CHRONIC PAIN, A PAINFUL PROBLEM ?

The presence of chronic pain with no apparent aetiology has been a major problem for more than a century. The complex range of causes has still not settled into the scientific mind in 2016. That is not so much because there is no evidence for it, buy more because the evidence is older than five years, and is therefore no longer taken seriously by scientists: old means useless. This booklet, the summary of which is published as a blog, takes the reader through time to reveal the efforts that have already been taken in making chronic pain measurable and visible, which is a very good reason to go back in time. That too is possible. These days chronic pain needlessly costs billions more than it should, not to mention the misery and sadness patients and their social surrounds have to go through to the extent that they do. However, no one can seriously promise complete relief from pain.

A publication by the "Chronic Pain Science Foundation"

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Written for the Dutch-speaking region.

A shortened version has been translated into English.

Contents

Preface

This article is not written solely for the professional, but rather for the reasonably educated layperson.

Chronic pain, especially when there is no demonstrable aetiology, has been a big problem all over the world for more than a century. It is not a sexy (life-threatening) condition which is why it is not investigated widely. Modern science does not remember or acknowledge the scarce amount of scientific publications that already exist on this topic with the argument that the bar is much higher these days. This attitude excludes rational scientifically grounded treatments which lead to a number of attempts to address the problem. The expression that "it doesn't hurt to try" does not seem to apply here, even though many treatments – or overtreatments – are implemented without success.

In effect, because modern medical science lacks the knowledge, technical skills and facilities to make this form of pain visible, the notion that chronic pain is psychosomatic has taken root. The cause might well be the consequence of a physical condition, but the alarm bell still falls on deaf ears. So there we are, the psychologically indicated treatment choice is the official scientific thought process originating from "we didn't find anything, therefore...".

One should expect scientists to do their best to find the source of a pain signal. Only then can you investigate if this source is justifiable, and only then can you say whether a psychological factor is at play. It is not by chance that this hardly happens or does not happen at all.

This article is an attempt to find an answer that is impossible to fit onto one sheet of paper.

Summary

This article addresses the history of the development of treating chronic pain that lacks a substrate (demonstrable aetiology). This history starts in 1898 and continues with the developments that motivated the author to occupy himself with the phenomenon of chronic pain.

This involves a search through the literature that was published mainly around and before the 1960s. It also involves delving into the examination and treatment methods that were applicable in that period. Milestones in the literature are the publications by Baumann and Ueckert in 1954 which report on skin temperature discrepancies of pain sites. A second milestone is the publication of the textbook by Hansen and Schliack in 1962, which is a very extensive work addressing early 20th century history of neuroanatomy and neurophysiology and their effects on symptom profiles: The groundwork for future research is included in this book. The third milestone is when the infrared video thermography equipment hit the market, which made the skin sites Baumann and Ueckert reported on and the work described by Hansen and Schliack visible and measureable.

It was just as well. Chronic pain without a known substrate has proven to be not only a disease with major psychosocial consequences, but it also has enormous economic consequences. Finally a research and measurement instrument existed that could investigate the phenomenon, except the interests of medical science and politics prevented further pursuit. That turned out to be the case shortly after the Hansen and Schliack textbook was published in 1962, so the publisher removed it from the market due to lack of interest at universities. An aversion by universities to collaborate in research meant that only one university the author had approached was interested; as a matter of fact so interested that there was even an attempt to steal the idea. Yes even back then. Chronic pain was described as originating from a segment, which is the area through which one spinal cord nerve runs. A good accessible area for investigation was the skin, since it is a part of such a segment.

The skin has indeed been reported on regarding this issue several times, but the problem of a viscerocutaneous element (the immediate connection between internal organs with other segment parts and the skin, currently referred to as viscerosomatic) in

humans has never been established experimentally. So what from the historical reports on viscerocutaneous pathways was true? This should be tested first before there can be any more talk on further research, which was conducted from 1981 to 1983. Journals for those studies refused to publish them for reasons that had to do with the special interests of the journals and science. Those interests are tightly interwoven. In the meantime the research proceeded. A treatment method was sought after under the assumption that recent outcomes that were found actually correspond with the historical reports. There already was one on accepted medication that could meet the specifications on the market in the Netherlands. The manufacturer was not able to or did not want to participate in a study because of commercial considerations.

