COLD

FINGER

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COLD FINGER
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Post Traumatic Cold Intolerance is the most common, and often the most prominent disability from hand trauma. The discomfort caused by cold is believed to be linked to a dysfunction of the digital vasoregulation, but its pathophysiology is poorly understood. Cold induced vasospasm, i.e. the pathologically increased reactivity of the digital vessels to cold, is commonly found in hands that have sustained trauma, especially with damage to vascular and neural structures.

This thesis is based upon a series of clinical and laboratory studies on cold induced vasospasm and cold intolerance in 35 patients treated for digital amputation. The replanted digit was used as a study model, since it represents a body part which at the moment of reconstruction is devoid of all innervation. Replantations were performed according to two different principles of vascular reconstruction; using long or short vessel grafts. Finger Systolic Pressure (FSP) was used as a parameter of digital vasoregulation at different temperatures, and cold intolerance was assessed using a logarithmic rating scale (Borg). Non-injured fingers and amputation stumps were used as controls. Clinical and laboratory investigations were performed at different intervals from one week to three years after the reconstruction.

During the first two weeks following replantation, whole body cold exposure, or cooling of the replanted part to 10°C, did not cause serious spasm in the replanted vessels. Follow up investigations demonstrated that a cold related vasospastic tendency is established in approximately 60% of the replanted parts within one year after trauma. The once established pathologic vasoregulation, is unlikely to normalize spontaneously. Whether a cold related arterial spasticity will develop in the replanted digit or not, is not related to the surgeon's choice of technique for vascular reconstruction. Cold related arterial spasticity was more common in amputation stumps than in replanted digits, Our findings suggest that there is a pathological reaction to cold in the distal palm vessels but the nature of this disturbance is not clear.

All patients developed some degree of Post Traumatic Cold Intolerance. Approximately 60% of the patients stated that some improvement took place, but none of the patients was free of cold intolerance 1-7 years after the injury. Patients with a pathological cold induced vasospasm is likely to present with severe cold intolerance, which indicates that the vasospasm is involved as one of the causes of Post Traumatic Cold Intolerance.

Key words: Human, vascular, microsurgery, replantation. Cold intolerance. Vasospa
From the Clinic of Hand Surgery and the Departments of Surgery and Clinical Physiology,
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COLD FINGER

Clas Backman

University of Umeå
1993
"Don't believe what your eyes are telling you. All they show is limitation. Look with your understanding, find out what you already know, and you'll see the way to fly."

Richard Bach
COLD FINGER
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IV Backman C, Nyström Å, Backman Ch, Bjerle P. Arterial spasticity and cold intolerance in relation to time after digital replantation. J Hand Surg 1993; (accepted for publication J Hand Surg B.)

V Backman C, Nyström Å, Backman Ch, Bjerle P. Cold induced vasospasm in the replanted digit: A comparison between different methods for arterial reconstruction (Submitted)
The first successful replantation on a human was performed by Malt (quoted from Urbaniak 1988) who in 1962, replanted the arm of a 12 year old boy. The first report on an digital replantation using microvascular technique was published in 1965 (Komatsu & Tamai 1968). Increasing experience and refinement of techniques (Hayhurst & O’Brien 1975), has made replantation of amputated digits routine in many surgical centers around the world. The success rates have improved and digit survival is reported to be over 80% of replantation cases. With avulsion or crush amputation injuries, the risk for anastomotic complications is considerably higher, hence a lower survival rate is often encountered (Bieber et al. 1987, Hamilton et al 1984, van Beek et al 1978). The functional improvement that results from a digital replantation, is questionable. It is therefore important to establish if the reconstruction per se may result in loss of function. An example of the latter would be, if cold intolerance and disturbances of digital circulation are more prominent and common after replantation than after a less sophisticated reconstruction, i.e. stump revision and closure.

The present investigation focuses on the clinical problems of Post Traumatic Cold Intolerance (PTCI) and cold induced arterial spasm, encountered in practice of hand surgery. The replanted finger in the human has been chosen as study model as it enables us to study the effects of various stimuli on the circulation of a digit where all neural structures have been completely divided (Morrison et al 1978). It also presents an opportunity to assess subjective problems such as PTCI in relation to parameters that can be objectively monitored, such as systolic arterial pressures at various temperatures.
COLD INTOLERANCE

"Exposure to cold weather gives rise to an icy cold feeling which can progress to pain, sometimes lasting for several hours after the exposure" (Engkvist et al 1985). The cold intolerance syndrome, which may have a wide variety of traumatic or non-traumatic causes, is easy to recognize but difficult to assess. There is no effective and simple treatment for cold intolerance.

A. Post Traumatic Cold Intolerance (PTCI)

The Post Traumatic Cold Intolerance syndrome (PTCI), includes cold induced pallor, numbness and eventually pain of the affected fingers (Blomgren et al 1988, Gelberman et al 1978, May et al 1982, Morrison et al 1978, Tark et al 1989). It is common after minor as well as major hand trauma (Barron & Saad 1980), and in cold climate areas it may actually prevent the afflicted individual from continuing outdoor work or recreational activities. In a study on digital amputation patients, it was rated as the most disabling symptom, regardless of whether the digit was replanted or not (Nyström et al 1991).

