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FROM THE EDITOR

Dear Readers and Colleagues,

Another year has rapidly flown by and the e-journal continuous to grow. It now has an **ISSN number and the articles are peer reviewed**. It is an ongoing challenge to manage the e-journal as the editors and contributors are volunteers. Again, a heartfelt thank you, for all those who contributed. As the Editor, I encourage each of you and your colleagues to submit to this International Biofeedback e-journal, which is the only biofeedback journal published in Europe.

Our field is rapidly expanding and we received a number of workshop submissions for the upcoming conference. Due to limited space, however, we were only able to accept about 50% of all workshop submissions. Most if not all of the submissions were outstanding. The committee wants to emphasize that the rejections of the submissions were not a rejection of their content or presenters—just not enough space. Thank you for your continued contributions to this field.

We invite all of you to submit abstracts for symposia, paper and poster presentations for the scientific sessions of the BFE meeting. We are looking forward to see you in Rome to share knowledge and visit the ancient history. In addition, we hope that the Rome meeting will also stimulate more applied psychophysiology in Italy. Thus, the BFE meeting can contribute to the growth and interest in this field. This issue of *Psychophysiology Today* focuses on peak performance training in golfers, chronic sexual pain and relaxation therapy for rehabilitation in ischemic heart diseases. In addition, it includes pragmatic stress management tips as well as relevant abstract. Finally it could be YOUR article that gets published in one of the upcoming magazines therefore let me invite you to submit your research, clinical reports, and pragmatic descriptions of practices that enhance clinical or educational practices.

Have a look inside and enjoy.

Monika Fuhs

Editor in Chief

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PRESIDENTIAL NOTE

Having just returned from the 13th BFE meeting in Eindhoven, I am already looking forward to attending the 14th BFE meeting in Rome and learning even more about the amazing educational and clinical biofeedback advances presented during the workshops and scientific sessions. In many cases biofeedback is much more than just attaching sensors and having a person practice some prescribed skill. Biofeedback, like any other clinical/educational health practice, implies integrating an appropriate procedure at a time that is right for the patient. The ongoing challenge for any biofeedback practitioner is knowing what, how and when to apply integrated holistic psychophysiological techniques.

Biofeedback allows the invisible to be visible, the undocumented to be documented. With equipment or enhanced guided sensory awareness, symptoms below conscious awareness may be observed and noted. As an active teaching and learning approach, clinical biofeedback techniques offer advantages such as: a) providing a psychobiological model of the disease process to be coupled with a believable/ congruent experience of positive change and hope; b) an opportunity for reinforcing the biofeedback information with actual physiological and cognitive skills that supports self-healing; and, c) an ongoing involvement with healing process through the practice of appropriate psychophysiologically derived homework. A broad range of biofeedback applications is again presented in this superb issue of *Psychophysiology Today*. For example, applications ranging from enhancing peak performance (e.g., with golfers) to treating specific disorders (e.g., with pelvic floor pain patients). In many cases, biofeedback treatment includes skill training to bring about effective change as well as to facilitate shifting any feelings of helplessness and hopelessness into hope, empowerment and health, through the experience of physiological mastery.

To keep abreast of the dynamic expanding field of applied psychophysiology, I invite you to attend and actively participate in the 14th BFE meeting in Rome. Submit your proposals for oral presentations or posters to the program committee so that you can be part of the program and share your knowledge.

I look forward to seeing you in Rome.

Erik

Erik Peper, Ph.D.
President, Scientific Advisory Board

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Understanding Chronic Sexual Pain in Women

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Case study
My battle with primary vulvodynia began at the age of 10. Until last year, at the age of 33, this condition was a nameless, unacknowledged, humiliating thorn in my side. It has caused me profound frustration and millions of silent tears. I had fought with medical opinion, expressed, implied or completely ignorant, that my problem was non existent because my vulva looked healthy enough. In the last 5 or so years I had completely given up looking for help because I have always come away feeling like it must have been 'all in my head' even though I've always known deep down it wasn't. I have lived with constant vulvar discomfort and/or pain, itching, burning, and a 'tight' feeling at the vaginal entrance. I have not been able to wear pants at all or ride a bike, as these things have been so painful. Obviously, not being able to wear pants has affected my life in many ways – not able to follow fashion, apply for jobs if pants are the uniform, or go to the gym.

The first tampon I used at age 15. It was such a memorable experience for many reasons. Firstly, I could barely get it in, no matter how much lubricant I used. I tried a hot bath, relaxing etc. But eventually I just used brute force. Mum said it would get easier and I was only having trouble because it was the first time. It never did. The first tampon would not come out and I ended up at the GP's after hours, and he cut my hymen to get it out. He didn't seem to think it was a big deal. I worried all through my teens about how I would ever have sexual relations, my external vulva area was always painful – it waxed and waned but was never good.

I had a gynaecological surgery at age 17 where my labial tissue was reduced (almost entirely removed as it turned out). The idea was that less labia the less irritation. Apparently many women have this operation and gain much benefit. I didn't.

I met my husband at age 20. I was still a virgin, and still hoping that somehow once 'passion' took over it would be okay. It was hard to have him fall in love with me and then have to tell him that our love life would be difficult. I feared rejection and felt like a failure as a women – I still do. Our sex life has always been hampered by my pain and penetration has always been slow and painful. Afterwards, I always have the feeling in my vagina of burning, likesomeone has scrubbed my insides with a scrubbing brush. This feeling lasts for up to an hour.

Over the last two years, the problem deteriorated to the point it is today. I avoid sex as I find it exhausting emotionally and physically to get beyond the pain barrier. I am sick of trying to guess what is causing my pain but find it still plagues my mind daily. It is hard to forget about something that is one of the most defining things in your life.

Summary
Human sexual function exemplifies the close relationship that exists between mind and body. Vulvodynia, as a chronic pain syndrome, can disturb this harmonious psychophysiological relationship and impact on the well being of afflicted women. This highly prevalent disorder leads to psychological distress and significant changes in sexual and social behavior. The affective management of vulvodynia requires a good understanding of the mechanisms involved in chronic pain. Preliminary evidence points to the efficacy of psychophysiological protocols which aim to normalize pelvic muscle function in conjunction with therapy which addresses the emotional issues associated with chronic pain.

Introduction

Human sexual function is a prime example of the intricate interaction between mind and body. A harmonious psychophysiological response to emotional arousal and sexual desire enables individuals to experience heightened pleasure, interpersonal bonding and personal fulfilment. Yet, the experience of pain during intimate sexual activity, can by contrast lead to disappointment, loss of self confidence, distress and even social isolation.

The most common form of chronic urogenital pain is vulvodynia, an unexplained vulvar discomfort for which there is no known physical or neurological explanation. Considering the prevalence of vulvodynia, it is imperative to ask why so little attention has been given to understanding its aetiology and management. This article will briefly look at the historical interest in sexual pain conditions, the prevalence of vulvodynia, the possible pain mechanisms involved and the role of interventions based on applied psychophysiology.

Historical Perspective

Medical history provides evidence of prehistoric accounts of pain associated with sexual intercourse. Earliest literature references date back 3,500 years to ancient Egyptian papyri.

The writings of Soranus of Ephesus, a physician living in the 1st and 2nd century BC, provide the first description of what is classified today as vulvodynia. The literature was subsequently silent on this topic of sexual pain until 1874 when T. Galliard Thomas, in his textbook 'Practical Treatise of the Disease of Women,' described hyperesthesia of the vulva. Thomas commented "this disorder, although fortunately not very frequent, is by no means very rare…that it becomes a matter of surprise that it has not been more generally and fully described…" After another century of silence, the last 25 years has seen over a 100 studies published focusing on the various etiologic, medical and psychological issues in vulvodynia.

Prevalence of Vulvodynia

The prevalence of vulvodynia has been estimated by various studies at 4-19 %. Until recently, it was generally accepted that vulvodynia represented a Caucasian disorder. Several recent studies have dispelled this myth by showing that the condition exists among various racial and cultural groups. A Harvard study of women living in the Boston area found the prevalence among Asian women to be 11.1%; Hispanic women 22.7%; African American women 16.2%; and Caucasian women 16.2%. In clinical settings, studies of African women attending a general medical clinic in Ghana also found rates of 22.8%; among Swedish adolescents attending an adolescent health centre, prevalence was reported at 34%. From these findings it is evident that the condition affects women of all races, ages, educational and socioeconomic backgrounds.

In a recent psychosexual profile of 744 vulvodynia patients, I examined the age distribution of patients as well as the age of reported symptom onset. The findings were unexpected and not only confirmed that vulvodynia affects women of any age, but for the first time showed that the highest prevalence of this condition occurred in women younger than 25 years of age (See Figure). Prevalence decreased noticeably from age 26 to 39, with only a slight increase during the perimenopausal stage of life, remaining low for the rest of the life span. A further analysis of the age of symptom onset identified young women in their teens and early twenties as being at greatest risk of developing vulvodynia. These findings contrast with some of the earlier studies where the average age of patients was shown to be in the late 30's and early 40's. Although some recent reports alluded to a 7-8 fold increase in reports of discomfort associated with early tampon use among vulvodynia patients, women in the early onset group, referred to as the primary vulvodynia subgroup, often reported unexplained vulvar pain predating menarche and tampon use. Such early onset could be illustrated by numerous clinical case studies, but is well exemplified by a patient who recounted early childhood memories of placing band-aids over her vulvar area because it was constantly hurting and the health care providers could not find any reason for the pain. Others reported difficulties in childhood wearing tight swim suits, riding bicycles and playing on recreational equipment. With reports of vulvar pain dating to preschool years, it is essential for future studies to assess in more detail how early reports of symptoms and pain-induced-preoccupation with the urogenital area impacts on the development of body image, self-esteem, personal confidence and attitudes towards sexual behaviour in adult years.

The psychosexual impact of vulvodynia on sufferer is particularly evident among the primary vulvodynia patients who, in contrast to the late onset cases, referred to as the secondary subgroup, were found to delay the first experience of sexual intercourse by an average of 2.4 years; had fewer sexual partners; rated first sexual intercourse as less satisfying; and experienced higher levels of depression and anxiety while single. During interviews, women spoke of their teen years as marked by a perpetual fear of future sexual relations and concerns about rejection and social failure (see case study). Others, especially those who were single at the time of referral expressed an ongoing reluctance to enter into relationships and spoke of themselves in very negative and deprecatory terms labelling themselves as “deficient” and “faulty.” It would appear that the early onset and longer duration of undiagnosed and unexplained symptoms may be a precursor to negative emotions, anxiety and even phobic attitudes toward sexual intercourse in later years.

Evidence of the impact of vulvodynia can be also seen in significant changes in sexual behaviour of women. The majority of patients (80%) reported a decrease in sexual desire and a reduction in the frequency of sexual intercourse. Women frequently found themselves declining their partner’s advances, as partners did not show a comparable decrease in their sexual desire. The existence of a ‘desire discrepancy’ between patient and partner can potentially lead to relationship disharmony and is one of the core concerns of patients. Due to chronic pain many patients (43.5%) reported being abstinent at the time of assessment with abstinence being highest among single women (51.9%). Patients consistently rate pain associated with sexual intercourse at the higher end of the pain rating scales, thus confirming that vulvodynia, more so then other chronic pain conditions, has an emotionally and physically disabling impact on patients. Pain, once provoked, can be so intense as to disrupt sexual activity, arouse fear, lead to loss of sexual desire, avoidance, or total abstinence. Unlike the normal distributions of pain ratings for other chronic pain conditions, vulvar pain is consistently rated high on pain scales, confirming that is has a disruptive and disabling impact on the quality of life of the women affected.

The inability to engage in intimate sexual behaviour impacts on women’s sense of self-esteem and is associated with higher levels of anxiety and depression. However, the primary motivation for seeking therapy was to improve their relationship opportunities or, if in a relationship already, to increase the frequency of sexual intercourse. When asked what activities they would like to increase upon completion of therapy, the majority of patients (85.5%) specifically expressed a desire to increase the frequency of sexual intercourse. This finding confirms that abstinence from sexual activity is not due to malingering, as sometimes suggested, but is the direct result of pain.

Reports of an early onset of symptoms have often raised questions about the possible role of sexual abuse in the etiology of vulvar pain. The reported incidence of unwanted sexual activity during childhood and adult years has been shown to be approximate that in the general population. Sexual abuse does not appear to be related to the onset of vulvodynia symptoms.

From the discussion thus far, we can see that the prevalence of vulvodynia is underestimated and that the condition interferes with sexual function, affecting an individual’s ability to engage in intimate sexual behaviour. In severe cases, pain associated with sexual activity, can prevent couples from sexually consummating their relationship which can also interfere with their ability to conceive and establish a family. The negative impact of vulvodynia on the general well being of women makes it critical to identify the condition as early as possible.

Psychophysiological Mechanisms in Vulvodynia

In order to develop successful therapeutic interventions for the management of chronic vulvar pain, it is imperative that greater emphasis be placed on understanding the underlying mechanisms of pain. This is a challenging task because chronic pain syndromes like vulvodynia are characterised by a complex interaction of psychological, physiological, and behavioural variables alluded to in the earlier discussion.

Pain is a sensory and subjective phenomenon, most commonly defined as an “unpleasant sensory and emotional experience.” Accordingly, the experience of pain involves both sensory peripheral mechanisms and higher cortical functions involved in interpreting and modulating the perception of

pain. The International Society for the Study of Vulvovaginal Disease (ISSVD) in defining vulvodynia as 'vulvar discomfort, commonly described as burning pain which occurs in the absence of relevant physical findings or a specific, clinically identifiable, neurologic disorder' has provided a clear definition of vulvodynia. The definition highlights three key points: (1) the location of the pain; (2) its sensory qualities; and (3) its unknown but potentially multi-factorial nature. In relation to the location of pain, the term vulvodynia is descriptive in that it identifies the anatomical area affected – namely the vulva and the Greek term odyno points to pain as the primary symptom. In regards to the common sensory descriptors used by patients these are of a thermal and incisive nature, highlighting the involvement of peripheral sensory mechanisms. Finally, the definition emphasizes the absence of any specific physical or neurological findings and in this regard vulvodynia typifies the chronic pain syndrome anomaly where pain is not proportional to, or explained by, visible pathology.

Unfortunately the ISSVD definition of vulvodynia makes one major omission in that it totally fails to acknowledge the role of emotional factors in the development and maintenance of symptoms. Without recognition of the role of sensory and subjective elements in pain, research has tended to focus exclusively on either the psychological or physiological, without scrutinizing the interaction between the two variables. Both extremes are contrary to current opinion on general chronic pain syndromes, chronic pelvic pain, and vulvodynia.

