. Chem Rev 2008;108:4670–4693.
6. Massaro EJ, Rogers JM. The Skeleton: Biochemical, Genetic, and Molecular Interactions in Development and Homeostasis. Humana Press, 2004.
7. Nakamura A, Dohi Y, Akahane M, et al. Osteocalcin secretion as an early marker of in vitro osteogenic differentiation of rat mesenchymal stem cells. Tissue Eng Part C Methods 2009;15:169–180.
8. Wanby P, Nobin R, Von S-P, Brudin L, Carlsson M. Serum levels of the bone turnover markers dickkopf-1, sclerostin, osteoprotegerin, osteopontin, osteocalcin and 25-hydroxyvitamin D in Swedish geriatric patients aged 75 years or older with a fresh hip fracture and in healthy controls. J Endocrinol Invest 2016;39: 855–863.
9. Carmagnola D, Botticelli D, Canciani E, Rossi F, Milani S, Dellavia C. Histologic and immunohistochemical description of early healing at marginal defects around implants. Int J Periodontics Restorative Dent 2014;34:e50–e57.
10. Tang CH, Yang RS, Huang TH, Liu SH, Fu WM. Enhancement of fibronectin fibrillogenesis and bone formation by basic fibroblast growth factor via protein kinase C-dependent pathway in rat osteoblasts. Mol Pharmacol 2004;66:440–449.

11. Varoni E, Canciani E, Palazzo B, et al. Effect of Poly-L-Lysine coating on titanium osseointegration: From characterization to in vivo studies. J Oral Implantol 2015;41:626–631.
12. Canciani E, Dellavia C, Marazzi MG, et al. RNA isolation from alveolar bone and gene expression analysis of RANK, RANKL and OPG: A new tool to monitor bone remodeling and healing in different bone substitutes used for prosthetic rehabilitation. Arch Oral Biol 2017;80:56–61.
13. Hayman AR. Tartrate-resistant acid phosphatase (TRAP) and the osteoclast/immune cell dichotomy. Autoimmunity 2008;41:218–223.
14. Klein-Nulend J, van Oers RF, Bakker AD, Bacabac RG. Nitric oxide signaling in mechanical adaptation of bone. Osteoporos Int 2014;25:1427–1437.
15. Frost HM. A 2003 update of bone physiology and Wolff's Law for clinicians. Angle Orthod 2004;74:3–15.
16. Berglundh T, Abrahamsson I, Lindhe J. Bone reactions to longstanding functional load at implants: An experimental study in dogs. J Clin Periodontol 2005;32:925–932.
17. Pellegrini G, Toma M, Francetti L, Cavalli N, Carmagnola D, Dellavia C. Implant placement in sites with adverse anatomical and biomechanical conditions: Critical review. Dental Cadmos 2018;86;9–23.
18. Heino TJ, Kurata K, Higaki H, Väänänen HK. Evidence for the role of osteocytes in the initiation of targeted remodeling. Technol Health Care 2009;17:49–56.
19. Lindhe J, Lang NP, Karring T. Clinical Periodontology and Oral Implantology, ed 4. Blackwell, 2010.
20. Zhou S, Yang Y, Ha N, et al. The specific morphological features of alveolar bone. J Craniofac Surg 2018;29:1216–1219.
21. Lekholm U, Zarb G. Patient selection and preparation. In: Brånemark PI, Zarb G, Albrektsson T (eds). Tissue-Integrated Prostheses: Osseointegration in Clinical Dentistry. Quintessence, 1985:199–209.
22. Devlin H, Horner K, Ledgerton D. A comparison of maxillary and mandibular bone mineral densities. J Prosthet Dent. 1998;79:323–7.
23. von Wowern N, Stoltze K. Pattern of age related bone loss in mandibles. Scand J Dent Res 1980;88:134–146.
24. Hollinger JO, Srinivasan A, Alvarez-Urena P, et al. Bone tissue engineering: Growth factors and cytokines. Elsevier 2011;5:281–300.
25. Pazzaglia UE, Zarattini G, Giacomini D, Rodella L, Menti AM, Feltrin G. Morphometric analysis of the canal system of cortical bone: An experimental study in the rabbit femur carried out with standard histology and micro-CT. Anat Histol Embryol 2010;39:17–26.
26. Marsell R, Einhorn TA. The biology of fracture healing. Injury 2011;42:551–555.
27. Raisz LG. Physiology and pathophysiology of bone remodeling. Clin Chem 1999;45:1353–1358.
28. Matsuo K, Irie N. Osteoclast-osteoblast communication. Arch Biochem Biophys 2008;473:201–209.
29. Goldring SR. Inflammatory mediators as essential elements in bone remodeling. Calcif Tissue Int 2003;73:97–100.
30. Greenstein G, Cavallaro J, Tarnow D. Assessing bone's adaptive capacity around dental implants: A literature review. J Am Dent Assoc 2013;144:362–368.
31. Zhang W, Tekalur SA, Baumann M, McCabe LR. The effects of damage accumulation on the tensile strength and toughness of compact bovine bone. J Biomech 2013;46:964–972.
32. Koehne T, Vettorazzi E, Küsters N, et al. Trends in trabecular architecture and bone mineral density distribution in 152 individuals aged 30-90 years. Bone 2014;66:31–38.
33. Chaichanasakul T, Kang B, Bezouglaia O, Aghaloo TL, Tetradis S. Diverse osteoclastogenesis of bone marrow from mandible versus long bone. J Periodontol 2014;85:829–836.
34. Liu XL, Li CL, Lu WW, Cai WX, Zheng LW. Skeletal site-specific response to ovariectomy in a rat model: Change in bone density and microarchitecture. Clin Oral Implants Res 2015;26:392–398.
35. Dellavia C. Compendio di anatomia oro-facciale per l'attività clinica odontostomatologica. Edises, 2016.
36. Mjör IA, Fejerkov O. Embriologia e istologia del cavo orale. Edi-Ermes, 1988.