Despite that, a study was still designed in accordance with the applicable rules of the Helsinki Convention at that time. The medicine was prescribed as off-label, which means it was used for a non-registered indication. The sense of pain was recorded and the pain sites with their reference sites were measured with infrared thermography. The images were stored on a hard disk.

The outcome was that an improvement was verified with infrared thermography in 70% of the cases. A statistically observable correspondence between pain and change in the thermographic image was shown. The outcomes were not reported then and no journals wanted to publish them, that is until 2011. They were accepted for peer review on the condition that the investigations would be verified for relevance and current importance in 2012. The studies that were carried out were successively accepted and published in 2013 and 2014. A hypothesis on the development of chronic pain based on the results of these studies was published in two parts. The first part covered a practical result by applying the hypothesis to the measurement and regulation system during the menstrual cycle, and then the hypothesis was elaborated on for chronic pain and CRPS, the most extreme form of chronic pain. A study was also published by a Danish research group that also showed the possibility of pain developing in the skin via a viscerocutaneous reflex (2008). The interest groups then remained very silent, that is until an announcement was made about the National Pain Week conference that took place on 28 and 29 September 2016 in the Eye Amsterdam venue.

According to the programme of that conference all sorts of new ideas would be published that were already public property in 1986, while the announcement stated that the problem was tremendous and that the general practitioner was still puzzled about how to treat pain. Why is this question still being asked when the solutions are already there?

This question is what has triggered this article. For thirty years this phenomenon has not been as allusive as it is being presented. It is even easy to verify, and no one knows or claims to know how.

How it began

In 1982 the new Junior Chamber International Den Helder was looking for an installation project. In that context for the first time research was conducted on the extent and impact of the surmised problem area: chronic pain. The statistics, supported by a video presentation, demonstrated the needs that resulted in the establishment of the *Pijnstichting Nederland* [a Dutch pain foundation].

Research has shown "that chronic pain has far-reaching consequences for the patient, his or her immediate social surroundings, and society as a whole". Chronic pain influences the patient's life and his or her social surroundings such to the degree that life might revolve around the phenomenon of pain. Life ultimately deteriorates into pain. Pleasant decent people become depressed, cannot see their way out of a situation and experience a change of character. Their social surroundings have the greatest difficulty with adjusting appropriately to this, all the more because the emotional situation can suddenly change when the pain, perhaps by chance, disappears again.

Approximately 17% of the population in the Netherlands is a chronic pain patient. This was true in 1982 and it is still the case in 2016. The cost before 1977 was estimated to be 10% of the gross national product.

The situation has essentially not improved in 2016. The figures were confirmed in a recommendation report to the Dutch government in 1986. In 2000 a governmental agency reported on the following based on a university study: *"Doctors and other health care professionals have too little tangible support to make a well-thought-out choice for a certain treatment because there is no scientific foundations for such a decision"*.

It is no surprise that too much overtreatment takes place since the nature of the disease "Chronic pain" is not recognized and treatment is ineffective. Patients continue searching for a solution and there are many health care professionals who promote their method as offering a "pain-free life". That method is not scientifically substantiated, and is not possible if one does not understand the fundament of the pain. The diversity of publications worldwide seem to indicate that this is not specific Dutch problem specifically. The symptoms are similar everywhere in the moderate climate worldwide.

What is chronic pain?

The International Association for the Study of Pain (IASP) uses the following definition of pain: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. The duration is longer than six months. In fact pain is considered to be chronic after it lasts for more than 4 months.

It is indeed an experience, as a child once said: "Pain is, if it hurts". The IASP keeps the possibility open that there may be actual tissue damage instead of using the traditional definition that the pain no longer has anything to do with actual tissue damage, but that it 'leads its own life" (1986 report from The Health Council of the Netherlands). That report is still valid today.