There is a high prevalence of depression and anxiety among vulvodynia patients, likewise physiological research has shown unique characteristics which include, increased density of superficial nerve endings, increased immunoreactivity, and nociceptor sensitivity. Such physiological peculiarities, when considered in conjunction with commonly reported psychological traits of elevated anxiety and depression, may potentially constitute a partial predisposition to chronic vulvar pain and moderate the severity of symptoms.

The interaction between emotions and physiology can occur at different levels of functional complexity. At the higher cortical level, functional Magnetic Resonance Imaging (fMRI) studies have demonstrated that mechanical/tactile allodynia primarily activates the anterior cingulate cortex and thalamus. These cortical structures are also known to be involved in the experience of conscious negative emotions. At the peripheral sensory level, a significant interaction has been demonstrated in vulvodynia between anxiety and nociceptor sensitivity. Studies using quantitative sensory testing (QST) in female genital sensation showed that vulvodynia patients have lower pain thresholds in the vulvar vestibule, leading some to suggest that the condition is a form of neuropathy. But, a closer analysis of the variation in pain thresholds between vulvodynia patients and controls revealed that the difference was mediated by anxiety. Vulvodynia patients experienced enhanced pain perception, greater emotional response, increased autonomic reactivity, and scored significantly higher than controls on measures of state anxiety (unpleasant emotional reactions) and trait anxiety (personal tendency to respond with state anxiety), demonstrating the significant role of emotional states on nociceptor sensitivity.

When acute vulvar pain enters the chronic phase (vulvodynia), normal sensory processes are affected by progressive sensitization of the peripheral and central nervous system. Sensitization, is an important property of nociceptors and manifests itself in decreased thresholds to nociceptor stimulation; an increased field of nociceptor reception (from localized to generalized); nociceptor responsiveness to normally non-noxious stimuli (allodynia); increased intensity of response (hyperalgesia); prolonged post-stimulus sensations (hyperpathia); and the occurrence of spontaneous pain. Such sensory changes are the defining characteristic of vulvodynia. From QST studies there is growing evidence that secondary hyperalgesia of the vulva is in part modulated by anxiety. This finding is consistent with studies of other pain syndromes showing that the most reliable predictors of hyperalgesia are anxiety, fear, and catastrophizing.

One of the known physiological correlates of anxiety is elevated muscle tension, a form of inefficient peripheral response to the hyper-alertness commonly seen in chronic pain patients. Muscles are the body's primary responders to pain, trauma, injury, and negative emotional states. Sensory and emotional stimuli can contribute to muscle over-activation, as assessed by sEMG, and can lead to stiffness, spasm and pain, especially in symptomatic muscle groups. SEMG monitored muscle over-activation has, in some conditions, proved to have diagnostic value. This symptom specific involvement of muscles has

been extensively documented in vulvodynia studies, linking pelvic muscle hypertonicity (sEMG over-activation) and muscle instability to symptoms of vulvar pain.
In a recent study of 529 vulvodynia patients, I examined the relationship between emotions, muscle over activation and pain in the context of vulvodynia. The study found a significant correlation between vulvar pain, anxiety, depression and global distress (as measured by three global scales of the SCL-90R), highlighting a positive relationship between pain and negative emotions. Although it is not possible to establish causality on the basis of correlation, the prevalence of anxiety and depression among vulvodynia patients necessitates that further research attention be directed to examining the relationship between severity of vulvar pain symptom and intensity of emotional distress.

The difficult question that arises from these findings is whether the pain contributes to emotional distress or emotions contribute to pain. Anxiety and depression have been shown to be prevalent in most other pain conditions and pain related hyper-vigilance is a common trait in chronic pain patients. Research with vulvodynia appears to confirm that elevated anxiety may be a contributing factor to secondary hyperalgesia. In a similar manner, it may be possible that depression, because of its high prevalence, may act not only as a passive pain coping strategy but another contributing factor in the psychophysiological complexity of this pain syndrome.

Data from sEMG findings in this study provided further information on the potential mechanisms underlying the symptoms of vulvodynia. In general, muscle over-activation has been identified as a factor in a range of chronic pain syndromes and the mechanisms by which muscle over activation leads to pain have been extensively discussed. It is accepted that muscle over activation leads to muscles decompensating in a painful manner. Irrespective of whether muscle tension is due to emotional or physical triggers, ischemia, hypoxia, build up of neurogenic metabolites (lactic acid, potassium, arachidonic acid), alterations in intramuscular blood flow, release of sensitizing agents (such as bradykinin and serotonin), inflammation, erythema, oedema formation, muscular rigidity, all can ultimately lead to pain. However the sEMG data showed no correlation between sEMG resting amplitude or standard deviation in relation to the intensity of pain. This is consistent with most of the earlier research findings, but contrary to an earlier vulvodynia study based on a small sample of patients. SEMG data from this study provides only qualified support for an association between pelvic muscle over-activation and vulvodynia symptoms.

A conceptually challenging finding arising from a further analysis of sEMG data is the negative correlations between resting amplitude and standard deviation readings, and the duration of pain. Chronicity of vulvar pain appears to be associated with a progressive reduction in muscle electrical activity. This paradoxical finding may be associated with a time related physiological “shut-down” of over activated muscles. Clinically, it is not uncommon to see long-term vulvodynia patients who complain of debilitating pain and introital muscle tightness but who on assessment, show sEMG readings that are exceptionally low and stable, resembling those of symptom free individuals. This anomaly may be associated with progressive shortening of over-active muscle tissue and the development of a muscle contracture that is consistently described by patients as painful and as an obstruction in the vaginal introitus.

Muscle contracture has been previously described as consisting of an electrically silent, involuntary state of maintained muscle shortness and decreased extensibility (i.e. increased stiffness) of the passive elastic properties of the connective tissue. In relation to vulvodynia, such a contracture appears to be mediated by an anteriorly oriented tractioning of the pubococcygeus muscle (in particular the pubovaginalis and puborectalis portions) with a progressive anal retraction and clitoral descent. The loss of pelvic muscle extensibility appears to be consistent with findings derived from assessments using graded vaginal dilators. At the commencement of therapy, patients were only able to accommodate the smaller size dilators. This also appears to coincide with difficulties using tampons and undergoing medical exams. However, it is also very evident that such pelvic contractures are not permanent but “functional” and can be reversed by conservative therapy involving the use of dilator exercises and sEMG assisted retraining.

Muscle contractures may also give rise to myofascial “trigger points,” tenderness, and pain. Physical pressure on hypertonic and shortened muscles produces symptoms of sharp, shooting and burning pain in the absence of physical evidence. These anomalies are consistent with myofascial pain, which

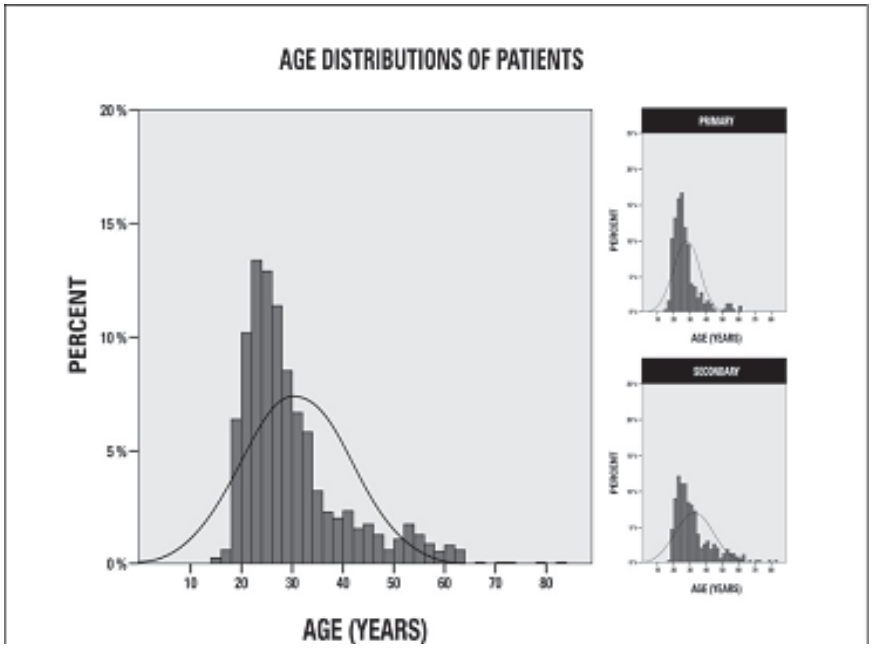
often causes functional difficulties but has no visible pathology.
Vulvodynia symptoms are frequently attributed to, and misdiagnosed as vaginismus. Vaginismus is defined in the DSM-IV as a "recurrent and persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with sexual intercourse." The similarities between the two conditions are that a functional contracture can resemble the acute shortening of muscles in spasm, but the main difference between a contracture and muscle spasm is the striking sEMG characteristic; contractures are electrically silent; However, in vulvodynia there is no sEMG evidence of any spasm-like activity in pelvic musculature. The existence of normal reflexes, such as the bulbo-cavernosus or sacral reflex and their modulation by the involvement of supraspinal sites, should not be confused with vaginismus. Considerable confusion surrounds the classification of vaginismus both in relation to vulvodynia, or as an independent diagnostic entity in itself. This study found no evidence of involuntary spasm in vulvodynia and sEMG assessment can assist in differentiating vulvodynia from other diagnostic conditions such as vaginismus.

Vulvodynia patients commonly report urethral and bladder symptoms in conjunction with vulvar pain. Previous studies have explored the possibility of parallel pathologies for vulvar pain and interstitial cystitis and there appear to be some similarities in symptoms reported (e.g., discomfort, burning and stinging sensation). Several studies of these co-morbidities have confirmed muscle over-activation, lack of voluntary control, inability to relax, shortening of muscles and trigger point referred pain, as well as hypersensitivity exacerbated by anxiety, sexual and physical activity. Ultimately, further research into these co-morbidities is required but may show that vestibulodynia, clitorodynia, generalized vulvodynia, urethral syndrome, and abacterial interstitial cystitis may share a common mediating mechanism and may be symptomatic of pelvic muscle dysfunction distinguished only by the locale of the pain.

A clinical protocol focussed on sEMG assisted retraining of pelvic muscles and the release of muscle contracture may alleviate, if not totally relieve the common symptoms of vulvodynia. Over 80% of patients resumed regular sexual activity at the conclusion of therapy. However, due to the complexity of human sexuality alleviating symptoms of discomfort and pain may not automatically lead to the commencement or resumption of sexual activity. Considering that 11-17% of patients upon completion of biofeedback assisted pelvic retraining remained sexually abstinent, highlights the need to address psychological difficulties that patients experience. With the prevalence of anxiety, sexual fear and depression, patients also need to receive sexual and relationship counselling and be helped to understand the relationship between emotional states and chronic pain.

Conclusion

The prevalence of vulvodynia has been underestimated. Although the condition affects women of all age groups, young women under the age of 25 are at the highest risk of developing this disorder. The negative impact of vulvodynia on the general well-being of women makes it critical to identify the condition as early as possible. As a chronic pain syndrome, this disorder may be most responsive to psychophysiological protocols which recognise the intricate interaction of psychological and physiological mechanisms in the experience of pain. Conservative therapy, followed by qualified counseling can successfully restore pain free sexual function.



* This paper is a compilation of findings and discussion published in two recent publications:
Jantos, M., & Burns, N. R. (2007). Vulvodynia: Development of a psychosexual profile. J Reprod Med, 52, 63-71.
Jantos, M., (2008). Vulvodynia: A Psychophysiological Profile Based on Electromyographic Assessment. Appl. Psychophysiol. Biofeedback, In press.

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Psykinetics: The New Science of the Golf Swing
Psykinetics the Interaction of Mental States and Physical Movement

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Source: www.biofeedbackrus.com/images/graphics/thewall/PsykineticsTrainingGolf.pdf

Golf, like any other field of human endeavour, is characterised by revolution. If you are a modern player, your clubs, your balls, your training, probably even your swing, are revolutionarily different from those of your predecessors.

Walk down the line at the practice range of a major tournament nowadays, and the players look like they came out of a rugby team, and their equipment looks like it came from outer space.

But what about the mental revolution? The best efforts of Dr. Bob Rotella and James Gallway notwithstanding, are you any better prepared mentally for your round than your father was when he played the game a generation ago? The modern pros hit the ball further than Jack Nicklaus or Gary Player would ever have dreamed of, but are they any tougher mentally?

I don't think so. Even as a sports psychologist for the last 10 years, I can say until now, the mental revolution in golf has been a damp squib, a non-starter. That's not to say sports psychology is a waste of time. It isn't. I've taught a lot of golfers to use mental techniques that Jack Nicklaus used, and it makes them play a lot better. It's just that there hasn't been a dramatic advance in this generation like there has in the other areas of golf.

One reason for this is that sports psychology grew out of a field called cognitive psychology, which means literally, "the psychology of thinking". The problem is that brain imaging studies show that when you hit your best shots, you're not thinking at all. You are using parts of your brain that are not involved in consciousness. In some ways, cognitive psychology is barking up the wrong tree.

But there are three few scientific fields that do cast light on the mental processes involved in hitting a golf ball. These are evolutionary psychology, psychophysiology, and sensory discrimination. Together these make up psykinetics, the new science of the golf swing, and hold onto your hats folks, because the mental revolution is upon us.

Evolutionary psychology says when human beings get into conflict, they respond aggressively, submissively, or assertively. Each of those emotions comes with its own physical, sensory and neurological settings - like Boston's 1976 hit says, emotions are more than a feeling. Emotions affect which muscles, brain regions, senses and hormones we turn on or off. Without even thinking about it, as we move from emotion to emotion, we also move through different mental and physical states. Sometimes these mental and physical states are appropriate to the situation, and sometimes they are not.

Psychophysiology is the field that describes the connection between the mind and the body. Human emotions always manifest physically – in the unconscious but predictable activation and deactivation of certain muscles in the face and body. In fact, part of the experience of emotion is the physical feedback we get from muscle states. Anger wouldn't feel the same without the tight feeling you get when you contract your forehead muscles in a frown, and happiness wouldn't feel the same without the stretching feeling you get when you smile.

Sensory discrimination is our ability to use our senses to gather and use information about our internal and external worlds. For example, we use vision to tell us where things are, touch to give us information about objects we are manipulating, and balance and proprioception to tell us where our bodies are.

Why are you reading this in a golf magazine? Here we go …

Human beings move in a variety of ways. One of these involves rotating the shoulders above the hips, then driving forward off one leg before releasing the power from the shoulders and extremities. This creates a lever system out of the joints, and uses muscle groups in sequence to generate very high levels of power. This archetypal rotational movement is used by human beings to throw, punch, strike with an object, and kick.