The pathophysiology of PTCI is poorly understood, and has received little attention in the literature despite its importance as a disabling factor. A common opinion is, that the discomfort caused by exposure to cold is linked to a spastic tendency of the digital arteries, such as is commonly found in replanted fingers (Nylander et al 1987). It is not clear if PTCI is the immediate subjective expression of an abnormal digital circulation, or if PTCI and vasospasm are linked through a common cause, neural or other. A number of factors that have been studied in connection with PTCI, will now be discussed:

1. PTCI and arterial circulation

Tark et al (1989) and Morrison et al (1978), found that long term PTCI was more prominent in digital replantation cases where only one of the two arteries had been reconstructed. Gelberman et al (1978) found better tolerance to cold among patients whose replanted fingers had a pulse pressure greater than 80% of that of the control digits, whereas a pulse pressure
lower than 75% was common in patients suffering from severe PTCI. Other studies have failed to confirm a correlation between PTCI and vascularity of a replanted finger (Freedlander 1986, Kay 1985, Schlenker et al 1980).

2. PTCI and peripheral nerve function

Measuring two point discrimination Gelberman et al (1978) found an inverse correlation between PTCI and sensory recovery in a study on 35 replanted digits. Koman & Nunley (1986) studied digital thermoregulation by Isolated Cold Stress Testing (ICST) in a prospective study, and found a correlation between normalization of circulatory functions and recovery of sensation. Kay (1985) found a tendency towards more pronounced PTCI with poorer nerve function. Other studies dispute this correlation (Freedlander 1986).

3. PTCI and Post Traumatic Sympathetic Dystrophy

In a study by Engkvist et al (1985), 24 patients with PTCI after digital amputation were treated with regional intravenous guanethidine block. One third of their patients were relieved of symptoms for two to twelve weeks after treatment.

4. PTCI and vasoregulation

Nylander et al (1987) studied Finger Systolic Pressure (FSP) at different digital temperatures in eight patients with replanted digits. Six patients showed marked vasospasm and reported severe problems from PTCI. Two patients showed no vasospastic reaction, one of them troubled by PTCI and the other, a child, having none. In a study by Blomgren et al (1988) on 21 cases with replanted or revascularised upper limb parts, all patients reported cold intolerance, and six were tested for critical opening pressures (COP). Significantly reduced blood pressures were recorded in three patients (values not presented). The authors concluded that disturbed vasoregulation is to be considered in the explanation of PTCI.

From the studies mentioned above different qualities of the replanted digit has been evaluated and correlated to Post Traumatic Cold Intolerance. The problem is complex and no
single explanation has yet been found. We believe the vasoregulation to be of major importance and have focused our studies on cold induced vasospasm of the replanted digit.

**B. Non traumatic cold intolerance**

Raynaud's phenomenon, which is described as "episodes of pallor affecting one or more fingers on exposure to cold often accompanied by cyanosis and numbness" (Raynaud 1862 quoted by Nielsen 1978), is idiopathic in 20% of cases (Raynaud's disease). A wide variety of other conditions, such as sympathetic dystrophy, thoracic outlet syndrome (TOS), scleroderma, systemic lupus (SLE), and other collagen diseases, present with this symptom. Pathological changes in the vessels in Buerger's disease, atherosclerosis and endocrine disorders may also reduce the tolerance to cold (Egloff et al 1982). The reaction to cold has been postulated as due either to increased sympathetic outflow (Raynaud 1862 quoted by Fagilus & Blumberg 1985), or to local disorders (Lewis 1930). Theories depicting local pathophysiological causes of Raynaud's phenomenon have been proposed by several authors (Fagilus & Blumberg 1985, Freedman et al 1989).

Tobacco use aggravates cold intolerance by increasing the vasospastic reaction to cooling (Ekenvall & Lindblad 1989, Strömblad 1959, van Adrichem et al 1992).

Prolonged use of hand held vibrating tools may eventually lead to the syndrome known as "Vibration White Fingers" (VWF), which is recognized by cold induced attacks of "white fingers", numbness and pain (Ekenvall & Lindblad 1986, Hyvärinen et al 1973, Marshall & Poole 1954, Pyykkö & et al 1982). The underlying mechanism is not clear. Hyvärinen et al (1973) suggested overexcitation of the pacinian vibration receptors through a link with the sympathetic nervous system. A high vascular tone (Ekenvall et al 1987), possibly resulting from a peripheral neuropathy causing hypersensitivity of the adrenoreceptors to cold, was suggested by Pyykkö et al (1986). In a biopsy study, Takeuchi et al (1986) found three principal pathological changes related to VWF, namely thickening of the muscular layers of the arteries, demyelination neuropathy, and collagen increase in perivascular, perineural tissue and in corium of the skin.
C. Treating cold intolerance

The treatment of patients with Raynaud's phenomenon should include an initial recommendation to avoid cold and to refrain from tobacco use. Calcium blockers may be beneficial (Stevens & Moulds 1986).

