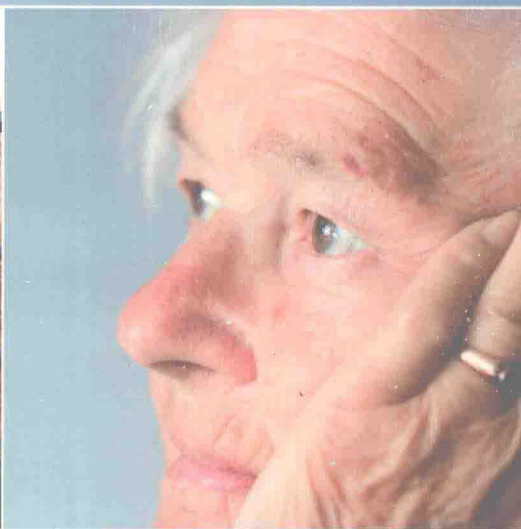


the management of
**Pain in
Older People**



Edited by Pat Schofield

The Management of Pain in Older People

Edited by

PAT SCHOFIELD, PHD RGN



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Contributors

Barry Aveyard BA (Hons) MA CertEd RMN RGN RNT

Barry is qualified in both adult and mental health nursing. His experience covers a wide range of clinical settings, but working with older people is his key area of interest. He has worked in nurse education for several years as a teacher and lecturer, and is also a member of the RCN Mental Health and Older Peoples Nursing Forum. Barry has published a number of book chapters and research papers on varying aspects of older people's mental health.

Amanda Clarke BA (Hons) MA PhD RGN

Amanda is a lecturer in the School of Nursing and Midwifery at the University of Sheffield. She completed her nurse training at Sheffield in 1986 and subsequently specialized in the care of older adults. During her nursing career, Amanda worked as a staff nurse and senior sister in stroke rehabilitation and acute medicine for older people. As a mature student, she undertook her undergraduate and post-graduate degrees in the Department of Sociological Studies at the University of Sheffield. She gained her BA in Social and Political Studies in 1994 and her MA in Applied Research and Quality Evaluation in 1996. In 2001, Amanda completed her doctorate exploring older people's accounts of their experiences and attitudes to later life, using a biographical approach. She also worked as a research associate on the European Funded project ACTION (Assisting Carers Using Telematic Interventions to Meet Older Person's Needs). Her research interests include life story work with older people, participatory ways of working with older people, active ageing, and end-of-life care in later life.

Mary Cooke BSc(Hons) MSc(Econs) PGCert PhD RGN CertMid

Mary has a clinical background in surgical nursing and midwifery, where she has held posts as clinical sister and manager and as assistant to a chief nurse. She still practices in accident and emergency care. Her academic career began in 1984 while she was a staff nurse, and progressed with a masters in planning and financing health care at the London School of Economics while working in higher education, followed by a PhD from the same institution. She has held consultant

research roles and was a research associate while completing postdoctoral studies at the University of Cambridge. Mary is now a lecturer at Sheffield Hallam University, seconded to the University of Sheffield, and specializes in health policy, health economics and service user involvement.

Rachel Drago RN MSc PGDipEd DipTher

Rachel is a lecturer in nursing at the University of the West of England, Bristol. She has staffed in hospitals in London, Leeds and Nottingham and worked as a lecturer in nursing since 1995, specializing in the teaching of physiology whilst working as a critical care practitioner. Rachel now teaches human physiology, functional anatomy and clinical pharmacology to nurses at all levels of study.

Margaret Dunham BA(Hons) MSc RGN

Margaret has over five years' experience of clinical practice in pain management and has maintained links with current pain management practice through her membership of the British Pain Society, Pain Network UK and the North Trent Pain Forum. She is on the national committee of Pain Network UK, supporting nurses in practice throughout the UK and Ireland. Since leaving the NHS in 2000 to lecture at the University of Sheffield Margaret has collaborated in the development of educational strategies to promote the needs of older people in pain. She has presented poster abstracts of her work at national and international conferences and is currently building a portfolio of publications. She currently lectures in nursing and pain management at Sheffield Hallam University.

Denis Martin BSc (Hons) MSc DPhil

Denis is a reader in rehabilitation in the Teesside Centre for Rehabilitation Sciences at Teesside University. He previously worked as a principal research fellow in the Centre for Health and Social Care Research at Sheffield Hallam University and was a director of the Scottish Network for Chronic Pain Research and award coordinator of the MSc Pain at Queen Margaret University College, Edinburgh. He graduated from the University of Ulster in 1988 with a BSc (Hons) in physiotherapy, was awarded his DPhil from the University of Ulster in 1993 and received an MSc in applied statistics from Napier University in 2000. He is a member of the International Association for the Study of Pain, the Pain Society and the Royal Statistical Society. His research interests lie in the assessment and management of the impact of pain. He has published widely in the field of pain. Denis is Chair of the Pain Association, a not-for-profit organization that provides training and support in the self-management of chronic pain.