My partner John Dickson and I call it the 1, 2, 3 movement, where 1 is the loading of the core, 2 is the initial release of power from the core, and 3 is the release of power into the extremities (Fig 1 and 2).

Fig 1: Positions 1, 2 and 3 in punch

Fig 2: Positions 1, 2 and 3 in throw

The modern golf swing involves taking the club back and rotating the shoulders around the hips until they are tight and loaded with energy. The swing starts with an explosive drive off the right leg, which pushes the hips forwards and coils the spine still further, creating an elastic energy which is then released into the shoulders, arms, and finally through the wrists into the club, and through the club head into the ball (Fig 3).

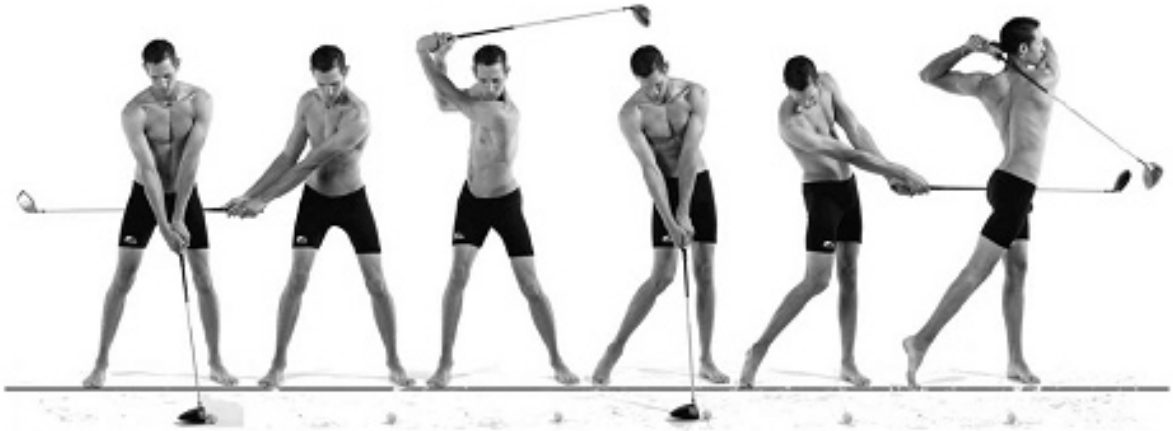


Fig 3, Correct swing sequence

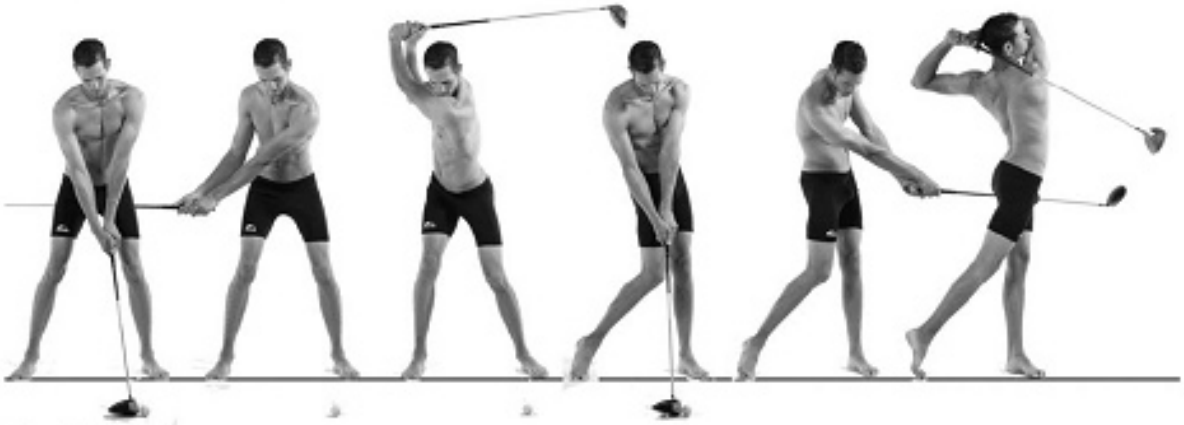


Fig 4, Pull swing sequence

Fig 5, Push swing sequence

A few years ago John and I started to realise that people hit the ball left primarily because of a failure to do 1 or 2 (Fig 4 & 6). When a golfer doesn't load his core sufficiently, or doesn't release power from his core sufficiently, his hips lag behind his shoulders in the downswing, which drags his swing plane or his club head to the left (Fig 7) causing pulls and hooks.

John and I also realised that people hit the ball right because of a failure to do 3 (Fig 5 & 6). When there is too much tension in the shoulders or arms, the wave of power from the core and spine cannot flow into the extremities. This drags the swing plane to the right (Fig 7), and takes the club-head late to the ball, causing pushes and blocks.

Fig 6: Pull (failure to implement 2), Push (failure to implement 3), Good shot

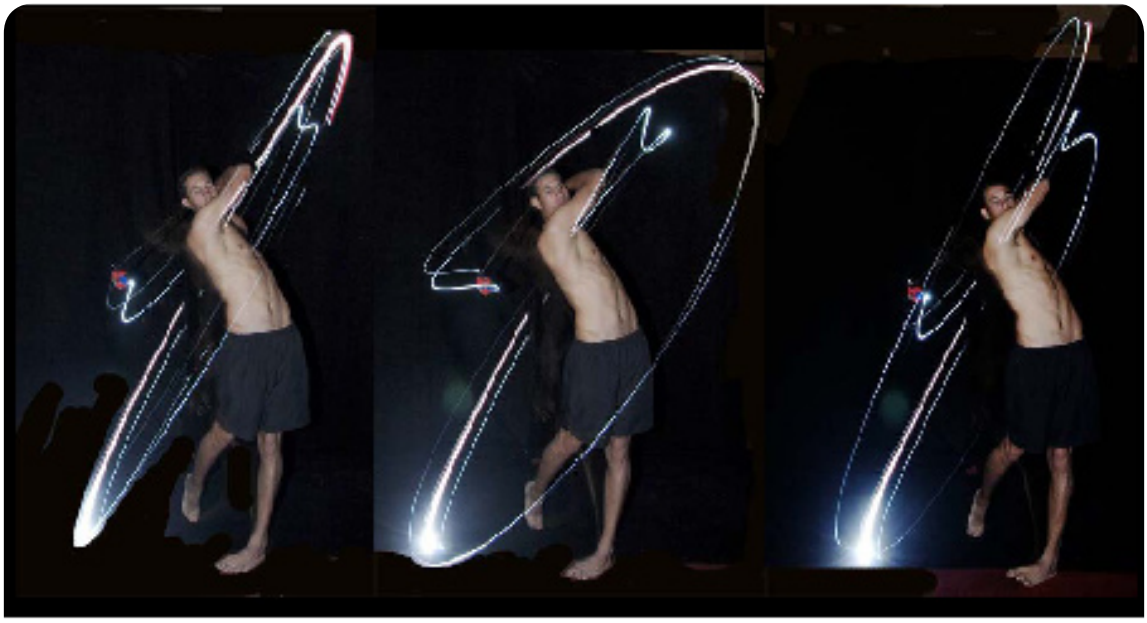


Figure 7, Correct, pull and push swing planes

This is where the evolutionary psychology and psychophysiology come in to make things exciting …

Remember, when I'm under pressure or in conflict, my evolutionary psychology gives me three choices; submission, aggression, or assertiveness.

On the golf course, submission is when I lack confidence or commitment to a shot, don't try hard enough, don't believe I can make it, or quit on it. Aggression is when I try too hard, when I feel I have to make the shot, or when I force it. And assertiveness is when I believe I can make the shot, and I decide to "just do it".

When we go into these different mental states, we involuntarily and subtly assume the corresponding physical position. This is body language. The body language of submission is a broken or disengaged core with soft hands, aggression is tensed muscles in the shoulders and forearms, and assertiveness is an engaged core with relaxed extremities (Fig 8).

Fig 8: Submissive, aggressive, and assertive body language

Evolutionary psychology and psychophysiology explain why we hit some shots left or right of our intended targets. When we become aggressive we tense up in our shoulders and forearms, causing pushes and blocks, and when we become submissive we weaken our cores, causing pulls and hooks.

One of the biggest tips I can give to golfers is 'protect your body!' What I mean is that when you get under pressure, that pressure attacks your body, changes your muscle settings, and messes up your swing.

Once I was watching Tiger practice his putting, and when his caddy threw him the ball he caught it with lovely soft hands. When I saw him putting on the course in the competition and his caddy threw him the ball, he caught it with exactly the same soft relaxed hands - he was protecting his body.

Tiger Woods, practice green, Carnoustie 2007

There are only two controllable reasons for bad shots in golf - bad swings and bad connections. Bad swings are caused by poor psychophysiology, and bad connections are caused by poor sensory discrimination.

Sensory discrimination refers to our ability to use our senses to gather and use information about the world. We've actually got nine senses, not five - sight, hearing, taste, touch, smell, balance, proprioception, visceral, and timing/rhythm.

Proprioception is my sense of where my body is in space. If you can close your eyes and clap your hands, that's proprioception. Your visceral sense is your sense of your insides - a full stomach or butterflies. Timing/rhythm is your ability to predict when the next event in a sequence will occur - for example, tapping your foot in time to a music beat, or closing your hand at the moment a ball arrives in it.

Tiger Woods about to implement pre-shot routine

Senses can get turned up and down. For example, when you listen carefully, you turn your sense of hearing up and hear sounds that you hadn't noticed before.

When you are under pressure the wrong senses for the golf swing (hearing, visceral) may get turned on, and the right senses (balance, proprioception, touch, timing, vision) might get turned off. When you are not under pressure (for example on the driving range), all the senses that you need are turned on, and all the senses that you don't need are turned off. And you connect the ball fine. But under pressure, this situation can start to reverse itself … and this explains the second category of bad shots.

Because if vision is turned off, you don't know exactly where the ball is, and if touch and proprioception are turned off you don't know exactly where the club-head is … and this is when you get bad connections.

It is important to think positively, but pressure doesn't just affect your thoughts, it affects your body and your senses also, and it's these two that make the difference between good and bad shots. Another big tip I can give to golfers is 'prepare for the shot!' Tiger's pre-shot routine is designed to get his body and his senses into the right arrangement for hitting a golf ball. It has the following steps:

1. Tiger checks his body is in the assertive setting (relaxed shoulders, arms, wrists and hands, strong, activated core).

2. Tiger only does a practice swing after he has corrected his psychophysiology, because if you practice swing with wrong muscle activation your swing feels wrong.

3. In the practice swing, Tiger feels the timing of his hips (2) and club-head (3). He turns vision on by targeting a blade of grass in the swing, and coinciding the timing of his power release with that specific target.

4. After the practice swing, Tiger stands still, and moves the club-head very slightly to turn on proprioception and touch, and rocks backwards and forwards on his heels, to turn up balance.

5. Now he's all switched on and all he has to do is go and hit the ball.

Psykinetics is a system that trains golfers to achieve the perfect physical and mental state for hitting a golf ball.

Just like your body, your brain has different parts that do different things. Neuroimaging pioneers like Brad Hatfield in the US and Claudio Babiloni from Italy are helping us understand what the brain does during a golf swing.

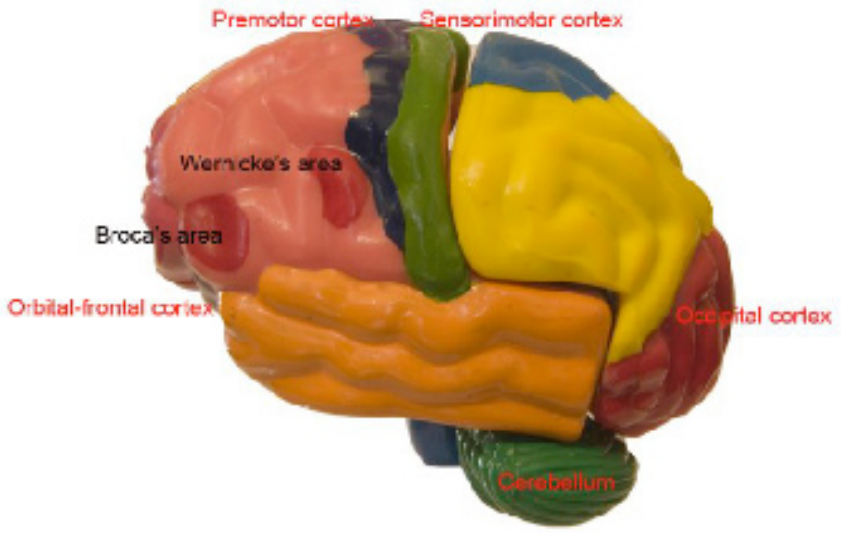


Fig 9: Anatomy of the human brain

Some of the relevant parts are:

- The cerebellum, which controls balance and coordination, and needs to be turned on
- The putamen, which stores muscle memory, and needs to be turned on
- The premotor cortex and sensorimotor cortex, which control motor planning and execution of movement, and need to be turned on
- Wernicke's area and Broca's area, which are involved in language and speech production and during a golf shot need to be turned off
- The orbital-frontal cortex, which is responsible for negative thinking, and needs to be turned off
- The occipital cortex which controls vision, and needs to be turned on

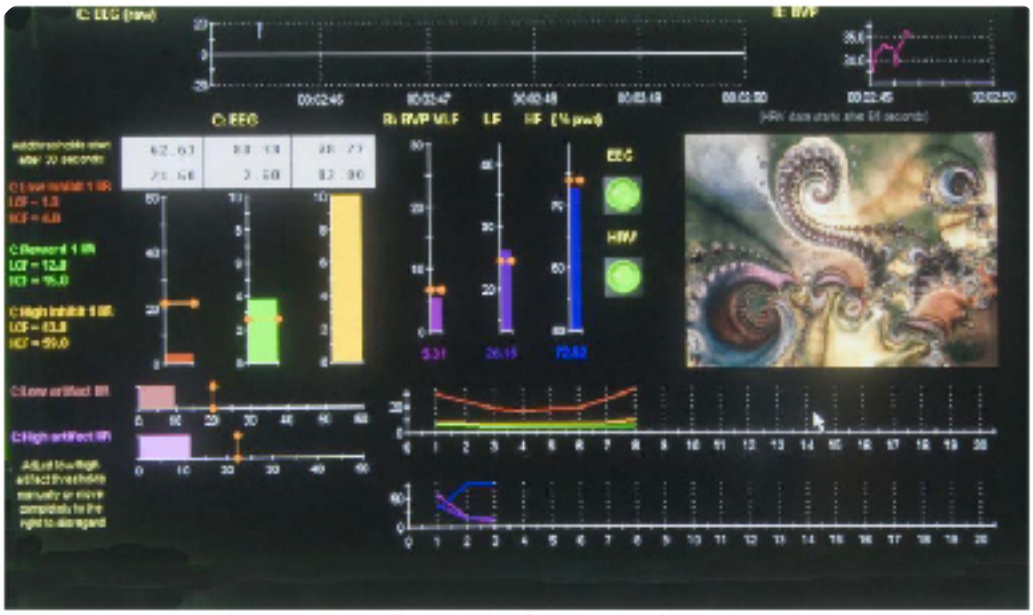
When these brain regions are activated correctly, the golfer will have all the right senses turned on, and all the wrong ones turned off. Babiloni showed in a recent research article that sensorimotor cortex activation during a putt predicted the result better than technique! So if your brain is turned on, the putt is going in the hole.