Classical conditioning has been reported to reduce cold intolerance among patients with Raynaud's disease (Jobe et al 1982), and PTCI (Brown et al 1986).

Digital perivascular sympathectomy (Flatt 1980) has proven effective in idiopathic, and post traumatic, or frost bite cases (Egloff et al 1982, El Gammal & Blair 1991, Wilgis 1984), whereas patients suffering from scleroderma respond poorly to surgical treatment (Wilgis 1984).

In four patients suffering from PTCI after digital replantation, Hussl et al (1989) implanted a vascular bundle subcutaneously in the affected finger. The reported results were encouraging.

None of the suggested treatments is entirely satisfactory and in the clinical practice we are often confronted with the cold intolerant patient without being able to offer an effective regimen.

D. Assessing cold intolerance

Any study on PTCI, its causes and treatment, requires a tool for grading of the subjective symptoms.

Cold intolerance is most commonly registered as present or not present, or, in some studies, as giving no, little or severe symptoms (Gelberman et al 1979). Nylander et al (1987) specified activities indoor, outdoor in the summer, and outdoor in the winter. A detailed questionnaire was developed by McCabe et al (1991), who used a rating scale referring to such specific activities as "holding a frozen package from the freezer".

In an attempt to obtain more objective numerical values representing cold intolerance, Engkvist et al (1985) used a thermostimulator to assess the threshold temperatures that caused cold induced discomfort.
In summary there is presently no parameter which can be monitored in the physiological laboratory, which correlates well to the subjective feeling of discomfort from exposure to cold.

**PERIPHERAL CIRCULATION**

**A. Vasoregulation**

The peripheral circulation is regulated by central and local mechanisms, which may have a synergistic as well as antagonistic effect upon one another. (Mellander 1976)

1. Central regulatory system

   a. **Neurogenic factors**

   In the central control of peripheral circulation the adrenergic sympathetic vasoconstrictor fibres dominate. The adrenergic terminal plexus found at the adventitio-medial junction of the vessel wall (Aalkjaer & Mulvaney 1982), is abundant in the muscular arteries and arterioles. The veins are also innervated by the adrenergic system though not to the same degree as the arteries (Owman 1980).

   Noradrenalin, which is released from the adrenergic nerve endings, causes smooth muscle contraction by binding to alpha receptors located in vascular muscle (Bevan 1983, Stevens & Moulds 1982, Vanhoutte & Shepherd 1985).

   Neurogenic vasodilatation is mediated by cholinergic and peptidinergic nerves and corresponding neurotransmitters, acting directly on the vascular smooth muscle, prejunctionally, or at the ganglionic level. Indirect vasodilation results from inhibition of the sympathetic nerve fibre activity. Factors that inhibit the vasoconstrictor tone are dopamine (ganglionic) and acetylcholine (peripheral), thus acting as vasodilators (Owman 1980). Vasoactive neuropeptides such as substance-P and vasoactive intestinal polypeptide (VIP) have also been found to act as vasodilators (Bevam & Brayden 1987, Owman 1980).
b. Circulating factors

Adrenaline from the adrenal gland, is a vasoconstrictor through alpha receptor stimulation. In some regions adrenaline can cause vasodilatation, through beta receptor activity (Bevan 1983, Mellander 1976, Vanhoutte & Shepherd 1985).

Peripheral vasoconstriction elicited by circulating noradrenaline or adrenaline is similar to that resulting from sympathetic adrenergic activity, but is of a lesser magnitude (Mellander 1976).

Serotonin is primarily a vasoconstrictor (Arneklo-Nobin et al. 1985) whereas histamine is more complex as it induces both constriction (H₁ receptor) and dilation (H₂ receptor) of the arteries and also alters the permeability of the vascular bed. (Arner & Högestätt 1986, Owman 1980).

Extravasated platelets, activated by contact with collagen, release serotonin. They therefore indirectly act as vasoconstrictors. This effect is enhanced by cooling (Hou et al. 1986, Lindblad et al. 1984, Vanhoutte 1989).

2. Local regulatory system

a. Local vasomotor activity

This function is responsible for vasoconstriction following either changes in vessel length, alteration in blood pressure, or stretching of the vessels (Heyman & Ahlberg 1968). The myogenic pacemaker activity is responsible for basal vascular tone. Adrenergic nerve activity is responsible for basal tone in the cutaneous a-v shunts (Mellander 1976).

b. Chemical factors

Acidosis, reduced concentration of oxygen, and high concentrations of tissue metabolites, have been shown to cause vessel dilatation and functional hyperaemia (Haddy & Scott 1968, Mellander 1976, Skinner & Costin 1968). The dilatation of vessels is brought about by reduction of adrenergic nerve transmission and the local effect on the vascular smooth muscle, thus inducing a relaxation.
c. Endothelial secretion

The response of blood vessels to a variety of vasoactive agents is modulated by the endothelial cells which can release substances causing either dilatation or constriction. Endothelin is identified among the most potent vasoconstrictors. (Eglen et al 1989, Yanagisawa et al 1988). There are indications that the major Endothelium Derived Relaxing Factor (EDRF) may be nitric oxide (Vanhoutte 1989).