Humanity has been involved with pain for a very long time. Pain is an alarm signal which does not seem to have a memory. The severity of pain can be fairly reliably reflected on a VAS scale, which is rating of pain on a scale of 1 to 10. If asked a day later what the pain was from the previous day, then it is somewhat of a guess. If the asked after just being anaesthetised, then its value before the anaesthesia can no longer be reliably given. (Pilot studies by the author). This also applies to chronic pain sites. This all points to an actual signal by an actual process and probably to actual tissue damage.

Another phenomenon in medicine called "Neural therapy" points in that direction too. If one injects a non-chronic pain site with a local anaesthesia, then the pain returns when the effects of the injection wear off. Frequently the opposite holds true for a

chronic pain site, where the pain subsides much longer, even for many hours after the effects of the anaesthesia wears off. If the anaesthesia is repeated, then the duration of being pain-free increases. It seems to lie on an exponential scale (2, 4, 8, 16 etc.). That is a bit odd for what is being called a mental condition.

Chronic pain sites

Diverse publications on pain sites – mainly German and Danish – in the skin have appeared since 1886, when Head presented his research on impaired skin sensitivity by internal organ diseases. This was followed by Wernøe from Denmark, who established reflectory connections between internal organs and skin in worms.

Baumann published a study in 1954 on measuring temperature differences in the segmental dermatomes of the duodenum and the liver using galvanic skin response equipment. That same year, Ueckert published a study on the Head's areas and skin temperature. Pain sites on the skin are colder than the surrounding areas in 75% of the cases, and are warmer than the surrounding areas in 25% of the cases. This evidence cannot be found in modern literature (after 1980); that is until 2008.

Infrared image of the abdomen from 1980. The square is the navel. The arrow points to Mc Burney's point.

It was already known in 1980 that internal organ diseases are accompanied by an achy nauseating pain along with a sharp identifiable pain. Mc Burney found that pain could be located in a very specific area in the abdomen caused by something such as appendicitis as an example. Doctors want to palpate that to diagnose an infection. That site lies right near the site Head indicated as the skin area that corresponds with the appendix. If the skin site indicated by Head that corresponds with the appendix is anaesthetised during appendicitis, then the sharp pain disappears immediately but the nauseating achy pain remains. Once again we have the skin being affected. It seems like it would serve as an excellent diagnostic tool, but alas, that was the last we heard of the skin regarding this issue. No one is doing anything with this information. There are no technical possibilities to make the viscerocutaneous "connection" visible. The pain site is right in front of our eyes and within our reach, but apparently it continues to be ignored.

The skin as segmental pain site

In an article by a Danish research group (Arendt-Nielsen et al. 2008) on the spreading of chronic pain caused by irritation of a stoma wound (a stoma is a surgical opening through the skin leading to the intestines), they report a specific corresponding skin area which was painful after the irritation developed, which supports the studies by Head and Wernøe involving human beings. It also supports the idea that internal organ disease can cause pain in the skin.

The fertilised egg divides itself countless times when developing into a human embryo. The cells that arise can develop into all sorts of cells. At a given moment some cells split off to form the brain, the spinal cord and nerves. Other cells develop to become connective tissue and skin cells, while others become muscle cells or bone. A third group of cells specialise to become internal organs. All of these developing groups receive a nerve connection with the later developing central nervous system: the brain and spinal cord.

The spinal cord sections itself off appearing like a worm with a large number of similar slices called segments. Tissue that also segments itself surrounds it and transforms into vertebrae. Each vertebrae has connection with an accompanying segment. Other tissues also have contact with their own segments via nerve cells. The body seems to be constructed out of one central computer (the brain) and many terminals (the segments). Each terminal has its own peripheral device (skin, muscles, connective tissue, bone, blood vessels and organs), that can receive signals from the main computer, but a significant part of the signals are controlled by each terminal. These terminals can receive signals from the peripheral devices that then function as sensors. These signals can also be transmitted to the central computer. Each of the components function as an interactive screen. This is how problems with joints, for example, are observed in the connective tissue of the same segment, in blood circulation and in the temperature of the segmental skin. All of these tissues have a complicated and corresponding "wiring".