David Reid BA(Hons) MSc

David is currently a lecturer at the School of Nursing and Midwifery, University of Sheffield. He has previously worked for the Alzheimer's Society, providing support to people with dementia and their carers and coordinating the development of a new branch of the organization in East Yorkshire. David has also previously been a research fellow, when he contributed to qualitative and quantitative studies in the areas of hospice-based adult bereavement support, end-of-life care, partnership and inclusion practices of the Alzheimer's Society in Yorkshire and adult protection. David has published a number of journal articles and has a particular interest in the ways in which the identities of people with dementia are negotiated in social interaction.

Tony Ryan BSc (Hons) MA PhD

Tony is currently a senior lecturer in the Faculty of Health and Wellbeing at Sheffield Hallam University. For almost eight years he worked with people with learning difficulties in a research and development capacity. Following a move to the University of Sheffield, he became involved in work with people with dementia. Tony was instrumental in local service developments, most significantly in the initiation and expansion of community-based provision. He worked within the Institute of General Practice and Primary Care where he completed his PhD (2003), before moving to the School of Nursing and Midwifery in 2004. In 2006 he joined Sheffield Hallam University, where he teaches on a range of programmes. His research interests centre on people with dementia and their family carers as well as stroke and pain for older people.

Pat Schofield PGDipEd DipN PhD RGN

Pat has worked in the field of pain management since 1989, first as a clinical nurse specialist and for the last ten years as a lecturer and now as a senior lecturer in the School of Nursing and Midwifery at the University of Sheffield. She was originally responsible for the development of the pain modules and is currently the unit leader to the acute, chronic and age-related units. Pat's research involves the use of Snoezelen environments for the management of chronic pain and palliative care. More recently, she has completed a study looking at resident's perceptions of pain in care homes and a systematic review of the literature in the first stage of a funded project to investigate pain in this setting within the UK. Pat has recently developed a distance learning pain education package for staff caring for older adults in care homes, being introduced around the UK in collaboration with the English Care in the Community Association.

Paula Smith BSc (Hons) MSc PhD DEN RN C.Psychol

Paula is a lecturer and programme leader for the MMedSci in Palliative Care at the School of Nursing and Midwifery, University of Sheffield. She has a background in Community Nursing and Health Psychology. In 2001 Paula completed her PhD which focused on the support needs of family caregivers in palliative care settings in the community. Since then she has worked on a number of research projects, primarily in community palliative care settings, and has interests in family caregivers, service development and evaluation. Paula is currently a steering group member of Help the Hospices Care for the Carers of the Terminally Ill Project.

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The anatomy and physiology of pain

Pat Schofield and Rachel Drago

1

The human body is able to experience a range of sensations, from the pleasant, soothing texture of velvet to the extremely unpleasant sensation of pain. For many years it has been acknowledged that the process of pain does not consist solely of a physiological set of sensations: it is a combination of physiological sensations that requires complex physiological, psychological and behavioural interactions to enable the human to interpret and subsequently respond (Wall and Melzack, 1999).

The aims of this chapter are:

- To discuss the concepts underpinning the physiology of pain.
- To explore the gate control theory of pain.
- To highlight the changes that occur within the nervous system as a result of ageing that may impact upon the pain experience as the person ages.
- To demonstrate how an understanding of these factors may influence practice.

Generally, everyone perceives the pain experience to be unpleasant and to be avoided at all costs. Only a few reported individuals are known to have never experienced pain, and this is now a recognized syndrome (hereditary sensory and autonomic neuropathy type 4). Pain is wholly subjective, and the perceived intensity and discomfort for any one known controlled stimulus varies from person to person. The actual perception of pain requires a complicated integration of sensory nerves, motor nerve pathways and chemicals that serve to enhance the

pain. All of these can be influenced by the genetic make-up of the individual, their past experiences and emotional contributors. This means that the sensation of pain is greater than the sum of its parts.

Although pain pathways, physiology and local hormone production play only a small part in the overall sensation of pain, the efficacy of analgesics and other pharmacological therapies is based on the modulation of the nervous system and its role in the sensation of pain. It is essential for any health-care professional to have good understanding of the anatomy and physiology of pain in order to make informed decisions regarding the most appropriate therapy.

Learning point

Revise some of the following terminology:

- peripheral and central nervous system
- spinal cord
- sensory cortex
- simple spinal reflex
- synapse, neurotransmitters and receptors
- sensory afferents
- motor and autonomic efferent
- autonomic nervous system.

You may wish to read the paper by Davis (1993) and the book by Melzack and Wall (1996) to support your learning.

