Jacques A. Baart · Henk S. Brand *Editors*

Local Anaesthesia in Dentistry

Second Edition

Springer

Local Anaesthesia in Dentistry

Jacques A. Baart
Henk S. Brand
Editors

Local Anaesthesia in Dentistry

Second Edition

Springer

Editors
Jacques A. Baart
Vrije University Medical Center
Amsterdam, The Netherlands

Henk S. Brand
Academic Centre for Dentistry Amsterdam
Amsterdam, The Netherlands

This work has been first published in 2013 by Bohn Stafleu van Loghum, The Netherlands with the following title: Lokale anesthesie in de tandheelkunde; tweede, herziene druk.

The first edition of the English language edition was first published in 2008 by Wiley-Blackwell with the following title: Local Anaesthesia in Dentistry.

ISBN 978-3-319-43704-0 ISBN 978-3-319-43705-7 (eBook)
DOI 10.1007/978-3-319-43705-7

Library of Congress Control Number: 2017937372

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

This book is dedicated to the memory
of Theo van Eijden en Frans Frankenmolen

Foreword

Patients in current dental care expect painless treatment. To this end, local anaesthesia is the key factor. Modern local anaesthetics are very efficient and safe drugs, and the great majority of patients will not encounter unpleasant side effects nor lasting local or systemic complications. The history of local anaesthesia goes back more than a century, and yet further developments in biological insight and clinical management are still ongoing.

This second edition of *Local Anaesthesia in Dentistry* has been written primarily for dental students, and as such, it covers basic knowledge and recent achievements alike. The editors have produced a balanced composition of essentials within pain physiology, neuroanatomy, pharmacology, aspects related to equipment and techniques for anaesthetising the regions of the oral cavity, local and systemic adverse events, special considerations in children, etc.

Improvements in this second edition of the English version include 45 more pages with new illustrations, a chapter on computer-assisted local anaesthesia, more boxes to emphasise facts, and much more.

The book was originally written and edited in Dutch by Dr. Baart and Dr. Brand. Like the translation of the first edition, the written English is flowing in an easy-to-read style with highlights in boxes and photographic and artistic figures of excellent quality.

The editors must be complimented for the success of an affordable, well-written, and edited textbook to provide theoretical background and practical guidance for dental students in the essentials of local anaesthesia. Also dental practitioners may benefit from the book to bring them on level with current standards.

Søren Hillerup DDS, PhD, Dr Odont
Professor Em., Maxillofacial Surgery
Copenhagen 2017

Contents

Contributors

J.K.M. Aps
University of Washington
Department of Oral Medicine
Seattle, USA
johan.apsdmfr@hotmail.com

J.A. Baart
Academic Centre for Dentistry Amsterdam/
Vrije Universiteit Medical Centre
Department of Oral and Maxillofacial Surgery
Amsterdam, The Netherlands
ja.baart@vumc.nl

L.H.D.J. Booij
Radboud University Medical Centre Nijmegen
Department of Anaesthesiology
Nijmegen, The Netherlands
lhdj.booij@upc.mail.nl

J.F.L. Bosgra
Tergooi hospital, Department of Oral and
Maxillofacial Surgery
Hilversum, The Netherlands
jflbosgra@hotmail.com

H.S. Brand
Academic Centre for Dentistry Amsterdam
Department of Oral Biochemistry
Amsterdam, The Netherlands
hbrand@acta.nl

W.G. Brands
Royal Dutch Dental Association
Nieuwegein, The Netherlands
wbrands1@kpnmail.nl

F.W.A. Frankenmolen[†]
Paediatric Dental Centre
Beuningen, The Netherlands

A.L. Frankhuijzen
Vrije Universiteit Medical Centre
Department of Pharmacology
Amsterdam, The Netherlands
bram.ank@zonnet.nl

G.E.J. Langenbach
Academic Centre for Dentistry Amsterdam
Department of Functional Anatomy
Amsterdam, The Netherlands
g.langenbach@acta.nl

H.P. van den Akker
Academic Centre for Dentistry Amsterdam/
Academic Medical Centre
Department of Oral and Maxillofacial Surgery
Amsterdam, The Netherlands
hpvandenakker@gmail.com

T.M.G.J. van Eijden[†]
Academic Centre for Dentistry Amsterdam
Department of Functional Anatomy
Amsterdam, The Netherlands

[†]Authors were deceased at the time of publication.