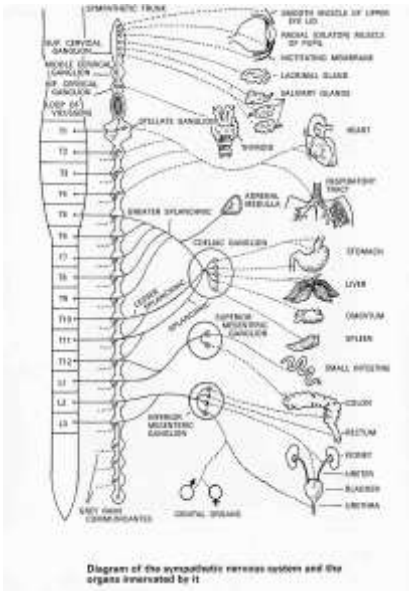
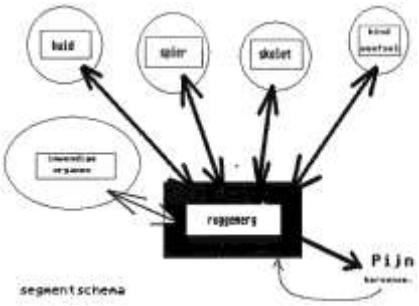


Diagram of the sympathetic nervous system and the organs innervated by it



Legend for the segment diagram:
Huid = Skin
Spier = Muscle
Skelet = Skeleton
Bind weefsel = Connective tissue
Inwendige organen = Internal organs
Ruggemerg = Spinal cord
Pijn = Pain
Hersenen = Brain
Segment schema = Segment diagram

An inflamed appendix can transmit pain to its corresponding segmental part of the skin via this wiring. Irritation of a stoma can also cause a reaction in a remotely lying skin area that is connected via neurons that have the same "terminal" as the stoma site.

That principle has already been described by Head in 1898 and experimentally tested by Wernøe in 1925. It is possible that the pain sites reported by Baumann and Ueckert in 1954 are also such sites, but this correspondence was not demonstrated in humans until 1980. In order to be able to acquire evidence that pain on the skin can actually correspond to internal diseases too, an experimental link between stimulation of an internal organ and skin temperature needs to be demonstrated first.

Experimental evidence

It has been possible to test the findings of Wernøe, Baumann and Ueckert on people since 1980 when non-contactual infrared thermography came on the market. The author of this article used Philips thermography to carry out four studies from 1980 to 1989.

The first study was with women who were having an intra uterine device (IUD) placed or replaced. It was known then that a side effect of an IUD – albeit not frequently – was inflammation in the cervical membrane. It was expected

that the IUD could cause a mild inflammatory reaction in the membrane. Depending on the device's settings, infrared thermography measures the heat radiation from objects such as houses, motors or human skin. Of course the medical device was set to measure human skin in this study.

If a direct connection (reflex) existed between the cervix and de skin, the reaction on the skin when the IUD was placed would be an evident temperature increase on the skin that would be observable with infrared thermography, which would establish a viscerocutaneous reflex in humans too. When a significant result was observed ($p<0.001$), it was necessary to test the sensitivity of the device.

The second study was also conducted with a group of pregnant women using thermography. An abrupt temperature reduction was found at a predefined specific skin area and nowhere else during the 34th week of pregnancy.

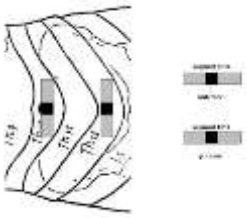


Figure: from left to right: 4 images of a pregnant woman in 1981. The woman lying supine with the head facing left. The black square is the navel, which is what is focused on sharply. The white rectangles are measurement areas, from which the mean temperature is calculated. The images were taken on diapositive film.

The results showed that during pregnancy, a significant temperature difference developed between the genital zone and the navel zone ($t=-6.019$), caused by lower temperature in the genital zone that became visible approximately in the 34th week of the pregnancy.

The findings of Baumann and Ueckert were also tested in patients with abdominal complaints without and traceable abnormalities. 70% of the designated pain sites were cooler than the surround area and 30% were warmer. Patients who had lateral pain on their arms and legs turned out to have the same results.