Pain and sensation

The definition of pain as

an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage (Merskey and Bogduk 1992, p. 210)

suggests that pain may be the result of actual or potential tissue damage and that it prevents the individual from bodily harm, or from the injury, disease or harm becoming worse. It is a dramatic mixture of emotional and physiological reactions (Mountcastle, 1980; Merskey, 1986; Wall and Melzack, 1999).

There are certain things that we now know to occur within the nervous system when a disease or injury arises, but there are still some things we don't know about. Research into these mechanisms is ongoing. In this section we discuss the basic physiological concepts and then we consider the issues that are particularly relevant for the care of older people.

Imagine putting your hand on to a hot stove. This will initiate a series of responses within the nervous system that will eventually be perceived as pain. The whole process begins at the site of the injury, or 'where your hand is touching the hot stove'.

Physiological pain arises from chemical, thermal or mechanical stimulus of the small-diameter sensory afferent fibres found in the tissue. These actually detect injury and are known as **nociceptors**, which derives from the Latin word meaning injury. It is important to be aware of this as it helps us to understand the concepts of **neuropathic** and **nociceptive** pain.

Learning point

Can you identify the differences between neuropathic and nociceptive pain?

Think about the types of neuropathic pain that you see in your area.

There are two types of nociceptors: **A δ** (A delta) and **C fibres** (Cesare and McNaughton, 1997). These are different from other sensory afferent nerve fibres in that the noxious stimulus has to be of a sufficient intensity and duration to bring about tissue damage. In other words, these fibres have a high stimulation threshold. Tactile fibres such as **A β** (A beta) fibres have a low threshold and follow slightly different spinal tracts to the brain. They also transfer information related to pressure and texture, but not pain. To illustrate this, imagine what it would be like if pain was initiated by a soft touch – such inappropriate misfiring would make life impossible. Equally, if the nociceptors' threshold is set too high then tissue damage would result before avoidance action could be taken. Hence the stimulation intensity is set to prevent unnecessary tissue damage or discomfort.

Modulation and regulation of all of the incoming information is carried out by nerves that descend from the brain to the spinal cord and contribute to the analysis of the sensations at this level. These **descending tracts** are responsible

for the regulation of sensations that actually reach the brain and allow the individual to divert their attention elsewhere. This is the rudimentary basis of the **gate control theory** which we will return to later.

We can consider two categories of pain:

- **Physiological pain:** The pain response to high-intensity stimuli is transient if the tissue damage is prevented by a simple spinal flexion reflex arc (Willer, 1979). Consider striking a match and touching the flame with your fingers – you would drop the match instantly before damage could occur. The speed with which this reflex occurs prevents deep tissue damage and allows only a brief moment of discomfort. This is caused by a simple spinal reflex mediated by the high-intensity thermal stimulation of small sensory nerve endings in your fingers.
- **Pathological pain:** This results from sensitization of the nerves in the periphery and the spinal cord. Peripheral nerve endings are made more sensitive to noxious stimuli through tissue damage, action of local hormones such as prostaglandins, histamine, serotonin and bradykinin, and also by direct nerve damage – this is called **peripheral sensitization**.

When the **neurons** involved with the transmission of pain along the spinal cord to the sensory cortex in the parietal lobe of the brain are sensitized by a barrage of impulses from the site of tissue damage, this is referred to as **central sensitization**. As a result the nerve fibres of the central nervous system begin to respond to non-noxious stimuli such as gentle touch as if they were pain impulses. Peripheral and central sensitization of the neural pathway can produce pain without a clear external stimulus. So, for example, gentle stroking can become pain – this is termed **allodynia**. Furthermore, an exaggerated response to low-threshold noxious stimuli can occur (**hyperalgesia**) (Woolf, 1989, 1991; Rang, Dale and Ritter, 1999). In acute pain, this is quite an important concept as potentiation of pain will encourage rest and thus prevent further tissue damage (Woolf, 1991). However, should this continue after the acute phase (i.e. in **chronic pain**) it will serve no useful purpose and become a clinical problem in its own right. This will be discussed later, in Chapter 6.

Summary

- Pain is an unpleasant sensation which warns of impending tissue damage.
- Pain develops as a results of chemical, thermal or mechanical stimuli.
- Activation of A- δ and C fibres occurs; these are known as nociceptors and they detect injury, not pain.

- A- β fibres transmit pressure, not pain.
- The physiological response to high-intensity is transient if the tissue damage is prevented by a simple spinal flexion reflex arc.
- Sensitization of nerves in the periphery and spinal cord is known as pathological pain.
- Tissue damage or local hormone action can make peripheral nerve endings more sensitive this is known as peripheral sensitization.
- When the central nervous system responds to A β fibres as if they were conducting pain impulses, central sensitization occurs.