SURGICAL ANATOMY OF THE MAXILLA AND MANDIBLE

CHAPTER 2

Edited by Filippo Fontana and Roberto Pistilli

INTRODUCTION

The purpose of this chapter is to provide clear and concise information on the main anatomical structures of interest to the oral surgeon.

The anatomical analysis is organized with the oral cavity divided into four areas: the mandibular symphysis, the posterior mandible (mandibular body), the anterior maxilla, and the posterior maxilla. Each area is further subdivided into the lingual aspect, the bone plane, and the buccal aspect.

This subdivision allows for easy and immediate identification of the relevant anatomical structures in each area where surgery is performed.

CHIN SYMPHYSIS AND INTRAFORAMINAL AREA

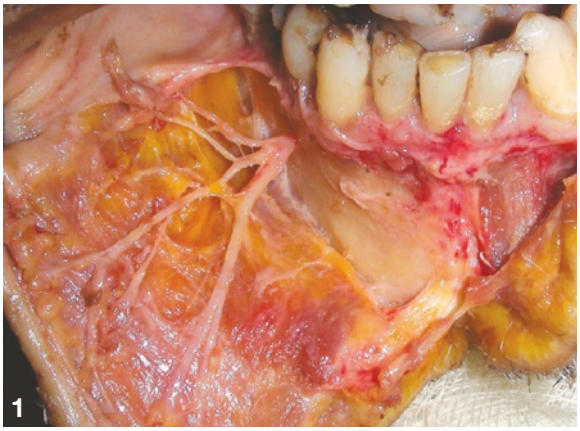
The mandibular symphysis is the portion of the mandible that lies between the two mental foramina. It is a strategically significant area because it is the site of many implant-supported restorations. This area encompasses several anatomical structures.

Buccal aspect

Mental nerve

The mental nerve is an anatomical structure of great significance for oral surgeons because many surgical procedures are performed in its proximity.

> *Successful implant therapy and the prevention of surgical complications depend on a thorough knowledge of the topographic anatomy of the jaw bones and surrounding anatomical structures.*
>
> *Massimo Simion*



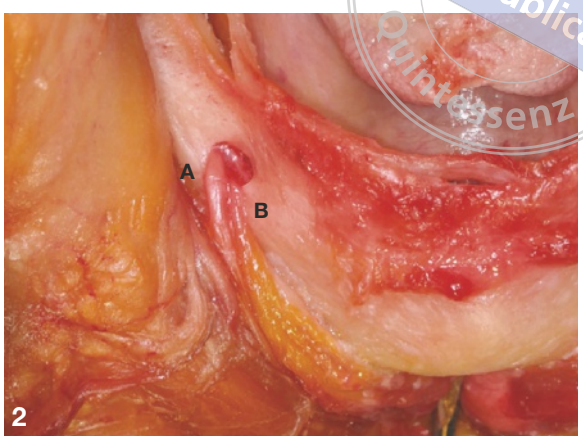


Fig 1 *Course and branches of the neurovascular bundle and the mental artery from the mental foramen to the submucosa of the lip.* ***Fig 2*** *Evidence of the mental foramen in the body of the mandible, where the neurovascular complex of the same name emerges. A: mental nerve; B: mental artery.*

Together with the incisive nerve, it is one of the terminal branches of the inferior alveolar nerve. It exits the body of the mandible through the mental foramen and branches out into the submucosa of the lower lip. It normally divides into three branches. One branch turns forward and downward to innervate the skin of the chin, and the other two turn anteriorly and upward to innervate the skin and mucosa of the lower lip and the mucosa of the inferior alveolar surface **(Fig 1)**.

Mental artery

The mental artery is the most voluminous of the terminal branches of the inferior alveolar artery. It emerges from the bony compartment at the level of the mental foramen to perfuse the soft tissues of the chin and lower lip. It anastomoses with branches of the inferior labial artery.

Mental foramen

The mental foramen is an opening in the mandibular canal at the lateral surface of the body of the mandible from which the mental neurovascular bundle emerges **(Fig 2)**. The mental foramen is frequently located between the first and second premolars or sometimes even inferior to the second premolar. On average, it lies at the midpoint between the inferior border of the mandible and the upper alveolar margin. It is important to note that the mandibular canal does not reach the mental foramen perpendicularly. It frequently turns externally upward and backward, forming a sort of knee bend. It is very important to know this anatomical feature to avoid damaging the neurovascular bundle. In edentulous patients, the mental foramen becomes more superficial occlusally with advancing bone atrophy. Radiographic and clinical identification of the mental foramen is fundamental in all oral surgical procedures.

Bone plane

Incisive nerve

The incisive nerve is one of the two terminal branches of the inferior alveolar nerve. It forms at the level of the premolar region after the other terminal branch (the mental nerve) leaves the body of the mandible through the mental foramen. The incisive nerve is smaller than the mental nerve and runs inside the body of the mandibular symphysis. It supplies dental, interdental, and interalveolar branches to the mandibular anterior teeth and the

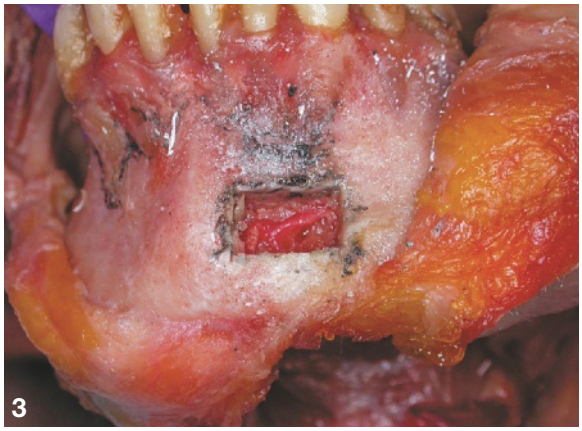


Fig 3 *The incisive nerve and its artery in the body of the mandible running in the direction of the mandibular symphysis.*

corresponding periodontal complex. Clinical and radiographic identification of the incisive nerve is challenging.