Introduction: A Short History of Local Anaesthesia

General anaesthesia already existed before local anaesthesia became available. Actually, general anaesthesia was introduced by the American dentist Horace Wells. In 1844, together with his wife Elizabeth, he witnessed a demonstration whereby the circus owner Colton intoxicated a number of volunteers with laughing gas. One of the volunteers hit himself hard on a chair but did not even grimace. Horace Wells noticed this and concluded that a patient, having inhaled laughing gas, might be able to undergo an extraction without pain. A few days later, Wells took the experiment upon himself and asked a colleague to extract one of his molars after he had inhaled some laughing gas. It was a success. Wells independently organised some additional extraction sessions, after which the Massachusetts General Hospital invited him for a demonstration. This demonstration turned out to be a fiasco. The patient was insufficiently anaesthetised since not enough laughing gas was administered. Wells' life, which had initially been so successful, became a disaster. The physician Morton, a previous assistant to Wells, absconded with the idea of general anaesthesia, but used ether instead of laughing gas for a 'painless sleep'. Morton denied in every possible way that he had stolen the idea from Wells. Wells was greatly incensed by this. Furthermore, Wells was no longer able to practise as a dentist. He became a tradesman of canaries and domestic products and became addicted to sniffing ether. Eventually he was imprisoned for throwing sulphuric acid over some ladies of easy virtue. At the age of 33 years, he made an end to his life in prison by cutting his femoral artery.

The discovery of local anaesthesia is a very different story. One of the first to gain experience with this form of anaesthesia was Sigmund Freud, in 1884. Freud experimented with the use of cocaine. Cocaine had been used for several centuries by the Incas in Peru to increase their stamina. Freud used cocaine in the treatment of some of his patients and then became addicted himself. The German surgeon August Bier observed a demonstration in 1891, whereby the internist Quincke injected – for diagnostic purposes – a cocaine solution into a patient's epidural area, thus anaesthetising and paralysing the legs. Bier took this discovery to his clinic in Kiel and decided to try the technique first on himself and only thereafter to operate on patients under local anaesthesia. Together with his colleague, senior doctor Hildebrandt, he decided to perform an experiment. Bier volunteered to be the guinea pig, and Hildebrandt administered a spinal injection to his boss. This failed, however, due to the fact that the syringe containing the cocaine solution did not fit the needle so a lot of liquor leaked through the needle. It was then Hildebrandt's turn as the test subject, and Bier succeeded in administering an epidural anaesthesia with a cocaine solution. After a few minutes Hildebrandt reported that his leg muscles were numb and his legs were tingling. Bier tested the efficacy of the local anaesthesia by sticking a large injection needle deep into Hildebrandt's upper leg. Hildebrandt did not feel a thing, even when Bier hit his femur skin hard with a wooden hammer. After 45 min, the local anaesthetic began to wear off. The gentlemen then went out for dinner and enjoyed cognacs and good cigars. The next morning, however, the local and systemic disadvantages of this local anaesthesia came to light. Bier had a raging headache after his failed anaesthetic, which lasted 1 week and

would only go away if he lay down. Nevertheless, he continued to operate. Hildebrandt was in worse shape. The next day he called in sick; he felt dizzy and was vomiting continually. Walking was difficult, partly because of haemorrhages in his upper and lower leg. On the basis of all these disadvantages, Bier concluded that he would refrain from treating his patients under local anaesthesia. Later, Bier strayed from regular medicine and became an alternative medicine fanatic. However, Bier's extensive observations and descriptions of his experiments with local anaesthesia did not go unnoticed.

In 1899, the French surgeon Tuffer was unaware of Bier's work but operated on a young lady with a hip sarcoma under local anaesthesia, applying a cocaine solution to the spinal canal. Several years later, he operated on patients under local anaesthesia in the kidney, stomach, and even the thoracic wall. The first use of local anaesthesia in dentistry is attributed to the American Halsted, who anaesthetised himself with a cocaine solution.