Neural pain pathways

When the sensory neurons synapse with the motor neurons and transmission neurons in the dorsal horn of the spinal cord, pain is detected. As seen in Figure 1.1, the nerve fibres within the dorsal horn (rear) carry information back to the spinal cord and brain. The ventral horn (front) carries autonomic efferents and motor nerves away from the spinal cord and brain back to the body.

The terminal nerve endings of the sensory nociceptors release the neurotransmitters **substance P** and **glutamate**. These chemicals in turn bind to the surface of the dendrites of the transmission neurons, propagating the signal forward either to a motor nerve or up to the brain via the spinal cord.

C fibres

These are fine **unmyelinated fibres**, 0.23–1.5 μm in diameter, which respond to chemical, thermal or mechanical stimuli. Because they have more than one mode

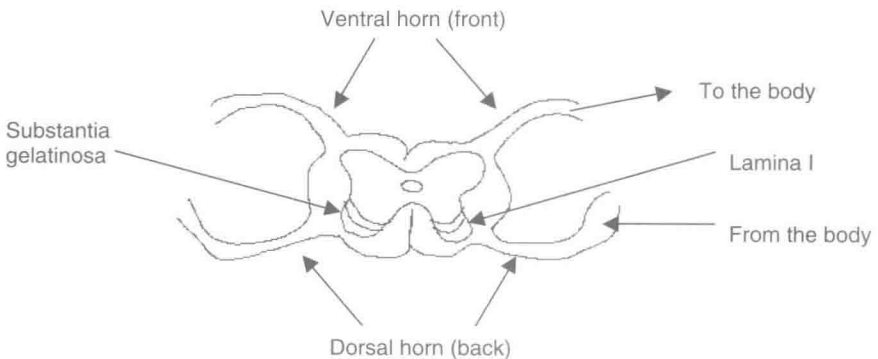


Figure 1.1 Cross-section of the spinal cord

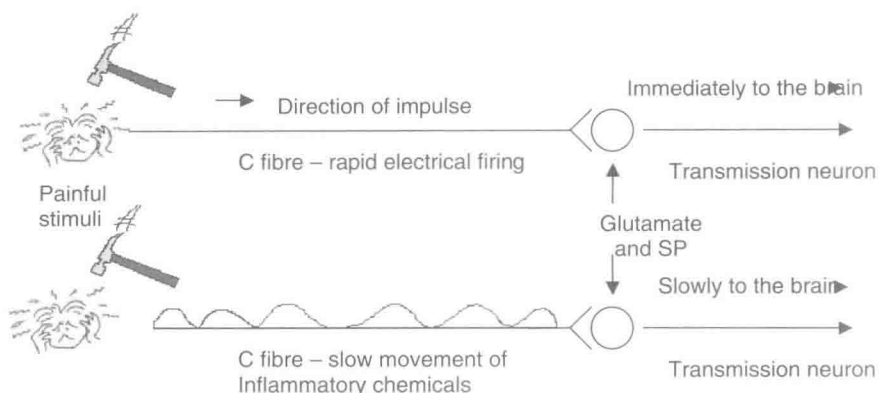


Figure 1.2 The rapid and slow effects of the C fibre in the dorsal horn

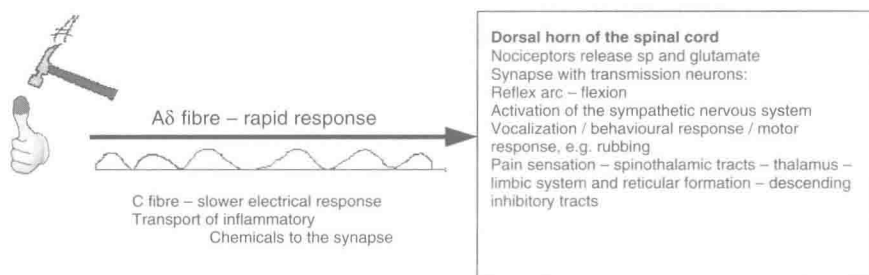


Figure 1.3 Physiological pain responses

of stimulation they are also known as **polymodal fibres**. It is believed that C-fibre activity is associated with dull, diffuse pain and once initiated can continue for up to 80 hours. The conduction velocity (speed with which the pain message travels) is <2.5 m/s (Figure 1.2).

Along with sending electrical messages to the spinal cord by the movement of potassium and sodium ions into and out of the axon, C fibres are also responsible for the absorption of inflammatory chemicals such as **bradykinin** along the length of the axon to be released within the spinal cord at the synapse with the transmission neuron (Wall and Melzack, 1999). This process provides a dull, diffuse and profound ache that often follows relatively minor injuries such as a sprained ankle, resulting in the whole leg aching for days after the injury.

Aδ fibres

These are medium-sized (1–5 μ m diameter), myelinated, fast-acting neurons with a rapid conduction velocity (>2 m/s) (Figure 1.3). It is believed that these neurons