Incisive artery

The incisive artery follows the course of the incisive nerve to form a neurovascular bundle. It is the terminal, smaller branch of the bifurcation of the inferior alveolar artery. It runs inside the mandible until reaching the midline and anastomosing with the contralateral artery **(Fig 3)**.

Lingual aspect

Sublingual artery

At the lingual aspect of the mandibular symphysis, it is important to check for the presence of terminal perforating branches of the anastomosis between the sublingual artery (a branch of the lingual artery) and the submental arteries (branches of the facial artery). Careful analysis with CBCT can be used to provide precise indications of the presence and location of these vessels. In the event of incorrect implant site preparation with perforation of the lingual cortical, a lesion of this anastomosis can occur with consequent and potentially dangerous extraosseous bleeding **(Fig 4)**.

MANDIBULAR BODY

The posterior mandible is an area of enormous anatomical interest to the dentist and oral surgeon because it is the site of numerous surgical procedures, ranging from simple tooth extraction to extensive bone regeneration for implant placement purposes. This area is characterized by the presence of many structures on its lingual and buccal aspects, as well as structures inside the body of the bone.

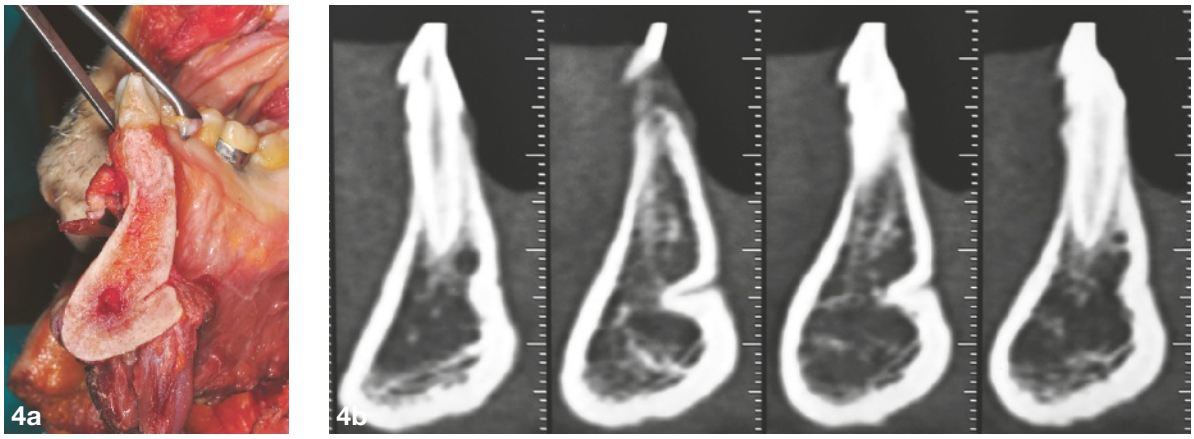
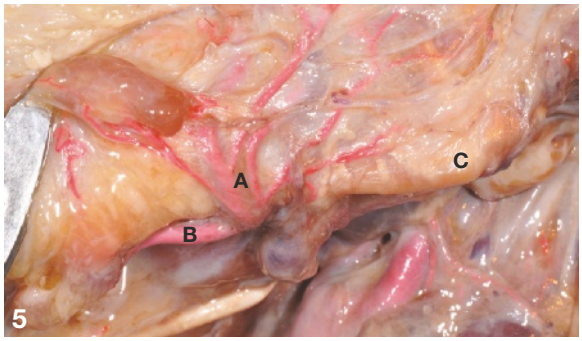


Fig 4 *Image and corresponding CT sections of a perforating branch from an anastomosis between the sublingual artery and submental artery at the lingual aspect of the mandibular symphysis.*



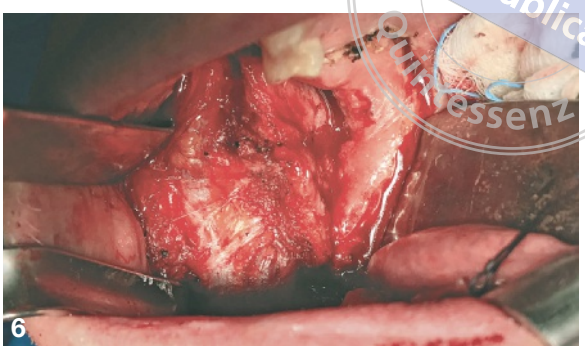


Fig 5 *A: facial artery; B: submental artery; C: angle of the mandible.* ***Fig 6*** *The buccal nerve running buccal to the third molar along the external oblique line.*

Buccal aspect

Facial artery

The facial artery is one of the anterior branches of the external carotid artery. It may be of significance for oral surgery in its course from the submandibular triangle to the lateral margin of the nose **(Fig 5)**. In fact, this artery reaches the inferior border of the mandible in front of the anterior limit of the masseter muscle to turn around the mandibular body and thus pass into the facial tissues in the direction of the labial commissure.

At the level of the mandibular molars, this vessel can be reached from the oral cavity by incising the buccal mucosa and the buccinator and passing through the underlying adipose tissue.