Because of the high toxicity and addictive effects of cocaine, a safer local anaesthetic was sought. This was eventually found in 1905 in the form of procaine, an ester derivative of cocaine. Procaine became known under the brand name of Novocaine ('the new cocaine'). This remedy was used for many years, but after a while, a stronger anaesthetic was needed. During the Second World War, the Swedish scientist Nils Lofgren succeeded in making the amide compound lidocaine. Lidocaine remedy works faster and more effectively than cocaine and is not addictive. However, how to administer the local anaesthetic remained a problem. In 1947, the American company Novocol marketed the cartridge syringe, glass cartridges with local anaesthetic and disposable needles. With this, modern local anaesthesia was born. Lidocaine and articaine, which was introduced in the 1970s, are now the most commonly used local anaesthetics in dentistry.

J.A. Baart
J.F.L. Bosgra

Further Reading

1. Bennion E. Antique dental instruments. London: Sotheby's Publications; 1986.
2. Richards JM. Who is who in architecture, from 1400 to the present day. London: Weidefeld and Nicolson; 1977.
3. Sydow FW. Geschichte der Lokal- und Leitungsanaesthesie. In: Zinganell K, editor. Anaesthesie – historisch gesehen. Berlin/Heidelberg: Springer; 1987.

Pain and Impulse Conduction

L.H.D.J. Booij

J.A. Baart, H.S. Brand (eds.), *Local Anaesthesia in Dentistry*, DOI 10.1007/978-3-319-43705-7_1

1

According to the World Health Organization, pain is defined as an 'unpleasant sensation that occurs from imminent tissue damage'. From a physiological perspective, pain is a warning system. During dental treatment, patients will experience pain as something unpleasant. Pain will also make it impossible for the dentist to work accurately.

1.1 Pain Receptors

Pain stimuli are primarily generated by the relatively amorph sensory nerve endings of the Aδ and C fibres. These free nerve endings (nociceptors; see Fig. 1.1) are sensitive to a variety of mechanical, thermal and chemical stimuli and are therefore called polymodal. Nociceptors do not display adaptation: nociceptive responses will occur as long as the stimulus is present. Nociceptors have a high threshold for activation, so only potentially noxious stimuli are detected. The detection of the stimulus is performed by the receptors, present on the sensory nerve endings. They consist of ion channels that respond to mechanical stimulation, temperature or chemical substances. The conversion of the stimulus into an electric signal is called transduction.

During tissue damage, several substances are released that are able to stimulate the nociceptors, such as histamine, serotonin, bradykinin, prostaglandin E_2 and interleukins. These substances activate the nociceptors and reduce their threshold (sensitisation). There is also feedback regulation from the central nervous system. Once pain has been observed, the receptors become more sensitive for nociceptive stimuli. This mechanism plays a role in the development of chronic pain.

Nociceptors are also present in the teeth and the oral cavity and are usually sensitive to a specific neurotransmitter. Important are the fluid-filled canals in the dentine, where free endings of the trigeminal nerve are present which are able to register pressure changes in the canals. Odontoblasts may play an additional role by releasing calcium and ATP. This ATP stimulates the endings of the trigeminal nerve. The sensory nervous system also contains 'physiological' sensors. These are small end organs of the sensory nerves, such as the Krause, Meissner and Pacini bodies (see Fig. 1.2). These 'physiological' sensors usually only respond to one specific stimulus (warmth, touch, smell, etc.) and are, as such, unimodal. Besides this, they exhibit the phenomenon of adaptation; the response to a stimulus disappears during prolonged or persistent stimulation. In the case of excessive stimulation, these 'physiological' sensors may also initiate pain sensation. Transduction occurs in ion channels.