A study was conducted with Pentoxifylline (PTX), which was already on the market in the Netherlands for a few years to improve circulation, with the hypothesis that improved circulation could reduce pain at cold sites. A pain rating of "the same, less or more" was kept. The pain sites became significantly warmer than the surrounding tissue after treatment. A significant relationship was found between sensed pain and skin temperature change. This is what is called a "typical result" or "The best from the test."

These are two infrared images of the same patient lying prone with the head to the left. The black band is her clothing and her legs are to the right. Black is cold and white is warm in the image. Both legs became practically symmetrical after treatment. The patient was free of symptoms at the time.

What was remarkable, and at the time not understood, was the fact that the warm pain sites decreased in temperature. Comparing from left to right we see the asymmetry on the left became symmetrical on the right. These studies were rejected by journals at that time. It was not until 2012 when the first article was accepted after verifying for current

importance and relevance. The second study soon followed and was published in 2013 with the other two studies published in 2014.

In 2008 a study on viscerocutaneous reflex pathways in humans and pain generation in the corresponding pain site conducted by the group of Arendt-Nielsen was also published. This study tested the old studies from 1925 and 1954 using modern methods and they were able to support the findings from the old studies. The probability that these results were founded on chance were one in a million.

The conclusions of these studies were: viscerocutaneous reflexes exist in humans. Processes in internal organs are reflected in temperature changes in specific skin areas and can be measured with infrared equipment. This equipment can detect changes of an organ at the physiological level. Pain can develop in the skin when an internal organ is painfully stimulated.

Literature:

Arendt-Nielsen L, Schipper KP, Dimcevski G, Sumikura H, Krarup AL, Jamberardino MA et al. Viscero-somatic reflexes in referred pain areas evoked by capsaicin stimulation of the human gut. Eur J Pain. 2008;12(5):544-551

Veen PHE van der, Viscero-cutaneous reflexes in relation to abdominal and pelvic pain. A study from 1982 in females with IUD insertions. Thermology international 23, 3(2013) 157-165)

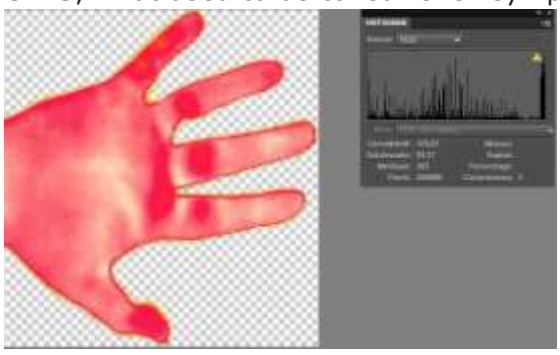
Veen PHE van der, Martens EP. Viscerocutaneous reflexes with abdominal wall pain: A study conducted in 1981 on pregnant women from a general practice. Thermology international 23/2 (2013)

Veen P. Henk. E. van der. Abdominal Wall Pain: Effects of Placebo Measured by Infrared Thermography. Thermology international 2014, 24(4) 157-165)

Veen P. Henk. E. van der. Infrared thermography for pain influenced by a Xanthine derivative: An attempt to assess chronic pain objectively. Thermology International 2014, 24(2) 39-48.)

Skin temperature with chronic pain

Chronic pain sites in the skin are predominantly cold, and are warm in 25% of the cases. This was unexplainable in 1982. That is different now. A serious form of chronic pain is CRPS, what used to be called reflex sympathetic dystrophy. CRPS is an intense



inflammatory reaction in joints, usually unilateral. The most affected joints are wrists, knees and ankles. Infrared thermography shows that very clearly. It is then a very warm area. At a certain moment this inflammation extinguishes leaving behind scar tissue. The area is then cold, despite the fact that the same inflammation factors were found in the tissue as in the warm area. (Groeneweg JG. Cold Case. Thesis, Erasmus University 2009).

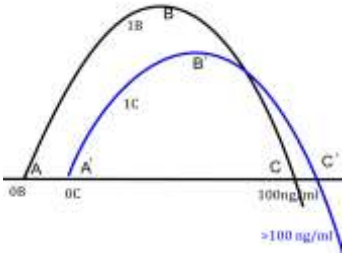
"Ordinary" chronic pain sites are usually cold, and 25% of them are warm. It has never been investigated if the inflammation factors are present in these pain sites and therefore it has also not been investigated if the same factors appear in warm pain sites as in cold ones.