It is possible to damage the facial artery in the process of performing partial-thickness flap elevation. Therefore, whenever possible, it is important to perform a subperiosteal elevation and prevent the flap from rotating and protect it from sharp instruments.

Buccal nerve

The buccal nerve is a sensory nerve that is distributed to the mucosal surface of the cheek, the gingiva of the mandibular molars, and the skin near the labial commissure. It is a branch of the mandibular nerve, which itself is the third branch of the trigeminal nerve, and crosses the submucosa at the level of the retromolar trigone **(Fig 6)**.

Bone plane

Inferior alveolar nerve

Many oral surgical procedures are performed in proximity to the inferior alveolar nerve. It is the intermediate branch of the mandibular nerve and runs lateral to the lingual nerve between the pterygoid muscles. It is a sensory nerve that enters the body of the mandible through the mandibular foramen at the level of the lingula (ie, Spix spine) and then runs entirely inside the mandibular canal to end in its two final branches, the incisive and mental nerves. Before entering the body of the mandible, the inferior alveolar nerve first emits the lingual nerve and then the mylohyoid nerve. During its course through the canal, the inferior alveolar nerve emits the lower dental branches destined for the posterior mandibular teeth and the corresponding periodontal complex.

Inferior alveolar artery

The inferior alveolar artery is a branch of the first (mandibular) segment of the maxillary artery. It follows the same course as the nerve of the same name in the mandibular canal, issuing dental and alveolar branches.

Mandibular canal

The endosseous mandibular canal represents a very useful radiographic marker for dentists and oral surgeons because it can be used to identify the course of the inferior alveolar neurovascular (plexus) bundle. It begins at the mandibular foramen, descends downward along the ramus, and then folds forward with a horizontal course inferior to the roots of the mandibular molars. At the premolar region, it divides into two canals: the narrower incisive canal, which continues the course of the mandibular canal, and the mental canal, which bends laterally upward and backward to open into the mental foramen **(Fig 7)**.

Lingual aspect

Mylohyoid nerve

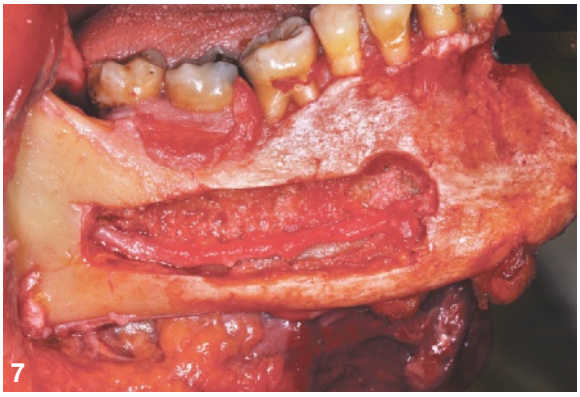
The mylohyoid nerve is separated from the inferior alveolar nerve before its entry into the body of the mandible. It turns downward, running into the mylohyoid line of the mandible, and then heads toward the floor of the oral cavity **(Fig 8)**. It sends branches to the mylohyoid muscle and the anterior belly of the digastric muscle. In 10% of cases, a sensory branch of this nerve penetrates the mandible at the level of the mandibular symphysis and participates in the innervation of the mandibular incisors. This anatomical variant can be very important for providing local anesthesia.

Lingual nerve

The lingual nerve is a sensory branch of the mandibular nerve that carries fibers for general sensitivity (touch, pressure, pain, temperature) to the tongue and sublingual region. On its way, it also receives visceral effector and gustatory fibers of the facial nerve via the tympanic cord. It initially runs close to the inferior alveolar nerve and then passes more medial and anterior, running lateral to the internal pterygoid muscle. In the posterior part of the oral cavity, at the lingual surface of the mandible at the level of the second and third molars, this nerve can be very superficial, just below the mucosa, which is of importance when performing surgery in the retromolar space, especially when extracting mandibular third molars **(Fig 9)**.

Mylohyoid artery

The mylohyoid artery originates from the inferior alveolar artery before it enters the mandibular canal. It follows the course of the mylohyoid nerve along the mylohyoid line **(Fig 10)**. The mylohyoid artery can be lacerated if implant site preparations



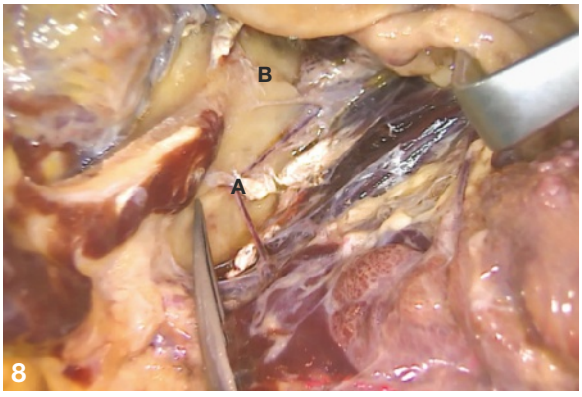
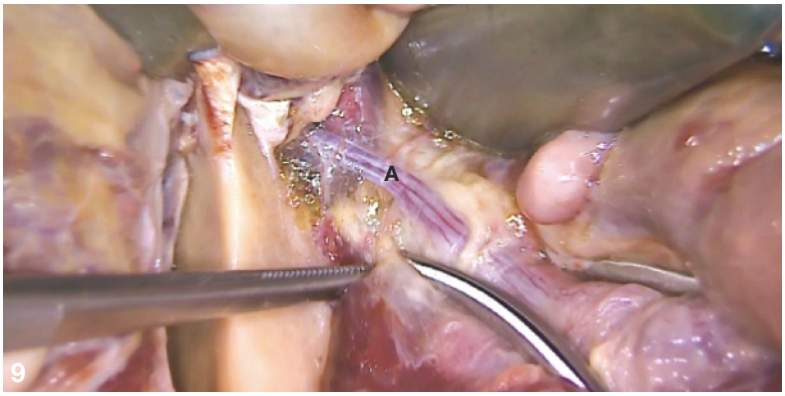
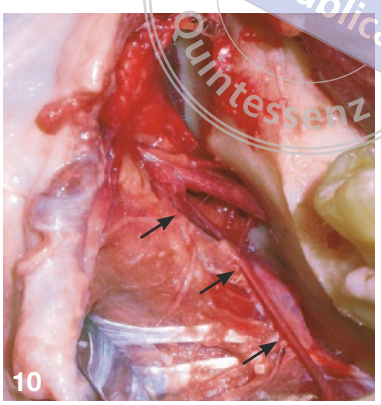