d. Temperature

Digital vascular resistance increases as a response to cooling, mainly by the influence of neural factors, and through the intermediary of adrenoreceptors. Lowering tissue temperature to 25°C reduces the release of noradrenaline from adrenergic nerves, and depresses vascular smooth muscle. There is a simultaneous increase in the alpha-adrenoreceptor responsiveness to a point where the resultant action is vasoconstriction. (Arner & Högestätt 1990, Bodelsson et al 1990, Ekervall et al 1988, Faber 1988, Vanhoutte & Shepherd 1970a, 1970b). Cooling also augments the contractile response to serotonin (Bodelsson et al 1989). Further lowering of the temperature below 10°C, however, causes vasodilatation as the vessels loose their responsiveness to vasoconstricting agents (Vanhoutte 1980).

"Following cold immersion there is an initial noradrenergic dependent vasoconstriction. Because of the poor insulation of the fingers the smooth muscle in the vessel wall is rapidly cooled. The cooling of the muscle reaches a point where it no longer responds either to nerve-ending derived or circulating cathecholamines. There is a consequent relaxation and increase in blood flow. This warms the muscle with a resultant rebound constriction" (Cooper 1981).

B. Acclimatization

In a healthy individual, the circulatory control mechanisms have an ability to acclimatize to prolonged and repeated cold exposure. Repeated whole body cooling has been shown to lower the threshold temperature for shivering (Shephard & Goode 1988). With chronic exposure of the hands to cold, hand skin temperatures were significantly higher during immersion in both
the initial constrictive phase and after vasodilatation and the acclimatized individual complained less of pain on cold stimulation (Elsner et al 1960, Leblanc et al 1960, Leblanc et al 1975, Nelms & Soper 1962, Krog et al 1960). The mechanism of acclimatization and improved tolerance to cold is not known.

C. Studies on digital circulation

To assess the effect of cold on digital blood flow, various methods have been used. The following techniques have been adopted in attempts to correlate cold intolerance and the arterial flow in digital replantation patients.

1. Digital pulse flowmetry (Darling et al 1972, Phelps et al 1979)

Gelberman et al (1978), recorded the digital arterial pulse pressure in 35 replanted digits and controls. The pulse pressure of the replanted digit registered as percentage of normal, was related to cold intolerance, correlation was good.

2. Laser doppler flowmetry (Holloway & Watkins 1977)

The doppler signal is linearly related to the microcirculatory blood flow (Stern 1975). Freedlander (1986) measured digital blood flow at room temperature and on exposure to cold, in replanted digits in 21 patients but found no correlation between cold intolerance and blood flow.

3. Venous occlusion plethysmography (Burton & Yamada 1951)

The flow rates of the feeding artery of a replanted digit can be calculated from the change in peripheral volume upon venous occlusion. This method was used by Kay (1985) in 14 patients but no correlation to cold intolerance was found.

4. Skin temperature (Koman et al 1984)

Digital pulp temperature and rewarming after cooling was registered and a specific pattern found in cases with severe cold intolerance. Using this method Nunley et al (1990) found a
normalization of the rewarming pattern in 11 replanted digits within one year of surgery.

5. Finger Systolic Pressure (Nielsen 1978)

This technique gives the opportunity to measure the blood pressure of selected digits after local cooling to preset temperatures and has been used in the evaluation of Vibration White Fingers (Arneklo-Nobin et al 1987)
AIMS OF THE INVESTIGATION

1. To assess the incidence and natural history of PTCI and cold related arterial spasticity in digital replantation patients.

2. To study the variations of PTCI and cold related vasospasm, with different principles adopted for primary and secondary surgical reconstruction.
MATERIAL

The patients

A total of 35 patients were studied.

The sex ratio M/F was 32/3, and their ages ranged from 15 to 63 years (median 33) Table 1. Replantation was performed on 26 patients (30 fingers) and nine patients were treated with stump revision and closure. The patients medical history did not reveal any medication with drugs that are known to influence peripheral vascular tone.

Direct vessel anastomoses, or short interpositional grafts were used in 14 cases (II, V). In 12 patients, the majority of which had sustained an avulsion amputation of a thumb, the replantation was performed using long arterial and venous grafts (I).

Pertinent patient data are summarized in Table 1.

1. Laboratory procedures

Recordings of Finger Systolic Pressure (FSP) at varying temperatures, have been made in all cases. Ten of the patients were examined on two or more occasions. Changes in digital pulp temperature of replanted and control fingers were recorded in all patients during 60 minutes of whole body cooling.

Within two weeks of replantation, 10 patients were studied to find out if arterial spasm of denervated vessels could be initiated by local or general cooling (III). Nine of these had been operated upon using long interpositional grafts and one long finger replantation had had a short arterial graft used in the reconstruction of the vessels.