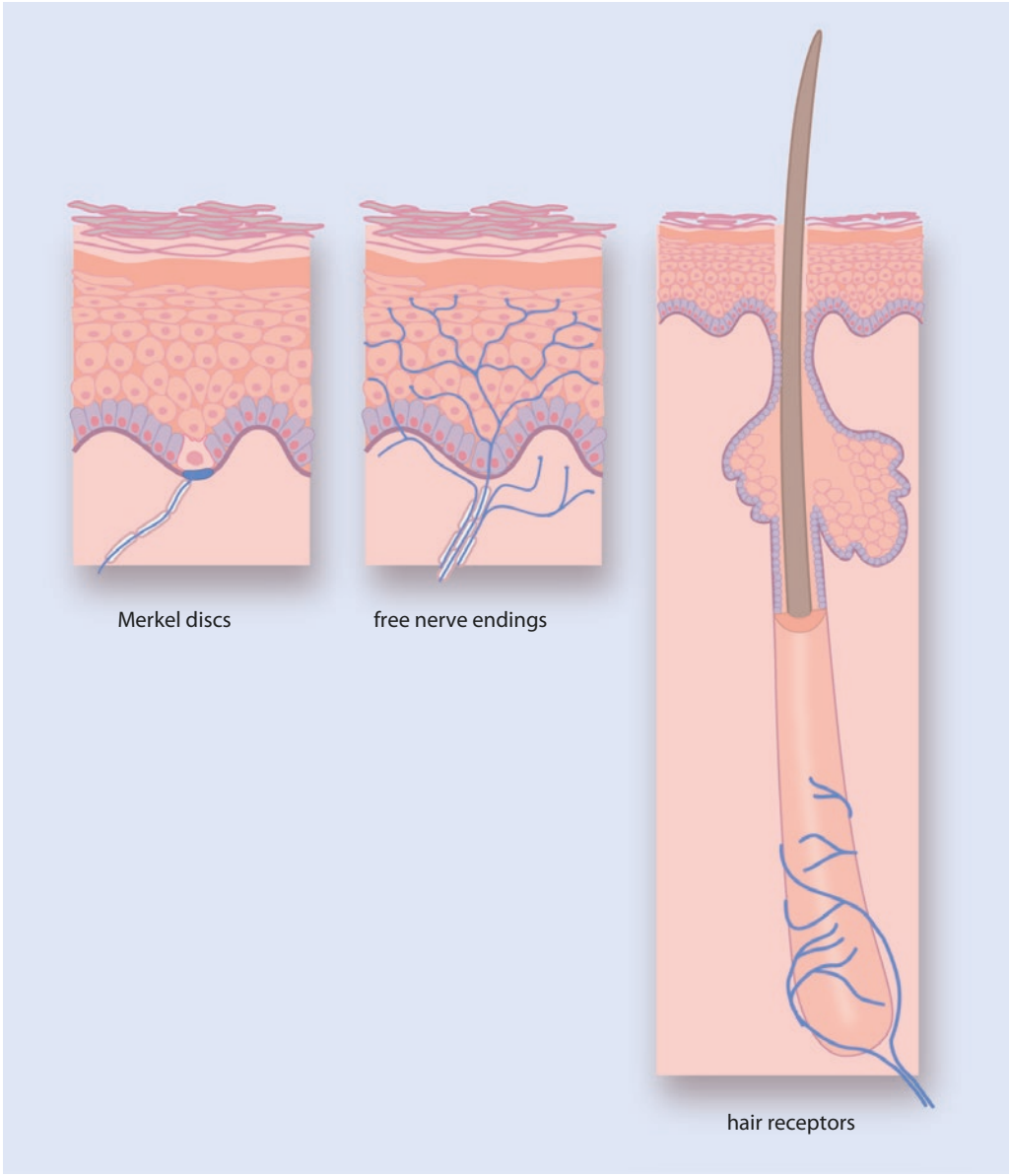


Fig. 1.1 Nociceptors

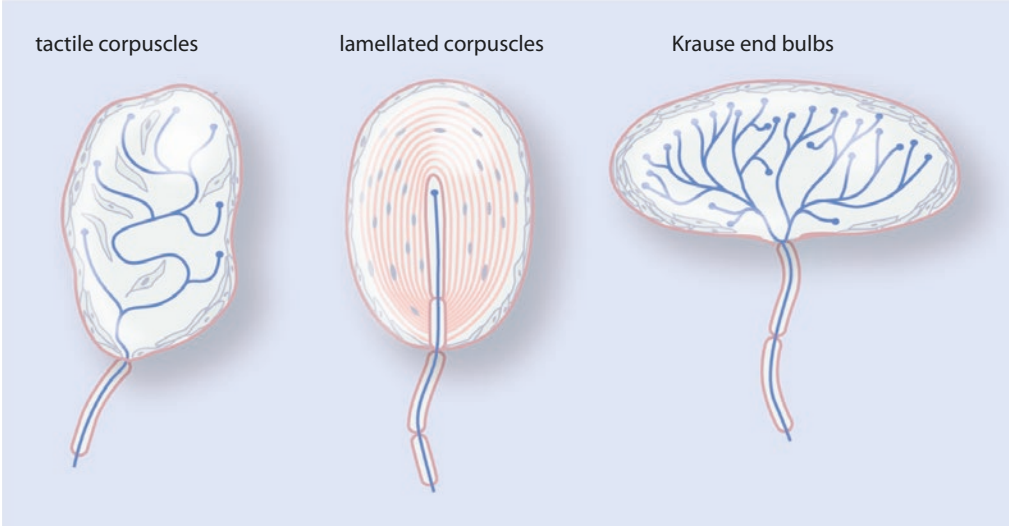


Fig 1.2 Physiological sensors

1.2 Nerve Impulse Transmission

The stimuli, received by the nociceptors and converted into nerve impulses, eventually must be interpreted in the brain. The nerve impulse is transported within the sensory nervous system, wherein three nerve fibres are successively linked. The first nerve fibres form the peripheral nerve. The second and third are present in the central nervous system and form nerve bundles (pathways or tracts). The cell nuclei of the individual neurons are grouped together in ganglia and nuclei.

1.2.1 The Structure of the Peripheral Nerve

Nociceptive stimuli are transported along sensory thinly myelinated Aδ and unmyelinated C fibres. Other types of nerve fibres are involved in the transport of other sensory stimuli (see Box 1.1).

A peripheral nerve is composed of nerve fibres from a group of neurons, enwrapped in a connective tissue network. The individual fibres may, or may be not, surrounded by an isolating myelin layer, Schwann's sheath.

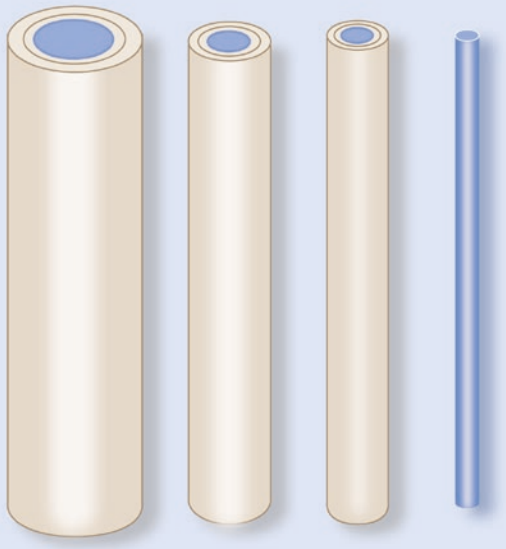
The cell body is the metabolic centre of the neuron (Fig. 1.4) where most cell organelles are produced. Dendrites transport impulses towards the cell body and axons transmit signals away from the cell body. Some axons are surrounded by a myelin sheath, while others are not. The axons and dendrites are elongated and form the nerve fibres. At the end of the dendrites, receptors are present that can receive signals. At the end of the axons are synapses, where the impulse is transmitted to another nerve cell or to a cell of the end organ.