Fig 7 *Evidence of the mandibular canal and the course of the inferior alveolar nerve bundle.* ***Fig 8*** *Course of the mylohyoid nerve on the lingual aspect of the mandibular body anterior to the mandibular lingula. A: mylohyoid nerve; B: mandibular lingula.*





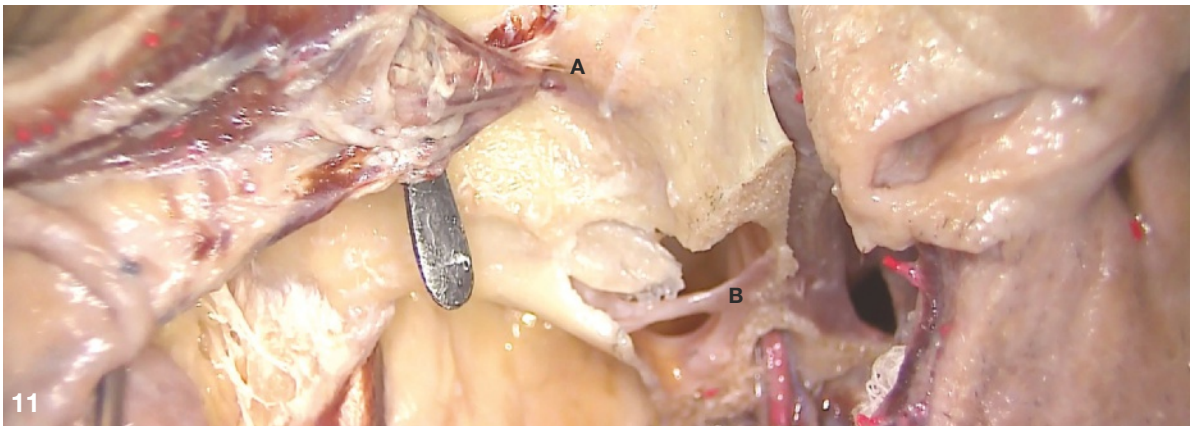


Fig 9 *Course of the lingual nerve near the body of the mandible at the level of the molars. A: lingual nerve.* ***Fig 10*** *Origin and course of the mylohyoid artery on the lingual aspect of the mandible.* ***Fig 11*** *The infraorbital neurovascular bundle emerging from the homonymous foramen and the descending palatine artery. A: infraorbital neurovascular bundle; B: descending palatine artery.*

DURATION: 27'29"

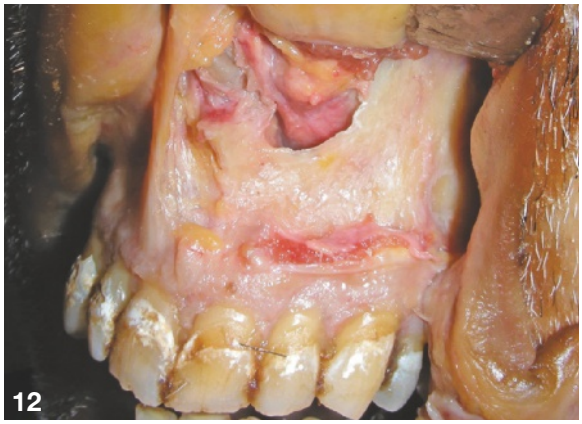
VIDEO: 1
ANATOMICAL DISSECTION OF THE MANDIBLE

(DR R. PISTILLI, A. NISI)

puncture the lingual cortical, which can result in significant bleeding. Special attention must be given to the location of the mylohyoid artery in patients where the body of the mandible presents with an undercut at the level of the molars. The anatomical situation must be identified during surgical planning, both by palpating the lingual surface of the mandible and by performing a CBCT examination.

ANTERIOR MAXILLA

The anterior maxilla is delimited by the maxillary canines and is the site of many dental surgeries, many of which aim to improve esthetics.



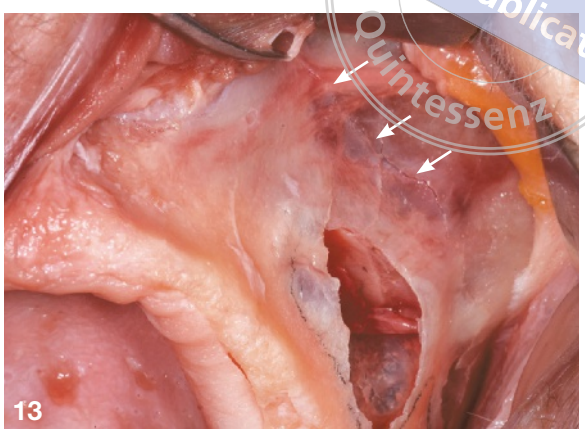


Fig 12 *Pyriform aperture at the level of the anterior maxilla.* ***Fig 13*** *Course of the canalis sinuosus.*

Buccal aspect

Infraorbital nerve

The infraorbital nerve is the intermediate branch of the maxillary nerve, which is the second branch of the trigeminal nerve. It enters the oral cavity through the infraorbital foramen before fanning out into its terminal segments. Its branches innervate a very large area that encompasses the lower eyelid, the wing of the nose and nasal pyramid, and the mucosa and skin of the upper lip. In its bony course, it also emits alveolar branches directed to the maxillary teeth and the corresponding periodontium **(Fig 11)**.