The same patients as those included in paper III, with one exception, were re-examined 12 and 36 months (median) after replantation (IV). The purpose of this study was to investigate the incidence, the approximate time frames for establishment of cold related arterial spasticity of replanted fingers, and the long term development of the phenomenon.

With the purpose of evaluating the possible significance of the surgical technique for the
development of PTCI, 19 patients were examined 29 months (median) after replantation, (V).

Long grafts had been used in ten cases (long graft group), and direct vascular anastomosis or short grafts in nine cases (short graft group). In the long graft group, 8/10 patients had sustained avulsion amputations, and suture of one or both of the digital nerves was performed in 3/10 cases. In the short graft group, 6/9 patients had sustained avulsion amputations, and suture of one or both of the digital nerves was performed in 6/9 cases, either at the time of replantation or as a secondary procedure.

Nine patients with replanted fingers (no or short grafts) were studied together with a matching group of nine patients who after digital amputation had been treated with stump revision and closure. The purpose of the study was to assess the incidence and degree of cold induced vasospasm in relation to the different principles for surgical reconstruction (II). The digital nerves were reconstructed in 8/9 patients in the replantation group.

2. Subjective rating of symptoms

Nineteen patients received a questionnaire 37 months (range 9-95) after replantation in an attempt to correlate subjective symptoms of cold intolerance and social or professional history of work with hand held vibrating tools, with the results of laboratory investigations (IV,V). Of these patients 11 were tobacco users and four had a history of exposure to hand held vibrating tools.
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<td>avuls</td>
<td>1</td>
<td>1</td>
<td>s-v</td>
<td>I</td>
</tr>
<tr>
<td>31</td>
<td>F</td>
<td>avuls</td>
<td>1</td>
<td>1</td>
<td>s-v</td>
<td>I</td>
</tr>
<tr>
<td>42</td>
<td>M</td>
<td>avuls</td>
<td>1</td>
<td>1</td>
<td>s-a</td>
<td>I</td>
</tr>
<tr>
<td>31</td>
<td>M</td>
<td>guillot</td>
<td>2-5</td>
<td>2,3</td>
<td>0,s-a</td>
<td>I</td>
</tr>
<tr>
<td>29</td>
<td>M</td>
<td>crush</td>
<td>2-5</td>
<td>3,4</td>
<td>s-a</td>
<td>I</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>crush</td>
<td>2-5</td>
<td>3,4</td>
<td>0,s-a</td>
<td>I</td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>crush</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>I</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>saw</td>
<td>2-5</td>
<td>3,4</td>
<td>0,s-v</td>
<td>I</td>
</tr>
<tr>
<td>40</td>
<td>M</td>
<td>avuls</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>44</td>
<td>M</td>
<td>avuls</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>51</td>
<td>M</td>
<td>crush</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>46</td>
<td>M</td>
<td>crush</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>19</td>
<td>M</td>
<td>guillot</td>
<td>2-5</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>guillot</td>
<td>2-5</td>
<td>-</td>
<td>-</td>
<td>I</td>
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<tr>
<td>22</td>
<td>M</td>
<td>crush</td>
<td>2-5</td>
<td>-</td>
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<td>I</td>
</tr>
<tr>
<td>29</td>
<td>M</td>
<td>guillot</td>
<td>2-5</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
<tr>
<td>41</td>
<td>M</td>
<td>crush</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>I</td>
</tr>
</tbody>
</table>

0 = no graft; s-a = short arterial graft; s-v = short vein graft; l-a = long arterial graft; l-a+v = long arterial graft lengthened with a vein graft
A. Surgical technique

1. Replantation

Two fundamentally different principles for vascular reconstruction have been adopted.

a. Long grafts.

The interpositional grafts were harvested from the foot/leg (greater saphenous vein), and the abdominal wall (inferior epigastric artery). The vessel grafts were tunnelled subcutaneously from the replanted part to the distal forearm, for proximal arterial and venous anastomoses. No bone shortening was performed. The surgical technique is described in detail in paper (I).

b. Short or no grafts

Vascular reconstruction was achieved with direct anastomoses of arteries and veins, or with short (10-15 mm) arterial or venous interpositional grafts. The venous grafts were harvested from the volar aspect of the distal forearm and the arterial grafts were parts of the inferior epigastric artery. The proximal anastomosis was performed end to end to either the common digital artery or the digital artery proper. Varying degrees of bone shortening were routinely undertaken in these replantations.

2. Stump revision and closure

No standardized surgical technique was used since the mechanism of trauma varied in the group, but bone shortening was performed in a majority of the cases.
B. Finger Systolic blood Pressure, FSP

A double-inlet cuff with circulating fixed temperature water, was placed around the base of the examined digit. A mercury-in-silastic strain gauge was applied around the finger tip to register differences in pulp volume. The skin temperature was monitored by a thermocouple element. The finger tip was exsanguinated and blood supply occluded for five minutes. Warming or cooling of the finger to the desired temperatures could thereafter be performed. Finger systolic blood pressure (FSP) was recorded at 15°C or 10°C, expressed as a percentage of the FSP at 30°C, and corrected for changes in central systolic blood pressure, according to the following formula (Nielsen 1978, Nielsen et al 1980).

\[
FSP\% = \frac{FSP_{15 \text{ or } 10°C}}{FSP_{30°C} - (FSP_{\text{ref } 30°C} - FSP_{\text{ref } 15 \text{ or } 10°C})} \times 100
\]

An FSP% of 100 at any temperature represents a Finger Systolic Pressure identical to that registered at 30°C. An FSP% of 0 represents total occlusive arterial spasm.

