PAIN

How and Why We Hurt

Part Two: Acute Pain

by Gary Bruce
photographs by Jonah Sutherland

Pain can suddenly flood into our body and capture our attention to the exclusion of everything else—and that’s a good thing. When we lean against a hot stove or brush against a spiny cactus, we need to move out of harm’s way, quickly. If we sprain our ankle, throbbing pain will force us to rest the injury, allow the joint to heal and prevent further damage.

Acute pain is a protective mechanism, for which we should be thankful. Without sensations of acute pain, we would persist in harmful activities until we damaged ourselves beyond repair. How else would we know to stop, if not for the pain? Pain is not just intense sensation. It is an elaborate system of warnings and responses that has evolved in our bodies over millions of years. Though pain is not much appreciated when we are held in its grip, without acute pain we would not long survive.

Acute pain and chronic pain are differentiated by their time frame. In general terms, acute pain is less than six weeks in duration. If acute pain persists beyond six weeks (or beyond the phase of tissue healing), then it becomes chronic pain.
Sensing pain

Nerve endings that respond to tissue damage and generate pain messages are not the same nerve endings that respond to gentle touch. Pain receptors are specialized nerve endings that respond only to noxious or physically harmful stimulations. They are called nociceptors. Extremely fine in diameter, they are designed to react to intense and potentially damaging stimuli. Sensations generated in the nociceptors are sent to the spinal cord by way of rapidly functioning, specialized nerve fibers. The type of nociceptor and its related nerve fiber will determine if we sense a sudden, sharp pain or a dull, throbbing ache, which develops more slowly.

Some nociceptors respond to a variety of types of potentially painful stimuli, such as pressure, heat and cold. Others are singular and selective, reacting only to a specific type of stimulation (a needle prick, for example).

In some areas of the body (such as the lips and the finger tips) there are as many as 1,300 nociceptors in every square inch of skin. The variety and vast quantity of nociceptors distributed throughout our body assure us that: 1) the pain message will get through; 2) we will know what kind of pain we are feeling; and 3) we know at what area the injury occurred. These are critical messages for survival, because they can provide information about dangers in the external environment and how to avoid them.

When we injure our body and sense pain as a result, several events are taking place at the cellular level. At the site of the injury, damaged cells begin to release specialized chemicals. Ruptured blood vessels disperse bradykinin, a plasma polypeptide that causes nerve fibers to send electrical impulses to the spinal cord. Substance P, an 11-amino acid peptide, sustains and prolongs the signals to the spinal cord and keeps the tissues alert for more incoming pain messages. Prostaglandins, biologically active unsaturated fatty acids, call for infection-fighting cells to arrive at the injury site to fight invading bacteria. Prostaglandins further expedite the flow of pain messages in and around the injury site to make sure the message gets to the spinal cord.

These three chemicals, bradykinin, substance P and prostaglandins, create redness, swelling and heat at the injured area. The heat and fluid that flows into the area in turn increase the pain messages being generated at the injury site. Measures taken to reduce swelling and heat help reduce the quantity and intensity of the pain messages.

Application of ice or cold packs reduces the tissue damage that results from accumulation of excess fluid in the injured area. Cold can also block the pain by acting as a counter-irritant. (Reduction of edema and pain impulses can speed the healing process.)

Messages from the specialized nerve endings at the injury site flood into the spinal cord through the dorsal nerve roots. Here the messages are sorted and routed. (At this level, we encounter the gate mechanism, which was introduced in “Pain, How and Why We Hurt, Part One: History & Definitions,” September/October.) The gate mechanism determines if the messages are urgent enough to be sent upstream to the brain for further processing.

Those messages that pass through the gate are directed toward the reticular formation sitting at the top of the spinal cord within the thalamus. Other messages are redirected to the injury site to stop the incoming flow of pain messages.

At this stage, the stimuli traveling through the nervous system are not pain signals. While these messages are generated by injury and are related to the process of pain, they are not sensations or signals of pain. They are impulses of information. Pain is only in the brain.

In the brain

Arriving at the reticular formation, pain messages enter a web of nerves that are in contact with multiple areas of the brain. The thalamus acts as the brain’s mailroom, receiving and sorting messages, forwarding some messages upstream to higher brain structures, while returning other messages back to the sender.

Once the brain gets the pain message, more pain messages are not needed, so some messages are routed back to...
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the injury site. Returning pain messages can serve to
decrease the number of pain messages sent from the injury
site. Messages are sent upstream from the thalamus to the
cerebral cortex. The cerebral cortex maps the incoming
information to discover where the damage is located, the
cause of it and how to stop it. (We could say that the mail
from Pain Central is opened, read and answered, at
this point.)
This connection from the spinal cord to the thalamus
to the cortex is crucial for the perception of pain. It is at

**Anatomy and Pain**

Pain is so important to our survival that almost every
structure of the brain is involved in sensing and
processing pain impulses. There is no one area of the
brain that can be properly called "the pain center." Pain
demands that the brain simultaneously process
emotions, sensory information, behaviors and
even memories.