Psykinetic training uses a neuroimaging technique called electroencephalography (EEG). EEG is a technique that has been used by medical doctors for years, and is beginning to be used in sports psychology. It works on the principle that when your brain works, it emits small electrical frequencies. The harder it works, the higher the frequency it emits. By attaching sensors to the scalp, we can monitor which parts of the brain are more active than others. Because we know what brain regions you should be using (and not using) to hit a putt, we can use EEG to train you to get into the right mental state to hit a putt.

But the body needs to be turned on also. By using sensors to monitor muscle tension, skin conductivity, and the link between heart rate and breathing, we can tell when you are in the right psychophysiological state – and also train you to achieve this state at will.

In training you are wired via a Thought Technology encoder to a computer with highly specialised BioGraph Infiniti® software that interprets the neurological and physical data. In one drill, when you get into the right physical state a green light goes on and a tone sounds, and when you get into the right

mental state another green light goes on and another tone sounds. When both green lights are on, that's when you are ready to hit the shot; you're in the psykinetic zone.



BioGraph Infiniti® software training screen

There's nothing that special about the tone itself, it's no heavenly chorus, but it signifies one of the biggest 'Aha!' moments you will ever experience as a golfer.
Think of the first time you saw yourself on video, and finally understood your swing, and when it was supposed to look like. Biofeedback is that times ten. You suddenly realises what proper readiness feels like. You can experiment with different approaches until suddenly you realise how to turn both lights on at the same time and it's 'Aha! That's where I need to be to play a golf shot.'

Psykinetic training happens in a fairly simple routine. The first stage is learning to achieve the psykinetic zone in the office. You have to experience it for yourself, and discover how to turn the green lights on.

What I find is many golfers can produce the zone quite easily, but it's very fragile. So you might be in the zone, but there is a slight distraction and you slip out and struggle to get it back. Also, golfers don't always recognise when they are in the zone or not – and so often play shots before they are actually ready.

The second stage of training is to make your psykinetic zone more resistant to pressure – because as a competitive golfer, the ability to produce the zone under pressure, and on demand is as important as a good swing.

The third stage of psykinetic training is to get onto the practice green or driving range, and use biofeedback to train yourself to stay in the zone all the way through a shot.

I find golfers fall into three categories. The first category is the carefree/feel golfer. These golfers do get into the zone, but only in the last second or so before the putt. If they get their timing slightly wrong, or rush the shot, they hit the ball before they are in the zone, and miss the putt.

The second category are the serious/mechanical golfers, and they also get into the zone, but have a tendency to slip out of it again just before they play the shot, because they start to worry about the result.

The third category of golfer is the type who is disposed to anxiety, and in some ways, they are the most interesting. These golfers tend to get into the mental zone, but only get into the physical zone after the ball is struck. The reason for this is that they only relax after they have hit the shot. Interestingly, they relax regardless of whether it was a good shot or not – they're just glad it's over. These golfers tend to rush their preparation because they want to get the anxiety of the shot over and done with.

When we're working with golfers, sometimes we find that they hole putts when they are not in the zone – it is possible hit good shots with the green lights off. But what we also find is that it's very rare to hit a bad shot when the green lights are on.

Psykinetic training allows golfers to discover when they are in and out of the zone, and teaches them to increase the duration and strength of their zones so they can cut down on errors and hit better shots.

The final stage of training is to play a full round while monitoring your zone. By combining this with video analysis, we can tell which situations or shots cause you to slip in and out of the zone, and we can train you to overcome this.

The end result is when even faced with your toughest shot or situation, you can stay in the zone, and give yourself your best chance. These are a lot of things you can't control on the golf course, but with the technological revolution of psykinetic training, one thing you can now control is your zone.

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Relaxation Therapy for Rehabilitation and Prevention in Ischaemic Heart Disease

a systematic review and meta-analysis

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Source: van Dixhoorn, J. and White, A.: Relaxation Therapy for Rehabilitation and Prevention in Ischaemic Heart Disease - a systematic review and meta-analysis. European Journal of Cardiovascular Prevention and Rehabilitation 2005; Volume 12 No. 3: 1541 – 8267

Aims:
To establish the effects of relaxation therapy on the recovery from a cardiac ischaemic event and secondary prevention.

Methods and results:
A search was conducted for controlled trials in which patients with myocardial ischaemia were taught relaxation therapy, and outcomes were measured with respect to physiological, psychological, cardiac effects, return to work and cardiac events. A total of 27 studies were located. Six studies used abbreviated relaxation therapy (3 hr or less of instruction), 13 studies used full relaxation therapy (9 hr of supervised instruction and discussion), and in eight studies full relaxation therapy was expanded with cognitive therapy (11 hr on average). Physiological outcomes: reduction in resting heart rate, increased heart rate variability, improved exercise tolerance and increased high-density lipoprotein cholesterol were found. No effect was found on blood pressure or cholesterol. Psychological outcome: state anxiety was reduced, trait anxiety was not, depression was reduced. Cardiac effects: the frequency of occurrence of angina pectoris was reduced, the occurrence of arrhythmia and exercise induced ischaemia were reduced. Return to work was improved. Cardiac events occurred less frequently, as well as cardiac deaths. With the exception of resting heart rate, the effects were small, absent or not measured in studies in which abbreviated relaxation therapy was given. No difference was found between the effects of full or expanded relaxation therapy.

Conclusion:
Intensive supervised relaxation practice enhances recovery from an ischaemic cardiac event and contributes to secondary prevention. It is an important ingredient of cardiac rehabilitation, in addition to exercise and psycho-education.

Introduction:
A quantitative review of psychosocial interventions such as stress management, cognitive behavioural therapy, health education and relaxation added to standard medical care for patients with coronary artery disease concluded that these interventions had a substantial effect in reducing distress, blood pressure and cholesterol as well as subsequent mortality and cardiac recurrence [1]. A later review [2] found positive effects of psycho-educational programmes on the same risk factors for cardiac disease and a reduction in cardiac mortality and recurrence of myocardial infarction (MI), but no effect on coronary artery bypass surgery or on anxiety and depression. Neither review provided evidence to recommend any specific form of treatment. Because it is not clear which component is effective, guidelines for cardiac rehabilitation deal with the issue of psychosocial treatment and stress management in a variable way, either recommending [3] or not recommending it [4]. Ades [5] suggested that all cardiac patients should be offered stress management classes, but it was not included as a core component of cardiac rehabilitation [6]. Relaxation therapy has been used in cardiac rehabilitation since 1970 [7], but the evidence for its effectiveness has not been reviewed.
We define relaxation training or therapy as teaching the individual to induce a reduction of tension within

himself, without using external means [8]. Relaxation therapy has been studied for almost a century [9] during which time different norms have been developed. Typically, techniques focus on: attention (active or passive); mental representations, images, and words (including response to cues); small movements or posture changes; muscle contraction and relaxation; breathing instructions; sometimes aided by biofeedback. Teaching may be in groups or individually. Different authors call this variously relaxation therapy, stress management, psychological or nursing intervention, or meditation. Traditional relaxation therapy is intensive, usually involving at least several months of training to master basic skills and education in implementing strategies. The subject learns to notice moments of low and high tension in daily life, to find restful moments for practice and to cope differently with high tension periods. Early warning signals indicate rising tension and need to unwind afterwards. These are secondary relaxation skills, which increase the awareness of stress and motivate better management. Later, abbreviated and simplified forms were developed, culminating in Benson's relaxation response [10], which can be taught in a single session. Subsequently, these simplified forms were expanded with cognitive and educational strategies, which has been present in traditional forms, and included in cognitive behavioural treatment. For this review we were interested in studies that teach relaxation skills as a central part of treatment. Moreover, we were interested as to whether the time spent on teaching relaxation skills affected outcome, so we distinguished between: (1) abbreviated relaxation therapy; (2) full, traditional relaxation therapy; and (3) expanded relaxation therapy (including explicit cognitive treatment).

Our research questions were in patients with myocardial ischaemia:

(1) Does relaxation training, given as an adjunct to usual care, improve outcome better than usual care, with or without exercise rehabilitation?
(2) Does relaxation training have any long-term beneficial effect on cardiac outcome?
(3) Does the effect of relaxation therapy vary with the extent and intensity of training?

Methods

A search was performed of Medline, Embase, Cinahl, PsychInfo and the Cochrane Register of Controlled Trials in February 2005, using the keywords: relaxation techniques, meditation, stress management, autogenic training or biofeedback combined with heart or coronary diseases.

In addition, the first author's own files were hand-searched, expert colleagues in the field were contacted, and reference lists in retrieved papers were searched.
For inclusion, studies had to be controlled trials, and any control intervention including usual care was permitted. Trials were included if their participants had myocardial ischaemia in one of its various forms of presentation. Congestive cardiac failure and chronic stable angina pectoris were included, but hypertension without clinical ischaemia was excluded. The intervention had to involve learning and applying relaxation therapy as the primary intervention. Interventions such as massage or listening to music were therefore excluded, even though they may have relaxing effects. Studies that taught relaxation as a secondary and minor component of a more complex intervention were excluded as were studies using biofeedback alone without relaxation therapy. The outcome(s) measured had to be clinical and relevant to recovery from myocardial ischaemia or the prevention of future events, in at least one of four categories of future events, in at least one of four categories (physiological, psychological, cardiac or fitness for work). Studies whose objective was to test the short-term effects of relaxation for anxiety secondary to hospital admission (e.g. for MI or cardiac surgery) or to procedures such as catheterization, were excluded, as not containing the notion of recovery from ischaemia. Studies that tested the effect of relaxation on risk factors but not directly on cardiac outcome measures were excluded. The decision to include each study was made by both authors independently, with one disagreement (as a result of interpretation) settled by discussion.

Data extraction

Data were extracted using purpose-designed data extraction sheets (see Table 1). When data were uninterpretable or not in suitable form for combining, this was noted. When data were missing, an attempt was made to contact the report's first author in the case of studies published in the past 5 years. In studies that compared relaxation therapy with more than one control group, the control that only differed

in respect of relaxation therapy was chosen, to measure the effect of the addition of relaxation therapy. The intensity of relaxation therapy was graded into three grades (see Introduction) based primarily on the time for practice and the content of the cognitive restructuring. All grades of relaxation training contain an important cognitive component, consisting of at least a rationale for relaxation practice and an explanation of the effects of stress. Grade 1 is abbreviated relaxation training. It consists of teaching a basic relaxation skill using at least one form of instruction with or without biofeedback, which takes approximately 3 hr or less of group instruction, or 2 hr or less of individual instruction. Grade 2 is full, traditional relaxation training, with longer training, practice of extended skills, and discussion. Grade 3 is expanded relaxation training. It involved grade 2 teaching of relaxation, together with teaching of other coping skills, e.g. for dealing with depression, hostility or anger. The differentiation between grades 1 and x is thus based upon the hours of supervised practice, the differentiation between grades 2 and 3 is based upon the cognitive content and coping skills. Interventions that taught other coping skills, but did not provide a longer time for practice of relaxation skills, were classified as grade 1.

All data extraction was performed by both authors independently and disagreements (e.g. on choice of outcome) were settled by discussion.

Outcomes

We extracted data on the following variables: (1) physiological, i.e. resting pulse and blood pressure (systolic and diastolic), maximum watts, heart rate variability (HRV), serum cholesterol and high-density lipoprotein cholesterol (HDL); (2) psychological, i.e. depression and anxiety; (3) cardiac, i.e. frequency of angina attacks, occurrence of arrhythmia and ischaemia, and cardiac events (defined as cardiac death, reinfarction, coronary artery bypass graft or percutaneous transluminal coronary angioplasty) over various periods after learning relaxation; (4) function, i.e. return to work (either partial or whole return to work, when the patient had worked before the illness). We selected the longest common follow-up time for each variable, with interim timepoints in some cases, e.g. for cardiac events.

Quality assessment

The quality of the study reports was assessed in terms of significant threats to internal validity [11]. This was assessed by awarding two points for true randomisation,one point for quasi-randomization (used, e.g. to prevent contamination between groups), one point if the assessor was blind to the patient's allocation, and one point for the description of dropouts. The maximum score for quality was four points (modified from Jadad et al., 1996). Studies were grouped arbitrarily according to risk of bias: low risk (3 or 4 points), intermediate risk (2 points) and high risk (0 or 1 points). Data on quality and quantitative data on outcomes were extracted by both authors independently, with disagreements resolved by discussion.

Data synthesis

For continuous outcomes measured by different instruments, standardized difference in means was calculated as the difference in the mean responses divided by the pooled standard deviation. The effect size was expressed in a statistical form, in which we considered an effect size less than 0.20 as no effect, effect size of 0.21–0.49 as a small effect, 0.50–0.79 as medium effect, and effect size 0.80 or greater as a large effect [12]. When more than one outcome measure was used in a study for the same construct, preference was given to those measures most similar to the symptom check list for depression, STAI state for anxiety, and peak treadmill time for maximum watts. For continuous outcomes in the same units (e.g. pulse, blood pressure) the weighted mean difference (WMD) was calculated and the effect was expressed in those units. For dichotomous outcomes such as the cardiac events and return to work, two-by-two tables were set up and the odds ratios and 95% confidence intervals (CI) were analysed. Data were entered into 'RevMan 4.2', and 'Metaview' was used for the analysis (RevMan 4.1; Update Software Ltd., Oxford, UK). Statistical homogeneity was tested and fixed effects were used if $P > 0.10$, otherwise random effects modelling was used and an explanation for heterogeneity between results was sought in clinical terms.

After initially combining all studies, sensitivity analyses were performed when appropriate to test for the effect of internal validity of the study (risk of bias) and marked clinical heterogeneity. The latter was described in terms of trial setting, patient samples, content of control treatment (with or without

exercise), and was used qualitatively to interpret the result of each individual meta-analysis. Predefined subgroup analyses were performed to investigate the effect of the grade of relaxation. The results of the meta-analyses were then interpreted in the light of those studies that could not be combined, either because data were not presented in a suitable form (e.g. absent SD, or test statistic only), or because the control group received a different and potentially effective intervention. The latter studies act to the disadvantage of establishing an effect of relaxation therapy. They were nevertheless included except for variables that could be influenced by the intervention: exercise and cardiac data for exercise training and lifestyle data (smoking, cholesterol and sedentary life-style) for health education. Funnel plots were constructed using the inbuilt software and were evaluated by inspection.