Infraorbital foramen

The infraorbital foramen is the opening of the infraorbital canal and is located approximately 5 to 8 mm inferior to the midpoint of the inferior orbital rim and 4 to 5 mm medial to a vertical line passing through the center of the pupil. It is the exit point of the infraorbital neurovascular bundle. The infraorbital foramen serves as an anatomical landmark of clinical relevance in procedures involving extensive bone regeneration or the insertion of zygomatic implants, as well as when performing extractions or the enucleation of cystic lesions in nearby teeth.

Bone plane

Nasal floor

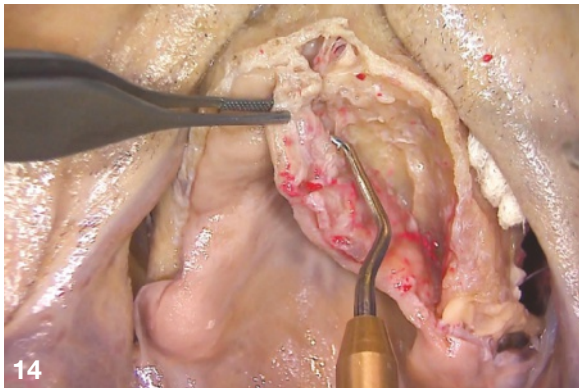
The nasal cavities are located superior to the anterior maxilla.

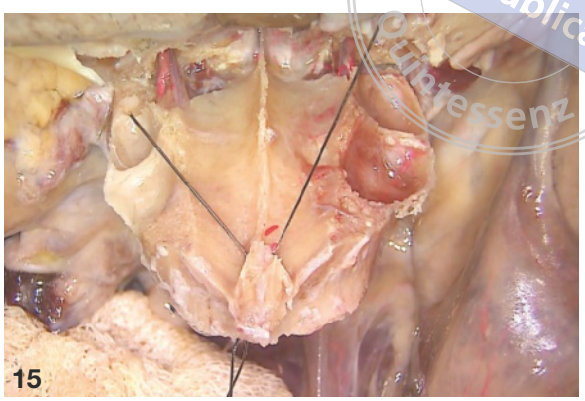
The anterior portion of the nasal floor consists of the maxillary palatine process, which is crossed at the midline by the incisive or nasopalatine canal. Especially in cases of extensive resorption of the anterior superior maxilla, it is possible to identify the pyriform aperture (ie, the common anterior orifice of the nasal cavities) by elevating the vestibular flap, which will give the perception that the nasal floor is inferior to the pyriform aperture **(Fig 12)**.

Canalis sinuosus

The canalis sinuosus is a neurovascular canal branching from the infraorbital canal. The anterior alveolar nerve runs through the canalis sinuosus, directed mesially in the canine region of the anterior maxilla.

The canalis sinuosus runs between the nasal cavity and the anterior margin of the sinus cavity. If this canal is disturbed during an implant placement procedure, discomfort and/or pain may result in the anterior maxilla **(Fig 13)**.





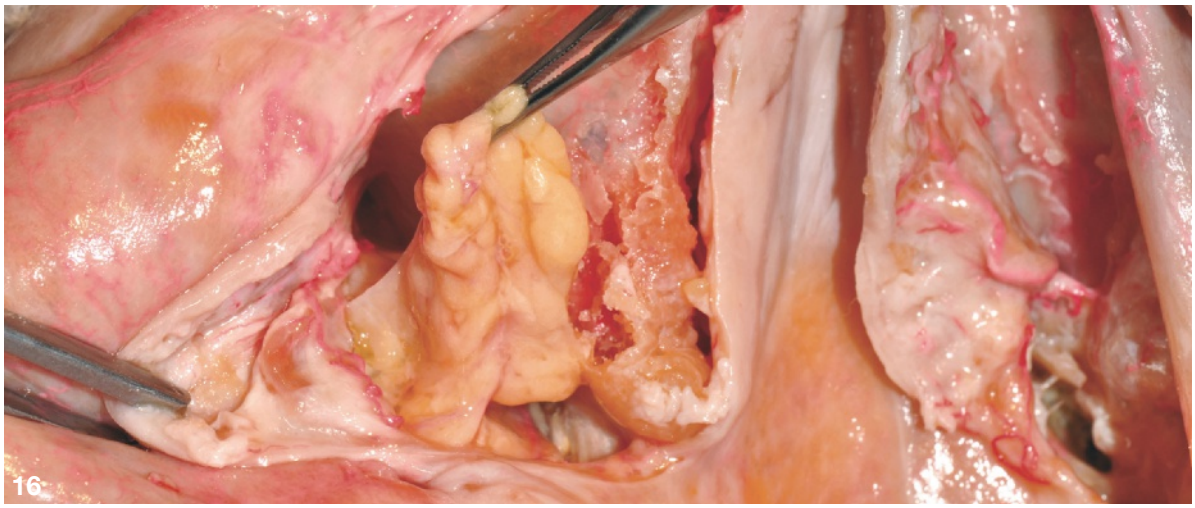


Fig 14 *The nasopalatine nerve emergence from the anterior palatine foramen.* ***Fig 15*** *The incisive canal at the level of the anterior maxilla is highlighted by the insertion of a wire.* ***Fig 16*** *Bichat's fat pad in the vestibular flap.*

Palatal aspect

Nasopalatine nerve

The nasopalatine nerve (Scarpa's nerve) is a nerve trunk that originates from the pterygopalatine nerve, which is an internal branch of the maxillary nerve (the second branch of the trigeminal nerve) **(Fig 14)**. The nasopalatine nerve is a particularly long medial branch that runs along the nasal septum to enter the buccal cavity palatal to the maxillary incisors via the incisive canal. It innervates a small area of the anterior palatine mucosa. It is important to identify this nerve during the surgical phase, though injury to the nerve does not pose problems for the patient. In some cases, intentional resection of this structure is performed for better passivation of the palatal flap.