Onset of CRPS from chronic pain sites is currently explainable:

Veen PHE van der. A theoretical model of biochemical control engineering based on the relation between oestrogens/progestagens and prostaglandins. Medical Hypotheses 84 (2015), pp. 557-569 DOI information: 10.1016/j.mehy.2015.02.021

Veen PHE van der. CRPS A contingent hypothesis with prostaglandins as crucial conversion factor. Medical Hypotheses. 85 (2015) 568-575. DOI information: http://dx.doi.org/10.1016/j.mehy.2015.07.017

Adapted from Horrobin & Manku



The presence of prostaglandins is the basis for that statement. It has been known since the beginning of the 1970s that all the active substances in the body are accompanied by a sort of "buddy". Without this buddy there is no mechanism of action. These buddies are called prostaglandins. The relationship of prostaglandins and pain has been scientifically investigated repeatedly. A few studies on the dose-effect relationship of prostaglandins and their effect on the substances for which they are buddies were published in 1977. Prostaglandins do not have their own effect without their effectors. The effectors also have no effect without prostaglandins. Only the combination of the two has an effect. In addition to the concentration of the effector, the concentration of the prostaglandin also influences the effect. The same studies show that the effect of certain types of prostaglandins have a bell shape. In a graph it is a parabola which the X-axis crosses at two points. Those are the two points marked "A" and "C" in the diagram.

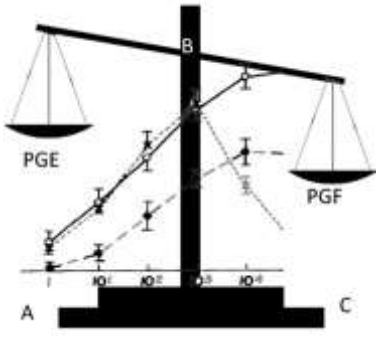
The effect of the effector reverses in substances with which this type of prostaglandin functions as a buddy. Examples in medicine are substances such as ergotamine which only relatively recently started being prescribed as an antimigraine drug. Ergotamine has a vasoconstrictive mechanism of action. It is prescribed in low doses as a blood vessel dilating drug, hence its effectiveness in treating migraines. Another example would be high doses of the cardiac drug "Digoxin" which provides the opposite effects that it counteracts in low doses. There are many substances in medicine that function this way. The doses that bring about opposite effects are not homeopathic doses, but these findings could put a number of homeopathic matters in another perspective.

Point C is like a dilation/contraction switch. Theoretically, one molecule can make the difference. Of course the opposite is also possible. A switch turned off can be turned back on again. There is something to be switched on and thus also switched off: the time of onset perhaps. Where do you see more onset times than during a woman's menstrual cycle? When the properties of onset are applied to varying hormone concentrations of oestrogens and progesterone during a woman's menstrual cycle, a basal temperature curve (BTC) then appears as a result of that. That is the periodic body temperature curve of a woman. A surprising, buy actually very logical effect. These results were published by the author in the Medical Hypothesis journal in 2015.

What does this have to do with chronic pain and CRPS?

The following was published as a hypothesis in 2015.

Prostaglandins are crucial substances in the development of inflammation. As mentioned earlier, biologically active substances in the body have no effect in the absence of their buddy. Biological substances are, for example, also inflammation-promoting proteins (cytokines) such as TNFα and IL-6 (Interleucine-6).



In a balanced situation two opposite active prostaglandins (PGF and PGE) are present in equal amounts. The balance is kept steady. The effect is at level B.

Imagine PGE is vasodilating and inflammation-promoting, and PGF is vasoconstrictive and inflammation-inhibiting. PGE then increases in concentration and the effect decreases. The balance tilts to the advantage of PGF.