Results

The searches located 27 studies that met our criteria [7,13–38]. The essential data are set out in Table 1. Thirty-four potentially relevant articles were excluded for using mixed interventions, not using relaxation therapy meeting our definition, not having outcomes relevant to our questions, or not being allocated appropriately. We obtained additional data from other published reports for Blumenthal et al. [39], van Dixhoorn and Duivenvoorden [8] and van Dixhoorn [40], and directly from the following authors: Blumenthal et al. [39] (data on anxiety and depression, 5-year follow-up); Wilk and Turkoski [35] (group sizes, SD) and van Dixhoorn [40] (2-year follow-up data). One study [19] comprises four arms that we analysed as two separate comparisons (stress management versus waiting list, and stress management with exercise against exercise alone).

Table 1 Overview of studies, by grade of relaxation and date of publication

	Patients			Intervention				Comparison therapy	Sample size		Design	
Author, year	Condition	Age, years (range/mean)	Recruitment	Name of treatment (components)	Group (h)	Individual (h)	Duration (weeks)		Ne	Nc	Allocation	Qual (0–4)
Brief relaxation training												
Hase and Douglas, 1987 [24]	MI	41–69	Early	Relaxation training (pmr + tape)	0	2	1–2	Usual	15	15	BI	2
Munro *et al.*, 1988 [27]	MI	52	Late	Relaxation therapy (rr + tape)	0.5	2	12	Exer	27	30	0	1
Amarosa-Tupler *et al.*, 1989 [13]	AP	60	Late	Stress management (pmr + tape)	0	0.5	4	Usual + educ	10	10	Ran	3
Gallacher *et al.*, 1997 [23]	AP	NS	NS	Stress management (NS + tape, cr)	3	0	10	Usual	198	209	Ran	3
Collins and Rice, 1997 [20]	MI, HS	59	Early	Relaxation intervention (pmr + tape)	0	1	6	Exer	20	23	BI	2
Wilk and Turkoski, 2001 [35]	MI, HS, PT	52–73	Early	Progressive muscle relaxation (pmr + tape)	3.3	0	4	Exer	7	7	Ran	2
Full relaxation training												
Kavanagh *et al.*, 1970 [7]	MI	<65	Late	Hypnosis (db, hyp)	50	3	52	Usual + exer	8	18	Ran (part)	2
Polackova *et al.*, 1992 [30]	MI	48	Early	Autogenic training (at)	16	1	16	Usual	131	48	0	0
Bohachick, 1984 [17]	MI, HS	NS	NS	Relaxation training (pmr)	9	0	2	Exer	18	19	0	0
Baer *et al.*, 1985 [15]	MI	55	Early	Stress management (pmr, db, cue)	8	1	1.14	Usual	33	37	BI	2
Ohm, 1987 [29]	MI, HS, AP	56	Late	Relaxation training (at, pmr, cue)	9	0	6	Exer	234	186	BI	1
Van Dixhoorn *et al.*, 1990, 1998 [40,41]	MI	55	Early	Relaxation therapy (pmr, move, br, cue, emg-bfb)	0	6	6	Exer	76	80	Ran	3
Winterfeld *et al.* 1991 [36]	HS	54	NS	Konzentrative entspannung (move, br, mpr)	9	0	9	Usual	20	14	Ran	2
Winterfeld *et al.*, 1993 [37]	HS	53	NS	Autogenic training (at)	14	0	14	Usual	16	14	0	0
Nelson *et al.*, 1994 [28]	MI	57	Early	Stress management (pmr, move, db, cue)	8	1	1.4	Usual + educ	16	19	BI	2
Zamarra *et al.*, 1996 [38]	MI, AP, IS	55	Late	Transcendental meditation (tm)	5	5	28	Usual	10	6	0	2
Luskin *et al.*, 2002 [26]	CHF	66	Late	Stress management (hrv-bfb)	0	10	10	Usual	14	5	Ran (part)	2
Kanji *et al.*, 2004 [25]	CA	63	Early	Autogenic training (at)	8	0	8	Usual	30	29	Ran	3
Del Pozo *et al.*, 2004 [22]	MI, HS, PT	67	Late	Biofeedback (hrv-bfb, db)	0	4.5	6	Usual	31	32	Ran	4
Extended relaxation												
Valliant and Leith, 1986 [33]	HS	38–72	NS	Relaxation training (pmr + tape; cr)	12	0	6	Usual	19	26	0	0
Bundy *et al.*, 1994 [18]	AP	54	Late	Psychological treatment (db, pmr, cue, tape; anger)	10.5	0	7	Usual	14	15	Ran	2
Turner *et al.*, 1995 [32]	MI, HS	59	Late	Stress management (at; type A, anger)	14	0	1.14	Exer	18	6	Ran	3
Trzcieniecka-Green and Steptoe, 1996 [31]	MI, HS	60	Early	Stress management (at, cue, tape; irritation, time urgency)	10	0	10	Usual	50	50	Ran	2
Blumenthal *et al.*, 1997 [16]	MI, HS, AP	60	Late	Stress management (pmr, emg-bfb; cr)	24	2	16	Usual	32	38	0	2
Appels *et al.*, 1997 [14]	PT	55	Early	Psychological intervention (br, rr, tape; hostility)	16	0	8	Usual	30	65	0	1
Bundy *et al.*, 1998 [19]	AP	56	Late	Stress management (db, pmr, cue, tape; anger)	10.5	0	7	Usual	42	16	BI	2
Cowan *et al.*, 2001 [21]	CA	NS	Late	Psychosocial nursing therapy (db, at, pmr, cue, hrv-bfb; depression, anger)	0	15	6	Educ	67	66	Ran	2

Patients condition: AP, angina pectoris; CA, cardiac arrest; CHF, congestive heart failure; HS, heart surgery; MI, myocardial infarction; PT, percutaneous transluminal coronary angioplasty. Recruitment: early, 2 months or less from index diagnosis; late, more than 2 months; NS, not stated. Intervention: Treatment components: at, autogenic training; bfb, biofeedback; br, breath relaxation; cr, cognitive restructuring procedure, cue, cue-controlled relaxation; db, deep breathing; emg, electromyographic; hrv, heart rate variability; hyp, hypnosis; move, movement, different postures; pmr, progressive muscle relaxation; rr, relaxation response. Dur, Duration of treatment; Group, training hours in group; Indiv, training hours in individual sessions. Control: educ, health education; exer, exercise training; usual, usual care; usual + educ or exer, a potentially active treatment in control group. Sample size: Nc, number of patients in control group; Ne, number of patients in experimental group. Design: Allocation: BI, in blocks; 0, no details given; Ran, randomized. Qual, quality of study by modified Jadad score, see text for full details.

Description of included studies

Patients

Relaxation therapy was taught to inpatients of a hospital in four studies [15,24,25,28], and a rehabilitation clinic in one study [29]. The remaining studies involved out-patients. Most studies included both men and women, usually with a preponderance of men; however, two studies [16,27] included only men. The mean age of patients was between 50 and 70 years for most studies, although it was only 48 years in one [30]. Patients were recruited after MI in most studies (n = 15), and patients after cardiac surgery or percutaneous transluminal coronary angioplasty were included in 12; three studies included only heart surgery patients, four studies included patients with angina only, and one study included patients with congestive heart failure. One study [21] was exceptional in recruiting only patients who had experienced sudden cardiac arrest.

Ten studies [14,15,20,24,25,28,30,31,34,35] recruited patients during the rehabilitation phase directly after the cardiac event. The time of recruitment was not reported for five studies [17,23,33,36,37], and the remaining studies recruited patients more than 3 months after the qualifying event, indicating a more chronic and stable condition.

Intervention
Six studies taught patients brief relaxation (grade 1) [13,20,23,24,27,35]. In 13 studies patients were taught full, traditional relaxation, mostly including multiple techniques (grade 2) [7,15,17,22,25,26,28–30,34,36–38]. All but three [22,26,34] used a group format, and in five group instruction was complemented by individual sessions. The group hours ranged from 5 to 16 h, with a median of 9 h, but one study [7] used 50 weekly sessions. Eight studies expanded relaxation training by teaching cognitive skills [14,16,18,19,21,31–33] typically in regular weekly meetings in small groups of six to eight participants led by a psychologist (grade 3). The time spent ranged between 10 and 24 h, with a median of 11.25 h.

Control intervention
The majority of studies compared relaxation with usual care, but in seven studies [17,20,27,29,32,34,35] the control condition included exercise rehabilitation. Three studies compared relaxation with a possibly active intervention, such as education [13,28] or exercise [7].

Internal validity
The internal validity (see Table 1) was likely to be high (score 3 or 4) in six studies [13,22,23,25,32,34] moderate (score 2) in 12 studies [7,15,19–21,24,26,28,31,35,36,38] and low (score 0 or 1) in nine [14,16–18,27,29,30,33,37]. Nine studies did not report allocating all patients either by randomization or by sequential blocks [14,16, 17,26,27,30,33,37,38].

Outcomes
Resting heart rate
A combined analysis of seven studies [16,20,25,26, 35,37,34] (Table 2) showed a small but clear effect of a weighted mean reduction of approximately 4 bpm (95% CI 1.2–6.4 bpm). The studies are homogeneous, the funnel plot shows no asymmetry, and the difference between groups is still significant when only the four studies with moderate or good validity [20,25,34,35] are combined. Interestingly, in three of these studies both arms included exercise rehabilitation, which is known to reduce heart rate, but a separate effect of relaxation was still seen. Four studies that measured heart rate could not be included in the meta-analysis: three lacked complete data, of which two [13,36] found a positive effect. One report [7] was excluded from this analysis because the control intervention included exercise training: after 1 year of treatment both arms had similar heart rate reductions. Subgroup analysis showed that relaxation therapy grade 1 (two studies, 54 patients) was more effective than grades 2 or 3, although with overlapping confidence intervals.

Blood pressure
Ten studies [16,20,23,25–27,32,37,34,35] provide no evidence that relaxation therapy has any effect on systolic blood pressure. The one study that was positive [37] has low internal validity (no randomization or blinding) and included only 30 patients. With three studies that have high internal validity and four that are randomized, the lack of effect is robust. The four relaxation therapy grade 1 studies result in a small rise in systolic blood pressure, although statistically not significant (4.5, CI - 5.6, 14.7), whereas relaxation therapy grades 2 and 3 yield a small, non-significant decrease in systolic blood pressure (– 4.1, CI – 8.5, 0.03).
Similarly, there is no overall effect on diastolic blood pressure. Heterogeneity is largely explained by the two studies using grade 1 relaxation therapy, but sensitivity analysis excluding them still shows no effect (– 0.86, CI – 3.0, 1.3, P = 0.4). No differential effect of grades of relaxation therapy is seen.

Exercise tolerance
Two studies [19,34] were excluded from this analysis as their control groups underwent exercise training. The remaining four studies [16,18,26,38] show a modest effect of relaxation therapy, and are homogeneous in their results and of moderate risk of bias. Two studies [18,19] (the second of which could not be combined) showed a significant difference in favour of relaxation therapy post- treatment,

which had disappeared by 8 weeks follow-up. Another study [7] found the increase in mean predicted oxygen consumption to be the same with relaxation therapy as with exercise training when both were continued for 1 year. This supports the result that relaxation therapy can improve exercise tolerance, in the absence of exercise training.

Heart rate variability
Three studies [22,26,40] showed a significant effect of relaxation therapy on HRV, standardized mean difference (SMD) = 0.35 (CI 0.04, 0.65, P < 0.03), which increased at 3-month follow-up in two studies [22,40], SMD = 0.58 (CI 0.24, 0.93, P < 0.0005). All three used relaxation therapy grade 2 and were of moderate or low risk of bias.

Serum lipids
Three studies [16,23,32] found no effect of relaxation therapy on total cholesterol, WMD = 0.08 (CI 0.22, 0.06; ns). A significant increase in HDL was seen, WMD = 0.06 (CI 0.01, 0.10, P < 0.001), although too small to have any useful clinical impact. The studies were homogeneous, and there was no differential effect of grade of relaxation therapy.

Depression
Nine studies [14,16,17,20,23,26,30,31,33] show a moderate overall effect on depression, but the result is heterogeneous (P < 0.00001) as a result of two studies with clearly divergent, strongly positive, results [14,30]. Both have low internal validity. Excluding these removes both the heterogeneity and the effect (SMD – 0.14, CI – 0.30, 0.03; ns). Only one study has high validity [23], only two were randomized [23,31], and there two studies, with more than half the total patients (n = 478) showed no effect (0.10, 95% CI 0.32, – 0.12). Relaxation therapy grade showed no consistent differential effect.

Anxiety
State anxiety is the outcome most commonly measured in these studies, and 13 [15–17,20,24–26,30,31,33,35,41] together show that relaxation therapy was highly significantly superior to controls, with no heterogeneity or evidence of major publication bias from the funnel plot. Eight studies have high or intermediate validity, and combining these still yields a positive result (SMD – 0.28, CI – 0.47, – 0.10, P = 0.003). Selecting four studies that compared relaxation therapy in addition to exercise rehabilitation did not show a smaller effect (– 0.31, CI – 0.57, – 0.05; P = 0.02). Subgroup analysis according to grade of relaxation therapy showed a smaller effect of grade 1, (– 0.08, CI – 0.27, 0.09) than grade 2 (– 0.51, CI – 0.71, - 0.31), but not different from grade 3 (– 0.24, CI – 0.51, 0.04). One study whose data could not be included [18] found no effect on anxiety.
No effect on relaxation therapy was seen in trait anxiety in four homogeneous studies with low and moderate validity. No greater effect was seen with more intensive grades of relaxation therapy

Frequency of angina pectoris at rest
Four studies [18,19,23,31] (of low or intermediate risk of bias) showed a statistically significant effect of relaxation therapy on angina pectoris. Statistical heterogeneity was caused by the study with the largest effect [31] in which patients were recruited after MI. After removing this study, the effect in three studies of moderate validity in angina pectoris patients is still significant (– 0.34, CI – 0.53, – 0.15, P = 0.0003). The results of four studies that could not be combined [13,15,24,28] are uniformly positive. There was a clearly greater effect in four studies that used grade 3 relaxation therapy (– 0.79, CI – 0.07, – 0.51) than the single study using grade 1 (– 0.26, CI – 0.48, – 0.05).
Incidence of cardiac arrhythmia

The incidence of any kind of arrhythmia was reported after being extracted from medical records at discharge [24] or at 6-month follow-up [15,28]. Relaxation therapy has a significant effect, with no heterogeneity and moderate risk of bias.
Myocardial ischemia
Four studies in 255 patients measured myocardial ischaemia, but the data are too varied to be pooled. Significant differences in favour of relaxation therapy were found in wall motion abnormalities during radio-nuclide angiography [16], and reduced ST-depression during exercise testing [34,38]. One study

[7] found the effect of relaxation therapy on ST-depression similar to that of exercise testing.