**Brain imaging** performed on volunteers during painful
stimulation indicates activity in diverse cortical areas.
These include the insula and the sensory motor,
parietal, frontal, cingulated and occipital cortices.

Pain also involves subcortical structures, including
the thalamus, putamen, caudate nucleus, hypothalamus,
amygdale, periaqueductal gray matter and
hippocampus.

Because pain triggers activities in all of these areas,
it directly affects our emotions, perceptions, actions
and memories. And they, in turn, affect our response to
pain. Recent research conducted at the University of
Michigan indicates that gender, hormone levels
and genetics can also affect the degree to which we
feel pain.

In a very real sense, our entire anatomy is involved
in sensing and processing pain. All of these factors
taken together may explain why sensing pain is such a
highly individualized and subjective process.

Footnotes

1. Wall, P.D. "The mechanisms by which tissue damage
and pain are relate," *Pain 1996—An Updated Review*

2. Zubieta, Jon-Kar, et. al., *Sex, Hormones & Genetics
Affect Brain's Pain Control System*, University of
Michigan Mental Health Research Unit (2003), online at

---Gary Bruce

the level of the cortex that we consciously perceive pain. If
the cerebral cortex doesn’t get the message, then we don’t
hurt. The relationship between consciousness, thought,
emotion and pain leaves open the continuing debate as to
what is consciousness. Until we have a universally accept-
ed definition of consciousness, we will not have a uni-
versally accepted definition of pain.

In the meantime, we can agree that the brain plays the
primary, essential and central roles of processing the feel-
ing of pain in the body. This fact led author, educator and
pain researcher Dennis Turk, Ph.D., to state, "The reign
of pain is mainly in the brain."

While the upstream connection of the sensory stimuli
to the cortex is essential to the perception of pain, the
cortex is only one of several brain structures involved in the
interpretation and perception of these sensory stimuli as
the phenomenon of pain. Creating painful stimuli in test
subjects, while at the same time performing brain
imaging, researchers have determined which brain struc-
tures are activated when pain is processed in the body.
Together, they have been referred to as the central pain
matrix.

Sight, sound and smell are also relied on to gather more
information about the injury and its cause. Stimulation of
the limbic system causes the cerebral cortex to pay
increased attention to the injury. In turn, like forwarding
an important e-mail to associates, nerve fibers from the
limbic system reach to several other structures in the brain
that trigger a variety of reactions, including loss of
appetite, depression, anxiety, fear and anger.

It is at the level of the cerebral cortex that we learn to
try to avoid future encounters with painful stimuli. Also,
at this level, our thoughts and attitudes affect the degree
of pain that we are sensing. The work of Henry K.
Beecher, M.D., revealed how the power of belief could
decrease the pain of horrendous battlefield injuries. Secure
in the knowledge that they would be sent to the safety of
the hospital, soldiers with gaping wounds seemed to sense
little pain. Placebos have become a standard control in
pain research. A placebo pill, unknown to the test subject,
contains no active ingredients. Instead of testing medica-
tion, the placebo tests the power of belief within the sub-
ject's mind to modulate pain generated within their body.
But how can a sugar pill reduce pain?

**Natural painkillers**

The gate theory led researchers to question the actual
mechanism that the body uses to shut off pain sensations.
Research led to the discovery that the body produces its
own natural painkillers, a group of proteins called
endorphin, named after the drug morphine, which for thou-
sands of years has been used as a painkiller.

Morphine (from Morpheus, the Greek god of dreams)
is extracted from the opium poppy, and has the ability to

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reduce or eliminate pain. Using the same mechanism by which morphine reduces pain, researchers found that endorphin (morphine-like substances from the body) attaches to receptors in the brain and spinal cord, blocking pain information and creating the same effect. Endorphin can be produced in the body by movement and stimulation of the soft tissue.

Exercise creates the so-called runner’s high, by triggering endorphin production. Production can also happen through vigorous massage and manual therapy. Candace Pert, Ph.D.’s discovery of the natural production of endorphin through stimulation of soft tissue provides a scientific basis for the value of therapeutic massage in the management of pain.

Managing the pain

So, how do we help manage the pain that a client is experiencing? First, we need to gather information about the pain. Some of the questions we might ask include: When was the onset of pain? Is there a known cause? Is the pain the result of a specific injury or activity? Is the pain constant? and, does movement or activity increase the pain? We need to determine if the pain is acute or chronic in nature. Acute pain has a rapid onset, associated with traumatic injury or repetitive strain. Chronic pain is persistent by nature and may not have a known cause.