Anterior palatal foramen

The anterior palatal foramen is the opening of the incisive canal at the level of the anterior maxilla **(Fig 15)** through which the nasopalatine vascular bundle reaches the buccal cavity. It is located on the midline posterior to the maxillary central incisors. In the case of advanced anterior maxillary atrophy, this foramen can be emptied of its

neurovascular component and filled in for the purpose of bone regeneration.

POSTERIOR MAXILLA

The posterior maxilla encompasses the entire area posterior to the canines up to the pterygopalatine fossae. It is affected by several neighboring anatomical structures.

Vestibular aspect

Buccal fat bad

The buccal fat pad (Bichat's fat pad) is a collection of adipose tissue located in the cheek in the area of the maxillary molars. It lies in the space between the masseter and buccinator muscles and can be reached, either intentionally or accidentally, when incising the periosteum for the release of the vestibular flap **(Fig 16)**.

Bone plane

Maxillary sinus

The maxillary sinus is a structure of enormous clinical interest to dental implantologists, both as the upper limit for implant placement and in cases involving bone grafting. The maxillary sinus is the largest paranasal cavity and occupies a large part of the posterior maxillary body. It has a triangular pyramid shape. The base is the vertical lateral wall of the nasal cavity, and the apex is oriented in the direction of the zygomatic process. The roof or upper wall of the maxillary sinus forms the floor of the orbit, the posterior wall is the tuberosity of the maxilla, and the anterior wall is at the canine fossa. This antrum communicates with the nasal cavities via the semilunar hiatus, which is located near the roof of the sinus. The size and extension of this structure are very variable and influenced by several factors. After tooth loss, this structure may extend into the alveolar process. The maxillary sinus is lined on the inside with a bilaminar sinus membrane, also called Schneider's membrane, which is characterized by a ciliated epithelium.

Posterior superior alveolar artery

The posterior superior alveolar artery is a voluminous vessel that originates at the maxillary tuberosity. It is a branch of the maxillary segment of the maxillary artery. In its course, it emits many branches destined for the posterior superior dental elements. The terminal part of this artery supplies the buccal surface of the alveolar process. It is of clinical interest to implantologists not so much because of its area of origin, which is far from dental surgical procedures, but because it contributes to the formation of the alveolar antral artery, which can be injured during sinus elevation procedures.

Alveolo antral artery

The alveolar antral artery originates from the anastomosis of the posterior superior alveolar artery with branches of the infraorbital artery. It normally runs within the bony lateral sinus wall at variable heights that sometimes coincide with the sites chosen for the antrostomy during sinus floor elevation. The diameter of the alveolar antral artery is also very variable, and in some cases, its size is such that it represents a risk factor to be analyzed during the presurgical planning phase.

Lacerating this vessel can result in intraoperative hemorrhages, which although rarely severe, can complicate the operation by interfering with visibility and lead to the appearance of diffuse hematomas and potential pathologic syndromes like hemosinus **(Fig 17)**.

Pterygo-palatine fossa

The pterygopalatine fossa is an anatomical space posterior to the maxillary tuberosity that includes some important anatomical structures, including

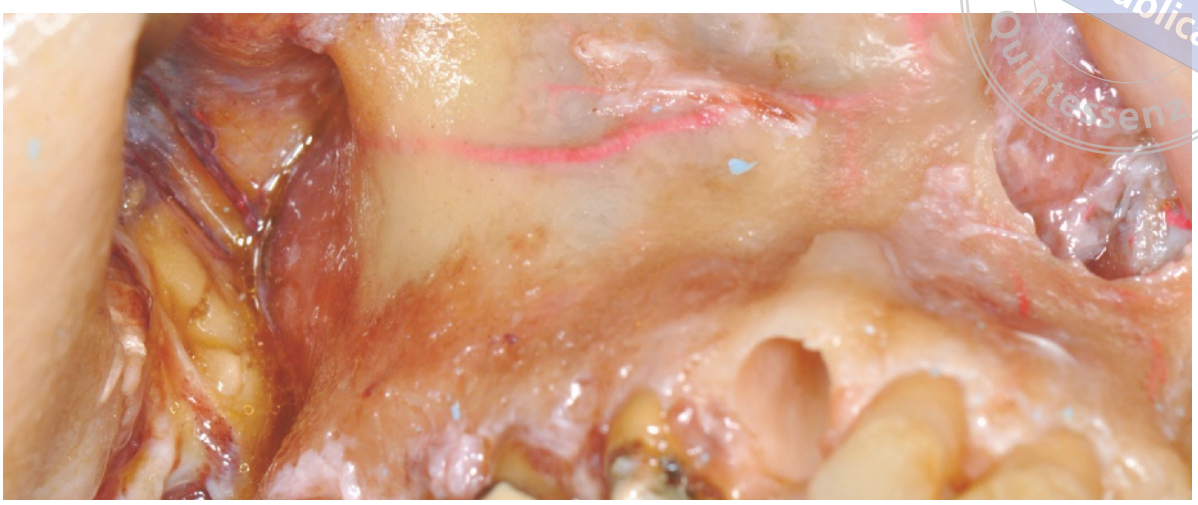


Fig 17 *Course of the alveolar antral artery in the lateral sinus wall.*

the maxillary nerve, the internal maxillary artery, and the pterygoid venous plexus. These anatomical structures are not usually affected by oral procedures, but they may be disturbed by careless anesthetic maneuvers or during tooth extraction.