Cardiac events
Cardiac events in the first 6 months were significantly reduced in three studies [15,28,29], and the effect does not appear to diminish with time, being sustained for 1–3 years in two studies [14,21] and 3–5 years in another two [8,39]. The effect is also seen in the two studies [8,29] that compared relaxation therapy with exercise rehabilitation (odds ratio 0.54, CI 0.31, 0.94, P = 0.03), which is known to reduce cardiac events. Cardiac deaths reported in four studies [8,21,28,29] for periods from 6 months to 2 years show a clear beneficial effect on relaxation therapy, although the evidence is of mixed quality. One study [39] that could not be combined showed no effect in 67 patients.

Return to work
Three homogeneous studies [28,29,34] (with validity scores 1, 2 and 3) show that the odds of being at work at 6 months after MI or coronary artery bypass graft is significantly increased by relaxation therapy.

Discussion
The main finding of this review is that the effects of relaxation therapy are manifold any extend well beyond teaching relaxation skills. Relaxation therapy can enhance recovery after a cardiac ischaemic event and encompasses all domains of rehabilitation. The clinically most relevant effects include a reduction in resting heart rate, a reduction in anxiety and in the frequency of angina pectoris, increased return to work and reduced risk of death. Most effects are complementary to exercise rehabilitation; that is, they were also present in studies in with the control treatment included exercise training. Relaxation therapy is therefore effective as an adjunct to medical care as well as standard cardiac rehabilitation. This confirms two previous reviews that established the overall contribution of stress management and psycho-education for cardiac patients [1,2], but not the specific contribution of relaxation therapy. In this review we focused on the role of relaxation therapy, and we conclude that intensive, supervised relaxation practice is an important ingredient of cardiac rehabilitation.

More specifically, relaxation therapy appears to be complementary to psycho-education, an intervention that provides patient information on risk factors and promotes healthy behaviour. Usually, it offers little skills training and no practice in the self-regulation of tension as in relaxation therapy. Psycho-education reduced risk factors such as blood pressure, cholesterol and smoking but hardly reduced anxiety, depression or angina pectoris [2]. Relaxation therapy by contrast had no benefit for these risk factors, but was beneficial for the emotional and physical state of the patient. There is thus evidence for an effect of relaxation therapy that is additional to psycho-education. This was confirmed explicitly in three studies [13,21,28], in which training in relaxation skills enhanced the effects of psycho-education.
The relationship of stress management and relaxation therapy is more complex and depends upon definition. Traditionally, stress management programmes are based upon relaxation training, and full relaxation training was seen as a form of stress management [9], because it helps individuals to deal with stress and tension. The classical relaxation methods aim to reduce the strain in the individual, which was seen to have accumulated in the course of time and exposures to stressors. In progressive muscle relaxation it is neuromuscular hypertension or elevated muscle tension [42]. In autogenic training the individual is helped to restore a disturbed psycho-vegetative balance [43]. Once these primary skills are learned, they are applied in daily life and the issue of stressors and how to deal with them arises. These were typically topics for the second part of full relaxation training in the studies reviewed. In modern stress management programmes, however, the main focus is the perception, appraisal and consequent coping with stressors. This cognitive–behavioural framework overlaps to a large extent with full relaxation training, which can be seen as a cognitive-behavioural treatment [44]. An important difference is the role of primary relaxation skills. Linden and colleagues [1] reviewed 23 studies with a wide array of psychological treatments, which were summarized as stress management, but only four of which contained relaxation therapy. They found an effect size for the reduction of psychological distress (anxiety and depression) and of heart rate, both d = 0.30, which equals the effect sizes of relaxation therapy found in this review. Psychological treatment, however, also greatly reduced cholesterol (d = – 0.95) and to a lesser extent blood pressure (d = – 0.14), which we could not confirm. Relaxation therapy thus does not influence risk factors like psychological treatment does, but equals the effect of psychological treatment or stress management on heart rate and anxiety.

We tried to differentiate the effects of relaxation training per se from relaxation training expanded with cognitive therapy. Unexpectedly, we found no evidence for the superiority of expanded treatment in cardiac patients. Similarly, in the treatment of general anxiety disorder, no differences were found in effectiveness between cognitive therapy and applied relaxation [45]. However, this is based on comparing different studies and it needs to tested experimentally by contrasting full and expanded relaxation therapy in a single trial.
Our findings may be due to the fact that full relaxation therapy contains sufficient cognitive themes that are relevant to cardiac patients. The impact of general themes such as teaching respect for rest, the need for balance between rest and effort, the influence of mental factors on physical function, and the differentiation of cardiac causes of chest pain from stress, which are part and parcel of full relaxation therapy, may themselves be meaningful enough for most patients. Other cognitive themes such as dealing with anger, depression and time urgency may be too specific or not relevant to all participants. Another possibility may be that the practice of primary relaxation skills has a value of its own, which could have been less present in expanded relaxation therapy. Although we selected grade 3 type of studies with clear evidence of sufficient relaxation practice, the exact duration of practice was rarely specified and may have been less than in the grade 2 type of study. Therefore, the cognitive part may not have come on top but partly also instead of relaxation practice, thereby reducing its specific effects.

It needs to be emphasized that the potential equality of relaxation therapy and stress management only applies when relaxation is not taught as a simple technique in its abbreviated form. We made a point of classifying studies by the duration of relaxation training (as mastery was rarely reported), and found a clear distinction between the shortened version of relaxation therapy taught in a maximum of 3 hr, and the full training format in which sample supervised relaxation practice is provided. The latter form took on average 9 hr. With the exception of heart rate reduction, the effects were smaller for the abbreviated format. The effects on arrhythmia and ischaemia, as well as improved return to work and prognosis have almost exclusively been studied in full or expanded relaxation therapy.

An important limitation is that the quality of many included studies has not high. Only 10 out or 24 studies were randomized. Dusseldorp and colleagues [2] made a similar reservation, and stated that many studies use a quasi-experimental approach with different treatment assignment in different time periods, instead of random assignment, to prevent patients in the conditions interacting with each other. However, they found no systematic influence of the quality of studies on outcome. Similarly, we checked the influence of the internal validity of the studies. Only in the case of depression was the influence marked. Another limitation is the size of the studies. Many were small scale and set up as pilot studies. Only six studies recruited 100 or more patients. Nevertheless, the total number of patients added up to substantial sample sizes for the outcomes and the conclusions were statistically highly significant. However, it is important to draw only general conclusions and to be cautious with respect to effect size.
This reservation applies specifically to the effects on prognosis. The odds for future cardiac events are remarkably low after relaxation therapy. Although the total number of patients approached 1000, it is small in comparison with trials in cardiology, and insufficient to allow a realistic estimate of probable effect size. The odds ratios for cardiac events of 0.39 and for cardiac death of 0.29 are extremely low in comparison with the reviews of psychosocial treatment, as well as with the effects of cardiac rehabilitation or beta-blockers [46,47], which are approximately 0.80. Another reason for caution is that we have no information on the mechanism for prognostic benefit. Interestingly, the finding of Dusseldorp et al. [2] that the long-term effect is greater in studies that were successful in reducing somatic risk factors in the short term was not corroborated, as we found very little effect on these. Nevertheless, the result does underlive the role of stress in prognosis, and even more, that an increased ability for internal self-regulation of tension and stress is somehow beneficial for health. For instance, it may modify the effect of risk factors. A recent study showed that the effect of ventricular arrhythmia on prognosis was absent in those with good ability for tension regulation [48]. We need new studies not only to replicate the result and estimate effect size more accurately, but also to obtain information or the possible pathways of such an effect. It will be important to measure all the outcome variables and inter-relate them in the short term, as well as using them as predictors for long-term outcome. This review may be the starting point and rationale for such studies.
As far as implications for practice are concerned, it is of course possible to add abbreviated relaxation

therapy to an exercise or lifestyle programme within cardiac rehabilitation, but the studies reviewed here allow very little success to be expected. If one wants to implement relaxation therapy, better do it well. This implies intensive supervised training in primary relaxation skills, in a separate programme, either in small groups or individually. This was done in the recent Dutch Guidelines for Cardiac Rehabilitation (2004) [49]. An independent relaxation programme was described and a multimodal approach was chosen, which requires approximately 9 h of supervised practice. Five studies in grade 2 used a multimodal approach and required or average 8 h (median 9, see Table 1). A unimodal approach such as for instance autogenic training requires extended practice.

Table 2 Results of meta-analyses of effects of relaxation therapy on various outcomes

Outcome	Studies	Patients	Analysis	Result (95% CI)	*P* value
Heart rate	7	381	WMD f	−3.83 (−6.43, −1.24)	0.01
Systolic BP	10	773	WMD r	−0.42 (−4.68, 3.84)	0.85
Diastolic BP	9	744	WMD r	−0.13 (−3.01, 2.75)	0.93
Exercise test	4	173	SMD f	0.44 (0.12, 0.75)	0.007
HRV	3	168	SMD r	0.35 (0.0, 0.65)	0.03
Cholesterol	3	527	WMD f	−0.08 (−0.22, 0.06)	0.3
HDL	3	527	WMD f	0.06 (0.01, 0.10)	0.008
Depression	9	957	SMD r	−0.48 (−0.88, −0.09)	0.02
State anxiety	13	1185	SMD r	−0.35 (−0.51, −0.18)	0.0001
Trait Anxiety	5	285	SMD r	−0.17 (−0.40, 0.06)	0.15
Angina pectoris	4	565	SMD r	−0.60 (−0.96, −0.23)	0.001
Arrhythmia	3	135	OR f	0.22 (0.10, 0.49)	0.0002
Cardiac events (<1 year)	3	475	OR f	0.44 (0.23, 0.83)	0.01
Cardiac events, total	7	916	OR f	0.39 (0.27, 0.57)	0.001
Cardiac deaths (2 years)	4	694	OR f	0.29 (0.12, 0.70)	0.006
Return to work	3	376	OR f	1.83 (1.18, 2.81)	0.006

BP, Blood pressure; CI, confidence interval; f, fixed effects; HDL, high-density lipoprotein cholesterol; HRV, heart rate variability; OR, odds ratio; r, random effects; SMD, standardized mean difference; WMD, weighted mean difference.

The five studies that used a cognitive unimodal relaxation format took on average 19.5 h of practice (median 14). It is thus recommended to offer techniques for attention control, for muscle relaxation, for breath regulation and for posture and small movements. Furthermore, it is recommended to offer this wide array of technique modalities any have each patient find out which modality suits best and produces the most concrete changes in tension. It is thus essential to assess mastery. Few studies provided information on whether patients actually learned the primary skills of relaxation. It is not easy to establish, because there is no single measurement for relaxation success, but effort should be made to estimate the degree of mastery.

For implementation it is important to ensure adequate training of the teaching staff and therapists. There is a lack of information on this aspect is the studies reviewed, but this is crucial to implementation. Possibly, studies have been undertaken mostly by individual researchers who acquired expertise and experience in relaxation skills by personal preference. As the ability to conduct full relaxation therapy is not provided in any healthcare professional education, the development of treatment protocols and staff training requires careful planning and preparation.

To conclude, this review shows evidence that supports the utility of supervised relaxation practice as a treatment per se and warrants the inclusion of full relaxation therapy in cardiac rehabilitation, because it enhances recovery from an ischaemic event and it contributes to secondary prevention, independently of the effect of psycho-education any of exercise.

References

- Linden W, Stossel C, Maurice J.:Psychosocial interventions for patients with coronary
- artery disease: a meta-analysis. Arch Intern Med 1996; 156:745–752.
- Dusseldorp E, van Elderen T, Maes S, Meulman J, Kraaij V.: A Meta-analysis of psychoeduational programs for coronary heart disease patients. Health Psychol 1999; 18:506–519.
- Wenger NK, Froelicher ES, Smith LK, Ades PA, Berra K, Blumenthal JK, et al.: cardiac rehabilitation. Clinical practice guidelines no. 17. Rockville, MD: US Department of Health and Human Survices, Public Health Service; 1995.
- Thompson DR, Bowman GS, Kitson AL, Bono DPD, Hopkins A.: Cardiac Rehabilitation in the United Kingdom: guidelines and audit standards. Heart 1996; 75:89–93.
- Ades PA.: Cardiac rehabilitation and secondary prevention of coronary heart disease. Freda 2001; 345:892–902.
- Ades PA, Balady GJ, Berra K:. Transforming exercise-based cardiac rehabilitation programs into secondary prevention centers: a national imperative. J Cardiac Rehabil 2001; 21:263–272.
- Kavanagh T, Shephard RJ, Pandit V, Doney H.: Exercise and hypnotherapy in The rehabilitation of the coronary patient. Arch Phys Med Rehabil 1970; 51:578–587.
- van Dixhoorn JJ, Duivenvoorden HJ.: Effect of relaxation therapy on cardiac events after myocardial infarction: a 5-year follow-up study. J Cardiopulm Rehabil 1999; 19:178–185.
- Lehrer PM, Woolfolk RL.: Principles and practice of stress management. New York: Guilford Press; 1993.
- Benson H, Beary JF, Carol MP.: The relaxation response. Psychiatry 1974; 37:37–45.
- Jadad AR, Moore RA, Carrol D, Jenkinson C, Reynolds DJ, Gavaghan D, et al.: Assessing the quality of reports of randomised clinical trials: is blinding necessary? Control Clin Trials 1996; 17:1–12
- Cohen J.: Statistical power analysis for behavioral sciences. New York: Academic Press; 1969.
- Amarosa-Tupler B, Tapp JT, Carida RV.: stress management through relaxation and imagery in the treatment of angina pectoris. J Cardiopulm Rehabil 1989; 9:348–355.
- Appels A, Bar F, Lasker J, Flamm U, Kop W.: The effect of a psychological intervention program on the risk of a new coronary event after angioplasty: a feasibility study. J Psychosom Res 1997; 43:209-217.
- Baer PE, Cleveland SE, Montero AC, Revel KF, Clancy C, Bowen R.: Improving post-myocardial infarction recovery status by stress management training during hospitalization. J Cardiac Rehabil 1985;5 :191–196.
- Blumenthal JA, Jiang W, Babyak MA, Krantz DS, Frid DJ, Coleman RE, et al.: Stress management and exercise training in cardiac patients with myocardial ischemia. Effects on prognosis and evaluation of mechanisms. Arch Intern Med 1997; 157:2213-2223.
- Bohachick P.: Progressive relaxation training in cardiac rehabilitation: effect on psychologic variables. Nurs Res 1984; 33:283–287.
- Bundy C, Carroll D, Wallace L, Nagel R.: Psychological treatment of chronic stable angina pectoris. Psychol Health 1994; 10:69–77.
- Bundy C, Carroll D, Wallace L, Nagle R.: Stress management and exercise training in chronic stable angina pectoris. Psychol Health 1998; 13: 147–155.
- Collins JA, Rice VH.: Effects of relaxation intervention in phase II cardiac rehabilitation: replication and extension. Heart Lung 1997; 26: 31–44.
- Cowan MJ, Pike KC, Budzynski HK.: Psychosocial nursing therapy following sudden cardiac arrest: impact on two-year survival. Nurs Res 2001; 50:68–76.
- Del Pozo JM, Gevirtz RN, Scher B, Guarneri E.: Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. Am Heart J 2004; 147:E11, G1–G6
- Gallacher JEJ, Hopkinson CA, Bennett P, Burr ML, Elwood PC.: Effect of stress management on angina. Psychol Health 1997; 12:523–532.
- Hase S, Douglas A.: Effects of relaxation training on recovery from myocardial infarction. Aust J Adv Nurs 1987; 5:18–27.
- Kanji N, White AR, Ernst E.: Autogenic training reduces anxiety after coronary angioplasty: a randomized clinical trial. Am Heart J 2004; 147:E10, K1–K4
- Luskin F, Reitz M, Newell K, Quinn TH, Haskell W.: A controlled pilot study of stress management training of elderly patients with congestive heart failure. Prev Cardiol 2002; 5:168–172.