It seems like a conflictual event. When the concentration of vasodilatory prostaglandin increases the vasoconstriction effect becomes greater. However, the concentration of the biologically active buddies such as TNFα and IL-6 also increase when the concentration of prostaglandin increases, but along with that the effect decreases until the concentration of X exceeds the limit of point C. The inflammation then expresses itself with all the classical symptoms. An unmanageable, self-reinforcing inflammation can develop under certain circumstances. Just like at the start of the inflammation, a phase of vasoconstriction occurs with a high concentration of inflammation factors during recovery. That is exactly the same situation that occurs with CRPS.

And it is also the situation that occurs with every injury that arises (infection, bleeding, bone breakage and bruising). So why are TNFα and IL-6 not present with chronic pain and a cold skin site? This has never been investigated. As such, this dynamic suggests the presence of actual tissue damage and a signal that indicates it.

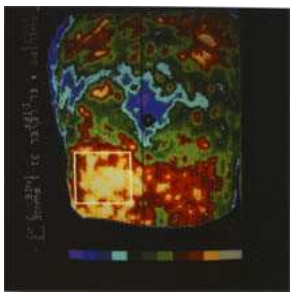
Diagnosis of chronic pain

The situation in the pain site can be detected with infrared video thermography, measured and followed, in addition to the more invasive and expensive skin blister technique with which fluid is extracted from the skin and analysed in the laboratory.

As far as the author knows, neither one nor the other method is used today. Compared to inventories and the Mc Gill Pain Questionnaire, there is an alternative, more objective, simpler and less expensive technique available for diagnosing chronic pain. It is amazing that it is still not being implemented.

Examples from 1980 to illustrate:

This is an image of a man with pain standing after bladder catheterisation. A red spot is visible in the urethra right in front of the bladder. That is a warm pain site with a skin temperature of 35 °C, which is 6.5 degrees warmer than the surrounding area. There was no blood contusion. The spot disappeared after two weeks.



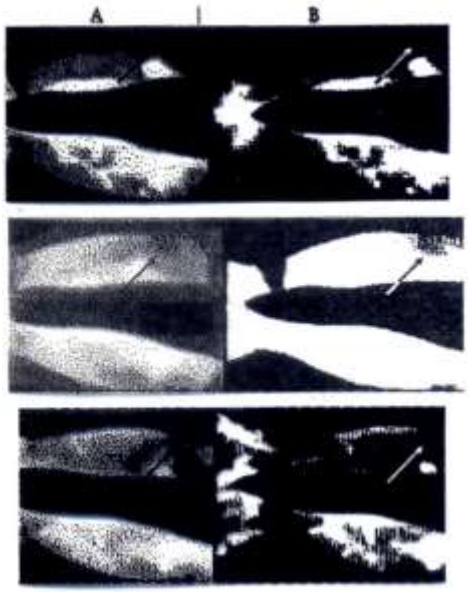
This is an image of a man lying down with gall bladder inflammation. The body is vertical in the image. The square area is a warm pain site in the upper right abdomen. The skin temperature there is a mean
35 °C, which is approximately 4 degrees higher than the average of the entire image.

This is an image of a man lying down with a swollen head of the pancreas. He is pointing to the site with his finger. The body is vertical in the image. The head of the pancreas is on the right. The finger is on the area of the navel.

These are merely a few images from a whole series. The images are evocative, but scientific research only has two starting points: starting where another ends or beginning with observations that are surprising. The latter applies here.

Here is a link to a PowerPoint presentation for those who would like more information about video thermography: Possibilities and Pitfalls.pptx :
https://www.dropbox.com/s/bnv2p4sktv8yva1/Possibilities%20and%20Pitfalls.pptx?dl=0

A video was presented in English at a conference in Sao Paulo in 2015:
https://www.dropbox.com/s/d00535ws62p8ht5/Presentation-Henk-van-der-Veen-lores.mp4?dl=0