Normal subjects demonstrate a decrease in digital blood pressure as the finger is cooled. A fall in FSP% from 100% to 67% at 15°C, or from 100% to 59% at 10°C, is considered a pathological vasospastic reaction to cold (Lagerkvist et al 1986). Only FSP% values obtained at 10°C are considered in this investigation.

C. Digital temperature response to whole body cooling

The patients, dressed in thin cotton underwear, were placed in a supine position in a draft-free chamber. Thermocouple elements were attached to the pulps of the replanted or amputated fingers, and of uninjured control fingers, and temperatures was registered at one minute intervals for 60 minutes. Body core and room temperatures were recorded simultaneously. The air temperature was set to 17°C-19°C (which is approximately 10°C below the temperature of equilibrium for man, undressed and at rest).
D. Questionnaire

A questionnaire was designed to evaluate the impact of cold intolerance on activities indoors and outdoors (summer/winter), and to determine the development of symptoms over time (IV, V). The estimation of subjective symptoms was made using a logarithmic rank scale designed by Borg et al (1981). Fig 1.

Fig 1

The logarithmic rank-scale with ratio properties that was used to assess the patient's perception of cold intolerance. (Borg)

0  Nothing at all.
0,5 Extremely weak. (just noticeable)
1  Very weak

2  Weak (light)
3  Moderate
4  Somewhat strong
5  Strong (heavy)
6
7  Very strong
8
9
10 Extremely strong (almost max)

- Maximal

The patients were also asked to give any history of professional exposure to vibration trauma, and their use of tobacco (smoke/smokeless). By calculating the total vibratory trauma to the hand according to ISO 5349 (Hagberg et al 1991), the patients could be delegated to either of two groups; with a history of significant or non-significant vibratory trauma.
RESULTS AND COMMENTS

A. Laboratory investigations:

1. 1-2 weeks of replantation

   It has been assumed that exposure to cold would be hazardous to a replanted digit because of the risk for vasospasm during the early postoperative period (Phelps et al 1979, Urbaniak 1988)

   The results (III) show that in nine out of ten patients there was no vasospasm in the replanted digits when exposed to cold during the first two post operative weeks. The mean values of the Finger Systolic Pressure showed a less pronounced cold induced vasospasm in replanted digits than in non injured controls. The reaction to whole body cooling showed a temperature increase in the replanted part, whereas the temperature dropped in all other digits. Table 2.

   As there is no serious vasospastic reaction to cooling of the body or local cooling of the digit, there should be no contraindication for cold exposure in the early period after replantation using long vascular grafts. The denervated vascular smooth muscle (VSM) is depressed by the exposure to cold (Faber 1988, Vanhoutte & Shepherd 1970a, 1970b), and dilatation is also induced from ischaemia dependent tissue metabolites. The circulating vasoconstrictors, and endothelial derived vasoconstrictive factors, seem to be of lesser importance to the regulation of vascular tone in the newly replanted digit. Bodelsson’s et al (1991) findings that free vein grafts respond to cooling with constriction, are not confirmed by our data.

   Even a very strong arterial reaction to cold is unlikely to occlude the radial artery in the forearm. This is not true for more peripheral arteries in the hand or fingers. Low environmental temperatures may therefore be a hazard to fingers that have been replanted with conventional techniques, i.e. using no or short grafts for arterial reconstruction.
Table 2
FSP%, and finger pulp temperature reaction to whole body cooling, during the early postoperative period.
Mean values

<table>
<thead>
<tr>
<th></th>
<th>Finger pulp temperature</th>
<th></th>
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<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>FSP% at 10° C</td>
<td>0 min</td>
<td>60 min</td>
<td></td>
</tr>
<tr>
<td>R</td>
<td>82.4 ± 18.6</td>
<td>33.1° C</td>
<td>33.2° C</td>
<td>n.s.</td>
</tr>
<tr>
<td></td>
<td>+0.1 ± 1.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CLR</td>
<td>58.2 ± 27.4</td>
<td>26.6° C</td>
<td>24.7° C</td>
<td>n.s.</td>
</tr>
<tr>
<td></td>
<td>-1.9 ± 4.1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = p<0.05
Students T-test was used for statistical analysis.

2. One to three years of replantation

Within one year of replantation, the arteries in replanted digits of 6/10 investigated patients had become hyperreactive to cold. Identical pathology was observed in five of these fingers two years later (IV) Table 3.

Whole body cooling causes a statistically significant drop in pulp temperature of the replanted digits as well as the controls. The differences were similar between the two groups of fingers Table 3. Once cold induced arterial spasticity is established in a replanted finger, the phenomenon is unlikely to fade with time.