- Munro BH, Creamer AM, Haggerty MR, Cooper FS.: Effect of relaxation therapy on post-myocardial infarction patients' rehabilitation. Nurs Res 1988; 37:231–235.
- Nelson DV, Baer PE, Cleveland SE, Revel KF, Montero AC.: Six-month follow-up of stress management training versus education during hospitalization for acute myocardial infarction. J Cardiopulm Rehabil 1994; 14:384–390.
- Ohm D.: Relaxation training and hypnosis for patients with coronary heart disease in residential rehabilitation [in German]. Regensburg: Roderer Verlag; 1987.
- Polackova J, Bockova E, Sedivec V.: Autogenic training: application in secondary prevention of myocardial infarction. Activitas Nervosa Superior 1982; 24:178–180.
- Trzcieniecka-Green A, Steptoe A.: The effects of stress management on the quality of life of patients following acute myocardial infarction or coronary bypass surgery. Eur Heart J 1996; 17:1663-1670.
- Turner L, Linden W, van der WR, Schamberger W.: Stress management for patients with heart disease: a pilot study. Heart Lung 1995; 24: 145–153.
- Valliant PM, Leith B.: Impact of relaxation-training and cognitive-therapy on coronary patients post surgery. Psychol Rep 1986; 59:1271–1278.
- van Dixhoorn J, Duivenvoorden HJ, Staal HA, Pool J.: Physical training and relaxation therapy in cardiac rehabilitation assessed through a composite criterion for training outcome. Am Heart J 1989; 118:545–552.
- Wilk C, Turkoski B.: Progressive muscle relaxation in cardiac rehabilitation: a pilot study. Rehabil Nurs 2001; 26:238–242.
- Winterfeld H-J, Risch A, Siewert H, Strangfeld D, Kruse J, Engelmann U, et al.: The influence of concentrated relaxation on blood pressure and haemodynamics in hypertensive patients with coronary heart disease and coronary artery bypass grafting (CABG) [in German]. Z Physiother 1991; 43:220–224.
- Winterfeld HJ, Siewert H, Bohm J, Frenzel R, Aurisch R, Ecke A, et al.: Autogenic training in hypertentive dysregulation after aortocoronary venous bypass operation of coronary heart disease [in German]. Z Gesamte Inn Med 1993; 48:201–204.
- Zamarra JW, Schneider RH, Besseghini I, Robinson DK, Salerno JW.: Usefulness of the transcendental meditation program in the treatment of patients with coronary artery disease. Am J Cardiol 1996; 77:867–870.
- Blumenthal JA, Babyak M, Wei J, O'Connor C, Waugh R, Eisenstein E, et al.: Usefulness of psychosocial treatment of mental stress-induced myocardial rehabilitation after ischemia in men. Am J Cardiol 2002; 89:164–168.
- van Dixhoorn J.: Cardiorespiratory effects of breathing and relaxation instruction in myocardial infarction patients. Biol Psychol 1998; 49: 123–135.
- van Dixhoorn J, Duivenvoorden HJ, Pool J, Verhage F.: Psychic effects of physical training and relaxation therapy after myocardial infarction.J Psychosom Res 1990; 343:327–337.
- McGuigan FJ.: Progressive relaxation: origins, principles and clinical applications. In: Lehrer PM, Woolfolk RL (editors): Principles and practice of stress management. New York: Guilford Press; 1993, pp. 17–52.
- Linden W.: The autogenic training method of JH Schultz. In: Lehrer PM, Woolfolk RL (editors): Principles and practice of stress management. New York: The Guilford Press; 1993, pp. 205–229. Med 1993; 48:201–204.
- Smith JC.: Steps toward a cognitive–behavioral model of Biofeedback Self Regul 1988; 134:307–329.
- Fisher PL, Durham RC.: Recovery rates in generalized anxiety disorder following psychological therapy: an analysis of clinically significant change in the STAI-T across outcome studies since 1990. Psychol Med 1999; 29:1425–1434.
- Oldridge NB, Gyatt GH, Fischer ME, Rimm AA.: Cardiac rehabilitation after myocardial infarction. JAMA 1988; 2607:945–950.
- Lau J, Antman EM, Jimenez-Silva J, Kupelnick B, Mosteller F, Chalmers TC.: Cumulative meta-analysis of therapeutic trials for myocardial infarction. N Engl J Med 1992; 327:248–254.
- Andre-Peterson LA, Engstrom GA, Hedblad B, Janzon LA, Stee B, Tyden PAC.: Prognostic significance of ventricular arrhythmia modified by the ability to adapt to stressful situations. Eur J Cardiovasc Prev rehabil 2004; 11:25–32.
- Rehabilitation Committee of the Netherlands Society of Cardiology and The Netherlands Heart Foundation. Guidelines for cardiac Rehabilitation 2004. The Hague: Netherlands Heart Foundation, 2004.

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Initial Severity and Antidepressant Benefits: A Meta-Analysis of Data Submitted to the Food and Drug Administration

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Abstract:

Background

Meta-analyses of antidepressant medications have reported only modest benefits over placebo treatment, and when unpublished trial data are included, the benefit falls below accepted criteria for clinical significance. Yet, the efficacy of the antidepressants may also depend on the severity of initial depression scores. The purpose of this analysis is to establish the relation of baseline severity and antidepressant efficacy using a relevant dataset of published and unpublished clinical trials.

Methods and Findings

We obtained data on all clinical trials submitted to the US Food and Drug Administration (FDA) for the licensing of the four new-generation antidepressants for which full datasets were available. We then used meta-analytic techniques to assess linear and quadratic effects of initial severity on improvement scores for drug and placebo groups and on drug–placebo difference scores. Drug–placebo differences increased as a function of initial severity, rising from virtually no difference at moderate levels of initial depression to a relatively small difference for patients with very severe depression, reaching conventional criteria for clinical significance only for patients at the upper end of the very severely depressed category. Meta-regression analyses indicated that the relation of baseline severity and improvement was curvilinear in drug groups and showed a strong, negative linear component in placebo groups.

Conclusions

Drug–placebo differences in antidepressant efficacy increase as a function of baseline severity, but are relatively small even for severely depressed patients. The relationship between initial severity and antidepressant efficacy is attributable to decreased responsiveness to placebo among very severely depressed patients, rather than to increased responsiveness to medication.

Abbreviations: d, standardized mean difference; FDA, US Food and Drug Administration; HRSD, Hamilton Rating Scale of Depression; LOCF, last observation carried forward; NICE, National Institute for Clinical Excellence; SDc, standard deviation of the change score.

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Stress Affects a Gastrin-Releasing Peptide System in the Spinal Cord that Mediates Sexual Function: Implications for Psychogenic Erectile Dysfunction*

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Abstract:

Background

Many men suffering from stress, including post-traumatic stress disorder (PTSD), report sexual dysfunction, which is traditionally treated via psychological counseling. Recently, we identified a gastrin-releasing peptide (GRP) system in the lumbar spinal cord that is a primary mediator for male reproductive functions.

Methodology/Principal Findings

To ask whether an acute severe stress could alter the male specific GRP system, we used a single-prolonged stress (SPS), a putative rat model for PTSD in the present study. Exposure of SPS to male rats decreases both the local content and axonal distribution of GRP in the lower lumbar spinal cord and results in an attenuation of penile reflexes in vivo. Remarkably, pharmacological stimulation of GRP receptors restores penile reflexes in SPS-exposed males, and induces spontaneous ejaculation in a dose-dependent manner. Furthermore, although the level of plasma testosterone is normal 7 days after SPS exposure, we found a significant decrease in the expression of androgen receptor protein in this spinal center.

Conclusions/Significance:

We conclude that the spinal GRP system appears to be a stress-vulnerable center for male reproductive functions, which may provide new insight into a clinical target for the treatment of erectile dysfunction triggered by stress and psychiatric disorders.

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Effects of Abdominal Breathing Training using Biofeedback on Stress, Immune Response and Quality of Life in Patients with a Mastectomy for Breast Cancer

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Source: http://www.ncbi.nlm.nih.gov/pubmed/16418556

Purpose:
This study was to determine the effects of abdominal breathing training using biofeedback on stress, immune response, and quality of life. METHOD: The study design was a nonequivalent control group pretest- posttest, quasi-experimental design. Twenty-five breast cancer patients who had completed adjuvant chemotherapy were enrolled. The experimental group (n=12) was provided with abdominal breathing training using biofeedback once a week for 4 weeks. State anxiety, cancer physical symptoms, serum cortisol, T cell subsets (T3, T4, T8), NK cell and quality of life were measured both before and after the intervention.

Result:
Though state anxiety, cancer physical symptoms, and serum cortisol were reduced after 4 weeks of abdominal breathing training using biofeedback, there was no statistical significance. It showed, however, improvement in quality of life (p=.02), and T3 (p=.04).

Conclusion:
Abdominal breathing training using biofeedback improves quality of life in breast cancer patients after a mastectomy. However, the mechanism of this beneficial effect and stress response requires further investigation with special consideration in subject selection and frequency of measurement. Nurses should consider this strategy as a standard nursing intervention for people living with cancer.

Cardiovascular Abnormalities in Patients with Major Depressive Disorder: Autonomic Mechanisms and Implications for Treatment

AD Brown, D. Barton and G. Lambert
Centre for Indigenous Vascular and Diabetes Research, Alice Springs, Northern Territory, Australia.

Source: http://www.ncbi.nlm.nih.gov/sites/entrez

This article provides a detailed review of the association of major depression with coronary heart disease (CHD), examines the biological variables underpinning the linkage and discusses the clinical implications for treatment. When considering the co-morbidity between major depressive disorder (MDD) and CHD it is important to differentiate between (i) the prevalence and impact of MDD in those with existing CHD and (ii) MDD as a risk factor for the development of CHD. Whether the same biological mechanisms are at play in these two instances remains unknown. Depression is common in patients with CHD. Importantly, depression in these patients increases mortality. There is also consistent evidence that MDD is a risk factor for the development of CHD. The relative risk of developing CHD is proportional to the severity of depression and is independent of smoking, obesity, hypercholesterolaemia, diabetes mellitus and hypertension. There is a clear need to identify the underlying neurochemical mechanisms responsible for MDD and their linkage to the heart and vascular system. Of particular interest are activation of stress pathways, including both the sympathetic nervous system and hypothalamic-pituitary-adrenal axis, and inflammatory-mediated atherogenesis. Elevated sympathetic activity reduced heart rate variability and increased plasma cortisol levels have been documented in patients with MDD. In addition to direct effects on the heart and vasculature, activation of stress pathways may also be associated with increased

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release of inflammatory cytokines such as interleukin-6 and tumour necrosis factor-alpha. Elevated levels of C-reactive protein are commonly observed in patients with MDD. The majority of investigations examining treatment of depression following myocardial infarction have focused on safety and efficacy; there is little evidence to indicate that treating depression in these patients improves survival. Given that strategies for preventive therapy remain incompletely formulated, future research should focus on generating a better understanding of the neurobiology of MDD and heart disease as a basis for rational and effective therapy.

Amplitude-Modulated Electromagnetic Fields for the Treatment of Cancer: Discovery of Tumor-Specific Frequencies and Assessment of a novel Therapeutic Approach*

A. Barbault, F. Costa, B. Bottger, R. Munden, et al., 2009

Contact: alexandre.barbault@gmail.com
Source: http://www.ncbi.nlm.nih.gov/sites/entrez

Purpose:
Because in vitro studies suggest that low levels of electromagnetic fields may modify cancer cell growth, we hypothesized that systemic delivery of a combination of tumor-specific frequencies may have a therapeutic effect. We undertook this study to identify tumor-specific frequencies and test the feasibility of administering such frequencies to patients with advanced cancer.

Patients and methods:
We examined patients with various types of cancer using a non-invasive biofeedback method to identify tumor-specific frequencies. We offered compassionate treatment to some patients with advanced cancer and limited therapeutic options.

Results:
We examined a total of 163 patients with a diagnosis of cancer and identified a total of 1524 frequencies ranging from 0.1 Hz to 114 kHz. Most frequencies (57-92%) were specific for a single tumor type. Compassionate treatment with tumor-specific frequencies was offered to 28 patients. Three patients experienced grade 1 fatigue during or immediately after treatment. There were no NCI grade 2, 3 or 4 toxicities. Thirteen patients were evaluable for response. One patient with hormone-refractory breast cancer metastatic to the adrenal gland and bones had a complete response lasting 11 months. One patient with hormone-refractory breast cancer metastatic to liver and bones had a partial response lasting 13.5 months. Four patients had stable disease lasting for +34.1 months (thyroid cancer metastatic to lung), 5.1 months (non-small cell lung cancer), 4.1 months (pancreatic cancer metastatic to liver) and 4.0 months (leiomyosarcoma metastatic to liver).
Conclusion:
Cancer-related frequencies appear to be tumor-specific and treatment with tumor-specific frequencies is feasible, well tolerated and may have biological efficacy in patients with advanced cancer.