Box 1.1 Nociceptive Pathways

There are several types of peripheral nerve fibres in the body. Nociceptive stimuli are received by nociceptors and then propagated via an A or C fibre (Fig. 1.3). The first are thinly myelinated with a fast conduction of stimuli (1.2–40 m/s), whereas the second are unmyelinated with a slow conduction (0.13–1.2 m/s). The A fibres have different subtypes: α, β, γ and δ.

The C fibres conduct impulses generated by temperature, mechanical and chemical stimulation. The Aα fibres conduct motor impulses for the body's posture and movement (proprioception); the Aβ fibres transport impulses generated by touch and signals from the skin mechanoreceptors. The Aγ fibres are involved in the regulation of the muscular tone, and the Aδ fibres conduct pain impulses and temperature signals.

The cell bodies of these primary neurons are located in the dorsal root ganglion and, for the face, in the nuclei of the trigeminal nerve. The axons run through Lissauer's tract to the dorsal horn of the spinal cord, where they connect to the secondary sensory neuron in Rexed's laminae. This secondary sensory neuron crosses the midline and ascends as the spinothalamic tract. The spinothalamic tract forms synapses with nuclei of the thalamus, where it projects onto the somatosensory cortex. Descending pathways from the somatosensory cortex modulate the nociceptive system. From these fibres, the neurotransmitters serotonin and noradrenalin are released. Also the secondary neuron of the trigeminal nerves crosses the midline and projects to the cortex through the thalamus.



axon type	Aα	Aβ	Aδ	C
diameter (µm)	13-20	6-12	1-5	0,2-1,5
speed (m/s)	18-120	35-75	5-35	0,5-2,0

Fig. 1.3 Primary afferent axons