*The sympathetic reinnervation of a replanted finger is sparse and inconsistent (Morgan & Wilgis 1986). This contributes to an imbalance between dilating and constricting factors, which might be responsible for the vasospastic tendency found one year after replantation. It is not possible to avoid this development with current
surgical technique, and selective digital sympathectomy might remain the treatment of choice in cases of severe PTCI after replantation (Egloff et al 1982).

Table 3

FSP% on local cooling and finger pulp temperature reaction to whole body cooling, investigated within 2 weeks after one year and after three years of replantation.

Mean values of FSP% 10° C

<table>
<thead>
<tr>
<th></th>
<th>I</th>
<th>II</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>82.4 ± 18.6</td>
<td>44.8 ± 44</td>
</tr>
<tr>
<td>CLR</td>
<td>58.2 ± 27.4</td>
<td>57.3 ± 32.2</td>
</tr>
</tbody>
</table>

Significant difference (p<0.05) is found between R II and RI.

Mean values of finger pulp temperature

<table>
<thead>
<tr>
<th></th>
<th>I</th>
<th>II</th>
<th>III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td></td>
<td>0 min 60 min</td>
<td>0 min 60 min</td>
</tr>
<tr>
<td></td>
<td>I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R</td>
<td>33.1° C 33.0° C</td>
<td>n.s 29.1° C 26.0° C</td>
<td>26.8° C 24.9° C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.1 ± 1.2</td>
<td>-3.1 ± 1.6</td>
</tr>
<tr>
<td>CLR</td>
<td>25.8° C 23.8° C</td>
<td>n.s 30.1° C 25.7° C</td>
<td>27.7° C 25.3° C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-2.0 ± 4.0</td>
<td>-4.4 ± 2.0</td>
</tr>
</tbody>
</table>

* = p<0.05

R = replanted digit. CLR = control finger, contralateral to R.
Examinations performed, I = (10 patients) Within 2 weeks of trauma, II = (9 patients) 12 (5-19) months after replantation, III = (7 patients) 36 (17-43) months after replantation.
Students T-test was used for statistical analysis.

3. Replantation as a cause for vasospasm

To elucidate (II) if the replantation causes the vasospastic reaction observed we compared Finger Systolic Pressure at 10° C in the replanted digit to amputation stumps.
The vasospastic reactions to cold were not statistically different between the two groups; replantation or stump revision and closure. Table 4.

Whole body cooling, showed a statistically significant decrease in temperature in the amputation stumps whereas the replanted fingers displayed a temperature decrease that was less marked. Table 4.

Replantation per se does not cause late cold related vasospasm. In fact, it is possible that replantation reduces the development of post traumatic cold induced vasospasm.

Of nine patients in the amputation group, four had a stump that was too short to fit the pressure tourniquet, and in these the adjacent finger was examined. Four out of five examined amputation stumps showed a vasospastic reaction to local cooling.

Table 4

FSP% on local cooling and Finger pulp temperature reaction to whole body cooling in replanted and amputated digits treated with revision and closure.

<table>
<thead>
<tr>
<th></th>
<th>FSP% at 10°C</th>
<th>Finger pulp temperature</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 min</td>
<td>60 min</td>
<td></td>
</tr>
<tr>
<td>R</td>
<td>37.3 ± 37.8</td>
<td>27.7⁰ C</td>
<td>25.8⁰ C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 1.9 ± 3.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Amp</td>
<td>29.0 ± 35.9</td>
<td>25.8⁰ C</td>
<td>21.4⁰ C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 4.4 ± 2.4</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

* = p<0.05
R= replanted digit. CLR= Control finger, contralateral to R.
Amp= amputated treated with revision and closure
Students T-test was used for statistical analysis.
4. Graft length

There was no statistically significant difference in arterial reaction to cold between digits replanted with long grafts, or with no or short grafts (V). The mean FSP% 10°C was 51.9 in the long graft group, and 28.9 in the short graft group Table 5. The correlation between replanted digits and controls was significant (rs= 0.747) in the short graft group. No such correlation was found in the long graft group (Spearman rank correlation coefficient). There were significant differences in FSP% between replanted and control digits in both groups (p<0.05 in long graft group and p<0.01 in short graft group). Five digits in the long graft group, but only two digits in the short graft group were non vasospastic.

The digital reaction to whole body cooling was similar in the two groups. The finger pulp temperature drop during one hour of whole body cooling was significant in replanted and control fingers. It was not more pronounced in the finger pulps of the replanted parts than in the controls, and there was no difference in temperature drop between the long graft group and the short graft group. Table 5.