*In: Journal of clinical Cancer Research; 2009 Apr 14; 28:51

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Is Neurofeedback an Efficacious Treatment for ADHD?*

A randomised controlled clinical trial

H. Gevensleben, B. Holl, B. Albrecht, et al.
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Source:http://www.ncbi.nlm.nih.gov/pubmed/19207632?ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_DefaultReportPanel.Pubmed_RVDocSum

Background:
For children with attention deficit/hyperactivity disorder (ADHD), a reduction of inattention, impulsivity and hyperactivity by neurofeedback (NF) has been reported in several studies. But so far, unspecific training effects have not been adequately controlled for and/or studies do not provide sufficient statistical power. To overcome these methodological shortcomings we evaluated the clinical efficacy of neurofeedback in children with ADHD in a multisite randomised controlled study using a computerised attention skills training as a control condition.

Methods:
102 children with ADHD, aged 8 to 12 years, participated in the study. Children performed either 36 sessions of NF training or a computerised attention skills training within two blocks of about four weeks each (randomised group assignment). The combined NF treatment consisted of one block of theta/beta training and one block of slow cortical potential (SCP) training. Pre-training, intermediate and post-training assessment encompassed several behaviour rating scales (e.g., the German ADHD rating scale, FBB-HKS) completed by parents and teachers. Evaluation (‚placebo') scales were applied to control for parental expectations and satisfaction with the treatment.

Results:
For parent and teacher ratings, improvements in the NF group were superior to those of the control group. For the parent-rated FBB-HKS total score (primary outcome measure), the effect size was .60. Comparable effects were obtained for the two NF protocols (theta/beta training, SCP training). Parental attitude towards the treatment did not differ between NF and control group.

Conclusions:
Superiority of the combined NF training indicates clinical efficacy of NF in children with ADHD. Future studies should further address the specificity of effects and how to optimise the benefit of NF as treatment module for ADHD.

*In: Journal of Child Psychology and Psychiatry. 2009 Jul; 50 (7): 767-8

Multifractality and Heart Rate Variability

R. Sassi, M. Sirgnorini and S. Cerutti

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Source:http://www.ncbi.nlm.nih.gov/pubmed/19566282?ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_DefaultReportPanel.Pubmed_RVDocSum

In this paper, we participate to the discussion set forth by the editor of Chaos for the controversy, „Is the normal heart rate chaotic?" Our objective was to debate the question, „Is there some more appropriate term to characterize the heart rate variability (HRV) fluctuations?" We focused on the approximately 24 h RR series prepared for this topic and tried to verify with two different techniques, generalized structure

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functions and wavelet transform modulus maxima, if they might be described as being multifractal. For normal and congestive heart failure subjects, the h(q) exponents showed to be decreasing for increasing q with both methods, as it should be for multifractal signals. We then built 40 surrogate series to further verify such hypothesis. For most of the series (approximately 75%-80% of cases) multifractality stood the test of the surrogate data employed. On the other hand, series coming from patients in atrial fibrillation showed a small, if any, degree of multifractality. The population analyzed is too small for definite conclusions, but the study supports the use of multifractal series to model HRV. Also it suggests that the regulatory action of autonomous nervous system might play a role in the observed multifractality.

A Review of the Impact of Hypnosis, Relaxation, Guided Imagery and Individual Differences on Aspects of Immunity and Health

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Source: http://www.ncbi.nlm.nih.gov/sites/entrez

This review considers psychological interventions involving relaxation and guided imagery targeting immune functions. The review provides evidence of immune control accompanied by reports of enhanced mood and well-being. Three recent investigations of the author and his colleagues with self-hypnosis training incorporating imagery of the immune system are outlined. In two studies, hypnosis buffered the effects of stress on immune functions in medical students at exam time, and the comparison of self-hypnosis with and without immune imagery confirmed advantages to targeted imagery for both immune function and mood, and importantly, fewer winter viral infections. The implications for health were investigated in a third study in patients with virulent and chronic herpes simplex virus-2 HSV-2. Six weeks of training almost halved recurrence, improved mood and reduced levels of clinical depression and anxiety. Immune functions were up-regulated, notably functional natural killer cell activity to HSV-1. Individual differences in hypnotic susceptibility and absorption have typically been found to predict efficacy. New replicable evidence is reviewed of the importance of cognitive activation, a personality difference whose neurophysiological underpinning is consistent with left hemispheric preferential influences over the immune system. Now that the validation of psychological interventions includes advantages for health, this field of enquiry, which has been characterised by modest, small scale, largely preliminary studies, warrants a greater investment in research.

Stress Significantly Hastens Progression of Alzheimer's Disease

A summary from our editor C. Ruf

Source: University of California - Irvine (2006, August 30). Stress Significantly Hastens Progression Of Alzheimer's Disease. ScienceDaily. Retrieved August 12, 2009, from http://www.sciencedaily.com¬ /releases/2006/08/060830005837.htm

A neurobiology and behaviour researcher team of the University of California Irvine (Frank LaFerla, professor of neurobiology and behaviour, James L. McGraugh, research professor of neurobiology and behaviour, Kim Green, postdoctoral researcher in neurobiology and behaviour, Lauren Billings, postdoctoral researcher, and Benno Roozendaal, assistant researcher in neurobiology and behaviour) found that stress hormones hasten the formation of brain lesions which are responsible for Alzheimer's disease. Following this line of thought it is necessary to support the elderly to cope with stress adequately and to reduce certain medication that contains glucocorticoids in order to slow down the progress of Alzheimer's.

In this study, genetically modified mice were injected for seven days with a glucocorticoid (dexamethasone) similar to the body's stress hormones. In the following there were 60% more beta-amyloid proteins in

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the brain. When these protein fragments aggregate, they form plaques, which is one of the indicators of Alzheimer's. The level of another protein, tau, also swelled. This probably is hypothesized to the second indicator of Alzheimer's, which is the formation of tangles. The findings show how the hormones play a direct role in aggravating the underlying symptomatology of the disease in a very short period of time. The injection of dexamethasone to transgenic mice (13 month old) that already had some plaque and tangle pathology led to surprisingly strong effects namely to an aggravation of the plaque lesions and to increased accumulation of the tau protein. Four-month-old mice received an injection that enhanced their level as high as it would be seen in humans under stress. After one week, their level of beta-amyloid appeared to be as high as in untreated eight- to nine-month-old mice. This outcome emphasizes the profound consequence of glucocorticoid exposure.

The transgenic mice that were used in the present study were developed by Frank LaFerla, professor of neurobiology and behaviour, and his research team. Today, these mice are a model for studying Alzheimer's disease all around the world.

The higher levels of beta-amyloid and tau enhanced the levels of the stress hormones, which would come back to the brain and speed up the formation of more plaques and tangles. Hence, this process can be seen as a "feedback loop" that hastens the progression of Alzheimer's.
Consequently, these findings have profound implications for the treatment of Alzheimer's disease patients, concerning stress management and the reduction of medications that contain glucocorticoids.
Down to the present day, 4.5 million to 5 million adults in the United States are affected by the Alzheimer's disease, a progressive neurodegenerative disorder. It is estimated that 13 million Americans will be afflicted with the disease by 2050 if no therapies are generated
difference whose neurophysiological underpinning is consistent with left hemispheric preferential influences over the immune system. Now that the validation of psychological interventions includes advantages for health, this field of enquiry, which has been characterised by modest, small scale, largely preliminary studies, warrants a greater investment in research.

STRESSTIPS

Do the Opposite

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The 'principle of opposites', an ancient concept, has been described more fully by de Mora (1982), who wrote *The principle of opposites* in Sanskrit texts, as well as Mann (1972), who wrote Acupuncture, a cure of many diseases. Both texts included a sections referring to 'The principle of opposites' that suggest ways for increasing awareness of mental-and-physical/ mind-and-body processes. For example, de Mora (1982) details how individuals may learn about their senses by comparing pairs of opposites, called Dwandas in Sanskrit, such as hard and soft, bright and dim, or loud and quiet, as well as learn about relationships with others by comparing opposites such as gain and loss, pleasure and pain, or love and hate. Mann (1972) details how Chinese acupuncture philosophy includes the 'yin' and 'yang' principle of opposites that help people learn about things like powerful versus weak urges, feminine or masculine characteristics, or hot versus cold sensations in the body.

In the field of applied psychophysiology and biofeedback, a 'principle of opposites' may be observed in clinical settings when providing instruction in stress reduction techniques. For example, bringing awareness to breathing patterns or muscle tension in the body may be facilitated by first gradually tightening the muscles before releasing tension. Similarly, bringing awareness to poor breathing may be facilitated by first gradually exhaling before taking a deep breath.

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Whereas some people have difficulty building awareness of their muscle tension, or of shallow breathing, teaching a relaxation/

stress reduction behavior first by raising awareness in the opposite direction of the desired outcome may facilitate skill mastery. For example, by combining breathing and muscle relaxation techniques, a greater degree of stress reduction may be achieved.
Following is a variation of a relaxation/stress reduction technique that incorporates the 'principle of opposites' as part of the instruction. The technique, sometimes called the half-Jacobson, is a variation on Jacobson's (1938) progressive muscle relaxation technique. There are breathing and muscle relaxation components. Following the principle of opposites, the breathing component requires exhaling before inhaling.

1. Breathing: Begin breathing by exhaling for six seconds, then inhaling for four seconds. Continue practicing for several breaths by exhaling slightly longer (a count of six) before inhaling (a count of four).
2. Muscle Relaxation: After practicing exhaling slightly longer than inhaling, begin standing by shifting weight to the left side. Continue the breathing practice by exhaling slightly longer than inhaling, then simultaneously begin stretching/tightening various muscles or muscle groups for six seconds before releasing muscles on the left side of the body. Following are a few steps: (A) Standing and placing most of the body weight on the left side, stretch/tighten the muscles of the feet by curling the toes of the left foot for six seconds, then release. (B) Following a few more cycles of exhaling for a count of six and inhaling for a count of four, continue by stretching/tightening, on the left side only, the calf-muscle (gastrocnemius) for six seconds, then release. (C) Continue the sequence up the left half of the body, progressively stretching/ tightening selected muscles or muscle groups (e.g. left side quadricep, gluteal, external oblique, trapezius, forearm extensor & flexor...) for six seconds before releasing. Follow each round of stretching/tightening with breathing practice. (D) After all of the muscles of the left side have been progressively tightened and released, and there is a reminder to practice breathing, then enquire whether there is greater awareness of muscle tension on either the left or the right side of the body. (E) After discussing any reactions the initial part of the half-Jacobson progressive muscle relaxation technique, then proceed with the same pattern by tightening and releasing muscles on the RIGHT side of the body. (F) When finished with both sides, enquire about the extent to which there is greater awareness of relaxation as a result of following the principle of opposite related to muscle tension release as well as breathing practice.

- De Mora, J.M. (1982). *The principle of opposites in Sanskrit texts.* Delhi, India: Pandit Rampratap Shastri Charitable Trust, Shree Publishing House.
- Jacobson, E. (1938). *Progressive relaxation*. Chicago: University of Chicago Press
- Mann, F. (1992). *Acupuncture, a cure of many diseases, 2nd Edition.* New York: Butterworth-Heinemann Medical.

Eliminate Stress Immobilization Syndrome

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When we work at the computer most of us experience fatigue, neck, shoulder, arm hand and eye discomfort which commonly referred to as repetitive strain injury. However, repetitive motion is only one of many components that contribute to discomfort. A much more significant factor is lack of movement. We did not evolve to sit hours in a chair while looking vigilantly at a monitor screen. We are dynamic beings, requiring movement to "oil" our muscles and joints. Stop the debilitating effects of inactivity and learn skills to avoid STRESS IMMOBILIZATION SYNDROME. Maintain your health by frequently changing activities and performing movements. Do some of the following:

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- Every hour take a 5-minute break (studies at the Internal Revenue Service show that employees report significant reduction in symptoms without loss in productivity when they take a 5 minute break each hour)
- Leave your computer station for the 15-minute mid-morning and mid-afternoon breaks
- Eat lunch away from your computer workstation
- Take a short walk or do other movements instead of snacking when feeling tense or tired
- Have walking meetings or walk during part of your meetings
- Drink lots of water (then, you'll have to walk to the restroom)
- Take a 1-2 second micro-break every 30 to 60 seconds; drop your hands to your lap as you exhale
- Perform a stretch, strengthening, relaxation, or mobilization movement every 30 minutes
- Change work tasks frequently during the day
- Move your printer to another room so that you have to walk to retrieve your documents
- Stand up when talking on the phone or when a co-worker stops by to speak with you
- may facilitate skill mastery. For example, by combining breathing and muscle relaxation techniques, a greater degree of stress reduction may be achieved.

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14th Annual BFE Meeting

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14th Annual BFE Meeting

We offer 1 and 2 day professional training workshops with international experts and an outstanding Scientific Day.

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Chronic Pain... and many others

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AAPB is traveling to the Town & Country Resort in San Diego, California for its 41st Annual Meeting. Our theme this year is Personalized medicine in the age of technology: Psychophysiology and Health. Mark your calendars for March 24-27, 2010 to attend this gathering of experts in biofeedback, neurofeedback, and applied psychophysiology. You won‘t want to miss the highly-rated educational content and the networking opportunities available!

Please stay tuned to www.aapb.org as additional details are announced.

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The University Initiative, which promotes the creation of biofeedback and neurofeedback courses at universities, has been an important part of BCIA's global outreach.

We are proud that Monika Fuhs at Sigmund Freud University of Vienna now offers biofeedback and neurofeedback courses based on the BCIA blueprints. We are also excited about the first BCIA-accredited neurofeedback curriculum offered entirely in French. This course is offered at the Institute of Neurofeedback du Quebec by Vincent Paquette and Johanne Levesque. Both universities have chosen student completion of BCIA certification as a program objective.

We are pleased to announce the first generation of BCIA online exams. This process started slowly with the introduction of the human anatomy/physiology exam as an alternative to completing a semester anatomy/physiology course. Based on our experience with this first online exam, we launched online exams for all three certification programs.

As of August 2009, we had successfully offered online exams in Mexico and South Africa, with many more to come. Secure online testing has benefited both our American and international colleagues by eliminating their travel costs, making it easier to arrange for exam proctoring, and significantly reducing their special exam fee.

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GUIDELINES FOR SUBMISSIONS

Psychophysiology Today is published and distributed by the Biofeedback Foundation of Europe as an e-magazine.

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Articles should be of general interest to the Biofeedback community.

The general interest of the BFE is to offer educational, informative and, where possible, factually based articles and information.

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If your files are too large for sending electronically please submit on a cd to the address below.
For the authors section submit a biographical sketch (30 words) and photo of the author. Graphics and photos may be embedded in Word files to indicate position only. Please include the original, high-resolution graphic files with your submission -- at least 266 dpi at final print size. GIF or TIFF preferred for graphs and JPEG for photos.
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