Since acute pain can be caused by traumatic injury, athletic exertion, overuse or repetitive strain, the importance of client history-taking must be emphasized. A definite

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The Brain and Pain

One of the roles of the thalamus is to be the reception area of the brain for signals arriving from the spinal cord. The thalamus connects with the cerebral cortex, which is the seat of consciousness, thought and emotion. This connection from spinal cord to thalamus to cortex is crucial for the perception of pain. Without perception, pain does not exist. While the connection of the sensory signals to the cortex is essential to the perception of pain, the cortex is only one of many brain structures involved in the process of feeling pain. In terms of the brain and pain, we must also include:

Anterior cingulate cortex: processes pain signals and attention processing; makes us aware of the pain.

Thalamus: receptor for stimuli, processes incoming information in a miniature pattern of the human form or homunculus (Latin for “small man”).

Primary and secondary somatosensory cortex: Disputed as to their role in pain processing, these structures discriminate the physical or spatial aspects of nociception.

Brain defense system: a functional relationship between the hypothalamus and the amygdale in response to stimuli that creates the emotional and autonomic dimensions of pain. Stimulating the endocrine system, it causes changes in mood.

Anterior insula: deals with visceral functions, crude emotions and instinctive reactions to pain.

Prefrontal cortex: Supervising behavior and attention, it is closely linked to our conscious perception of pain.

Posterior parietal cortex: helps to create a physical sense of self and reveal the location of the source of pain.

These anatomical structures show, through their functional relationship, how much of the brain is involved in the perception of pain. Because these structures are also associated with other brain activities, like consciousness, mood and emotions, defining pain is part of the larger problem of understanding the difference between the organic brain and the functioning mind.

The relationship between consciousness, thought and emotion makes for a contentious debate. Can or does the mind exist as a separate entity from the brain? Can we divide the spark of consciousness from the physical matter of the body? We cannot ignore the role that pain plays in convincing us that we exist.

Being conscious is a prerequisite to experiencing pain. Descartes, the 17th-century French mathematician and scientist, considered the father of modern philosophy, and a proponent of mind/body dualism, stated, "I think, therefore, I am." It would be no less accurate to say, "I hurt, therefore, I am." However, until we have a universally accepted idea of consciousness, we will not have a universally accepted idea of pain.

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With practice, the therapist can learn to help the central nervous system modulate acute pain, stop the injury process and trigger the body's innate healing response.
cause cannot be determined in all cases. Sometimes there is a rapid onset of pain without a reason that can be recalled by the client. If a physician refers the client, we have the benefit of a medical screening. Because not all clients in pain arrive at our door by way of their doctor’s office, it is important to decide if medical attention is warranted.

If in doubt as to the nature or extent of an injury, err on the side of caution and refer the client to their physician or emergency medical personnel. Massaging bruised areas is contraindicated. Manual pressure can increase bruising and pain. It is also very possible that a blood clot could be released into the circulatory system, then lodge in the lungs or the brain and result in serious injury or death.

In a less-serious case, it may be decided that addressing the acute pain is within the skill and scope of practice of the therapist. When the pain can be attributed to a recent trauma, it is essential to avoid more tissue damage at the injury site. Therapy should focus on stabilizing and cooling down the injury; stopping the ongoing tissue damage caused by excess fluids in the area; and the prevention of additional painful stimuli.

Especially when extremities are involved, the basic rule for acute injury is rest, ice, compression and elevation. This provides a good rule for placement of the client on the therapy table. Securely support the affected limb and apply cold packs to reduce inflammation. Remove the cold packs to work on the area, and then reapply them. Limit the application of cold to a maximum of 20 minutes at a time. A cooling balm may be applied to the area at the end of the session, as appropriate.

The function of pain, at this stage, is to avoid further damage and to force the person to rest the injured body part, allowing the tissue to heal. Our therapy should follow suit. Manual techniques may best be limited to stimulating the peripheral nerves through rubbing and stroking near the injury site. Don’t use brisk friction techniques near the site. We should be working to reduce heat in the tissue, not increase it. Physical movements of the affected area should also be limited. Palpation is performed to reveal the presence or absence of muscle spasm, muscle knotting or tender points. The degree and intensity of our palpation and therapy techniques are limited by the client’s reaction to pressure in the painful area.

Based on our assessment and the patient’s response, subsequent therapy sessions can be more vigorous and work toward mobilization of the affected region. With practice, the therapist can learn to help the central nervous system modulate acute pain, stop the injury process and trigger the body’s innate healing response. Acute-pain management is a great skill to cultivate. Yet, at times, regardless of our best efforts, the client will continue to be in pain for weeks, months and even years after the initial injury has apparently healed.

Part One of this series explored the definition of pain, and historical philosophies of pain. Part Three will explore the complex and sometimes puzzling challenges presented by chronic pain, and its often-debilitating effects.


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