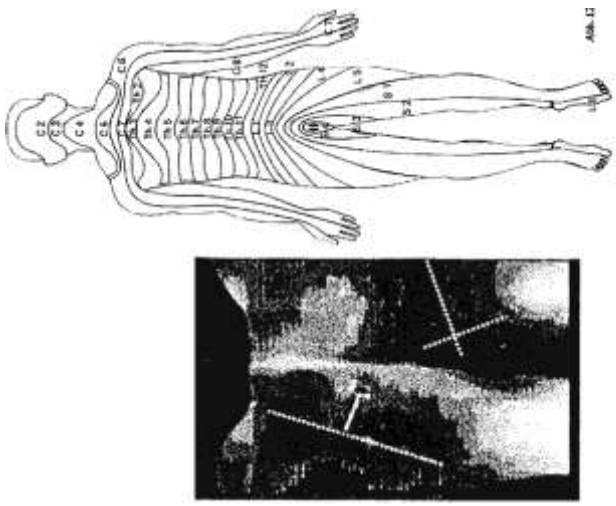
Finally, this image is a typical example of chronic pain taken in 1981. It is an image of a lower arm of a flower shop owner who broke it at the elbow after tripping over a bucket. Because this image was made using a mirror on the hanging camera, the right is actually the left. Please note the wristwatch on the left wrist (B2). It looks like it is the right arm, but it is the left. This was treated by an orthopaedist. It healed well clinically. However, some complaints remained, particularly symptoms affecting temperature (cold), fluid and strength. Rest lead to improvement. He was severely limited in his ability to work in his shop.

The images under column A are the original thermography images (grey). From top to bottom: under normal exertion, after rest, after moderate exertion.

The images under column B are the same images after entering a "threshold value". The image becomes clearer when all values below a set temperature are filtered out. White is a normal temperature and therefore also normal blood circulation. Black is where blood circulation is less in comparison, hence cooler. The arrow points to the breakage site

The old breakage site is still colder than the surrounding area even during a rest phase. Upon exertion a large difference develops between the images in column A and the images in column B, namely, the right side shows a much more disadvantageous situation reflecting much more pain than the left side. This would be impossible to see without infrared thermography. An assessment of the severity of the complaints would also be impossible. Here too we see inflammation at the start that leaves behind a scar that is either visible or not after recovery. In the case of the flower shop owner, we do not see a visible scar with the naked eye. These images suggest a relation between chronic pain and a now invisible scar, which is tissue damage.

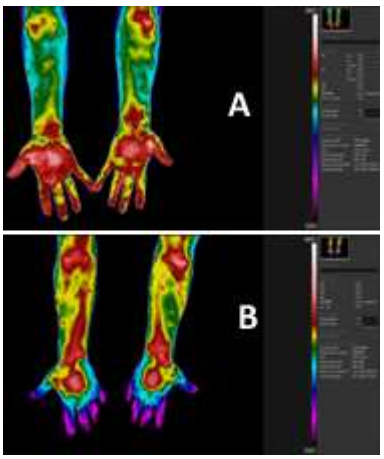
Would this man be helped very much with some form of psychological treatment given that the assumption is always that the problem lies somewhere "between the ears"? This is the conclusion drawn because there is no longer any tissue damage, which in all probability is actually present, but gets overlooked.



Here is one more example. The patient is lying on his stomach showing the area from the buttock to the knees. This patient was bitten in the buttock and upper legs by an attacking dog. The areas of the bite are indicated with dotted lines. He healed well clinically, but pain remained. The areas of the bites are black in the image, and hence cold. Is this psychological in nature like a senseless alarm, or is there actual tissue damage with inflammation factors under resting conditions? Therapy is also possible based on these diagnostics.

Therapy not yet implemented

As mentioned earlier, most of the chronic pain sites are cold with a limited number of them being warm. A statistical relation was found between changes in temperature and changes in the sense of pain. Pentoxifylline (PTX) was the medication used in this study, which is still prescribed for the same indication and is therefore inexpensive. It makes cold sites warm and warm sites cold. We close with an example from 2015.



Currently the same medication is frequently used worldwide in laboratory animals with bleedings and bone breaks, and when specific inflammation in which the same cytokines play a role in CRPS. This medication has also been used in recent research on inflammation situations in the central nervous system (brain and spinal cord) and sensitivity changes for nerve conduction.

It also seems to have an inhibiting effect on cytokines TNFα and IL-6. That would explain the reduction of warm pain and is also an indication for the involvement of these cytokines in chronic pain.

Image A above shows two warm hands and two cold lower arms. After treatment with PTX the hands became cold and the lower arms became warm (image B). (Images taken in 2015)

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