Palatal aspect

Greater palatine foramen

The greater palatine foramen is the exit of the palatine canal at the level of the oral cavity through which the palatine neurovascular bundle passes. It is located at the level of the hard palate at the second and third maxillary molars, frequently at the transition between the horizontal plane of the hard palate and the vertical plane of the maxillary alveolar process.

It is important to identify this anatomical landmark to avoid injuring the neurovascular bundle during palatal flap elevation or free/pedicle graft harvesting procedures **(Fig 18)**.

DURATION: 33'39"

VIDEO: 2
ANATOMICAL DISSECTION OF THE MAXILLA

(DR R. PISTILLI, A. NISI)

Greater palatine artery

The greater palatine artery is the main branch of the descending palatine artery, which is a branch of the maxillary artery. It enters the oral cavity through the greater palatine foramen.

Emerging from the latter, it turns anteriorly and runs within the submucosa of the palate, frequently resting inside a groove between the hard palate and the alveolar process. Its terminal branch reaches the incisive foramen to anastomose with the branches of the nasopalatine artery. It

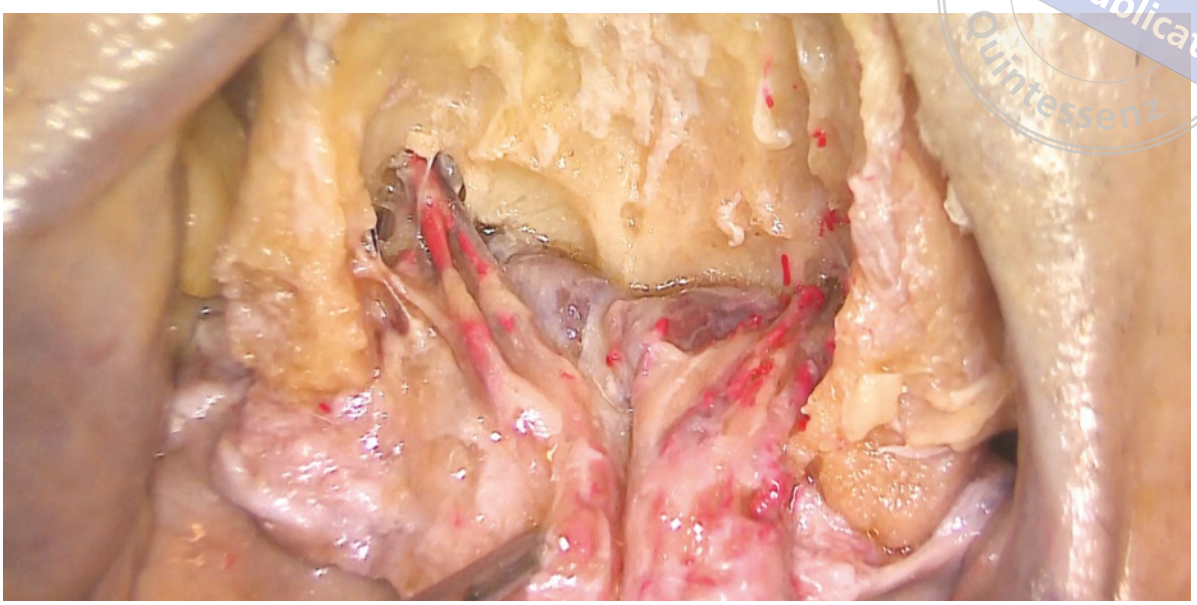


Fig 18 *The major palatine foramina and the emergence of the neurovascular bundle.*

vascularizes the mucosa of the hard palate and the gingiva on the palatal aspect.

Anterior palatine nerve

The anterior palatine nerve originates from the pterygopalatine nerve, which is a branch of the maxillary nerve. It runs in the palatine canal between the maxillary tuberosity and the pterygoid process of the sphenoid bone, exiting through the greater palatine foramen to enter the palate. It follows the course of the greater palatine artery to innervate a large portion of the ipsilateral palate and the gingiva of the palatal aspect of the alveolar process.

RECOMMENDED READINGS

Du Brul LE. Anatomia Orale di Sicher. Milano: Edi Ermes, 1982.

Netter FH. Atlante di anatomia umana. Milano: Edra, 1989.

Pistilli R, Felice P. Anatomia e chirurgia del cavo orale - Mandibola e Pavimento Orale. Pescara: Editalia Medica, 2018.

"After working in the field of osseointegrated dental implants and bone regeneration for 35 years, I decided to write a book that would encompass my entire journey with these fascinating tools. My aim was to produce a work available to everyone—a useful textbook for dental students; a helpful guide for those approaching osseointegrated implant techniques for the first time; and a handbook for experts wishing to refine their techniques for bone regeneration, peri-implant soft tissue management, and the satisfactory resolution of esthetically complex cases.

I spent over 3 years completing this book, searching for historic and recent clinical cases to compile a comprehensive collection of concepts that can be used to solve almost all challenges that may be confronted in implant and regenerative clinical practice.

I dedicate this book to all those who realize that being an "expert" means learning to ask more and more questions and that many of those questions remain unanswered."

M. Simion

ISBN 978-1-78698-141-7

9 781786 981417

www.quintessence-publishing.com