The correlation in FSP% between replanted digits in the short graft group and controls, suggest that the vessels in the distal palm reacts to cold similarly in both hands. This correlation was not found in the long graft group where the proximal anastomosis was performed end to side to the radial artery in the distal forearm. The radial artery being the feeding vessel in the long graft group does not respond to cold in the same way as the common digital or digitl artery proper. This means that the cold induced vasospasm in the long graft group originates from the arterial graft and that the advantage of a long arterial graft, anastomosed end-to-side to a major artery in the forearm, seems not to be permanent. In the replanted digit the reinnervation of the vessels is by overgrowth across the suture line for a short distance (Waris et al 1988, Waris et al 1991), and by sprouts from the regenerating digital nerve (Morgan & Wilgis 1986, Waris et al 1988). The latter route is the quantitatively more important, which is the theoretical background to our hypothesis that the re-routed arterial graft,
in a replanted finger where the digital nerves are avulsed from far proximal, would be less subject to adrenerg dependent vasospasm.

### Table 5

FSP% on local cooling and Finger pulp temperature reaction to whole body cooling in replanted digits, reconstructed with long or short grafts.

<table>
<thead>
<tr>
<th></th>
<th>FSP% at 10°C</th>
<th>Finger pulp temperature 0 min</th>
<th>Finger pulp temperature 60 min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R (lg)</td>
<td>51.9 ± 37.3</td>
<td>27.8°C</td>
</tr>
<tr>
<td></td>
<td>R (sg)</td>
<td>28.9 ± 30.23</td>
<td>27.2°C</td>
</tr>
</tbody>
</table>

|                  | - 2.4 ± 2.1  | - 3.0 ± 2.6                  |                               |

* = p<0.05

R = replanted digit. Revascularization method: (lg) = long grafts, (sg) = short grafts

Students T-test was used for statistical analysis of temperature difference in each group. Wilcoxon nonparametric test was used for the analysis between the groups.

### 5. Mechanism of trauma, Nerve reconstruction

Twentysix patients, representing all of the replantations included in our series of studies, were compared as to the type of trauma and its influence on FSP%. They were also evaluated as to the significance of the presence or absence of a nerve suture. An analysis of these data failed to reveal any significant difference in FSP% at 10°C between fingers that had been replanted after avulsion (n=16, mean value 39.3 ± 35.5) or after crush/clean cut amputations (n=10, mean value 42.3 ± 36.5). Similarly, there was no significant difference in the obtained values of FSP% in fingers where nerve reconstruction had been performed or not. (nerve reconstruction FSP% = 40.5 ± 35.18, nerve not reconstructed 40.4 ± 36.8)

Some authors state that cold intolerance and disturbances in digital circulation are
related both to the mechanism of trauma and to reinnervation of the replanted part (Gelberman et al. 1979, Glickman & Mackinnon 1990). Our study dispute these differences as no such correlation was found in FSP%.

B. Questionnaire (V)

In general, patients do not experience PTCI during the first weeks following a hand trauma. The onset of cold related symptoms is thus delayed, but was established within four months in all 19 patients without exception. Six patients stated that their PTCI started to become less troublesome at about one year after trauma, and in one patient, at 2 years. The improvement seems not to be dramatic, and four patients state that they still improved one to seven after the injury (one of the patients was investigated 1½ year after trauma).

Eight patients claimed that their PTCI reached a plateau within the first year of the trauma, and that it since then has not improved (detailed information on the questionnaire are presented in paper V).

Five of the eight patients who have not experienced any improvement in PTCI belonged to the short graft group. Five out of six patients that experienced decrease in PTCI after one year belonged to the long graft group. There was no difference in PTCI scores relating to use of tobacco, or previous use of vibrating hand held tools.

*Several authors have put forward the opinion that PTCI normally improves in all afflicted patients (Early & Watson 1984, Gelberman et al 1979, Morrison et al 1978, Tark et al 1989), and that in children it may disappear completely (Nylander et al 1987). The adaptation to a new situation, should not be confused with a true reduction in PTCI, nor with the acclimatization found in normal individuals with repetitive cold exposure over longer periods of time (Adams & Smith 1962, Elsner et al 1960, Krog et al 1960, Leblanc et al 1960, Nelms & Soper 1962). Our results indicate an increased acceptance of discomfort during cold exposure, rather than a reduced vasoconstrictor fibre response to a given cold exposure (Krog et al 1960).*
The scores on PTCI that were obtained from the questionnaires (V), were compared with the FSP% values from the laboratory investigations. No correlation was found between these two variables. However the seven patients that were non-vasospastic (FSP% of >59) rated cold intolerance lower than the 12 patients with pathological vasospasm, and the difference between the groups was statistically significant (p<0.05, Wilcoxon nonparametric test).

*Several papers have dealt with the problem of finding a parameter reflecting the cold intolerance. The results vary and are often disputed. Gelberman et al (1978) reported a correlation between digital circulation, and patient assessments of PTCI, recorded as giving no, little or much symptoms. The results in our study show that the numerical values of FSP is not a good parameter of PTCI, but in the cases of a pathological vasospasm we are likely to find severe problems from cold intolerance. It also illustrates the fact that no reliable method of objectively assessing discomfort from cold exposure, i.e. PTCI, has been presented.*
DISCUSSION

A. DIGITAL REPLANTATION

The decision as to whether or not an amputated digit should be replanted is influenced by a multitude of factors.

A catalogue of indications and contraindications will be interpreted differently depending upon the state of surgical skill, the quality of the available equipment, the economy of both the society and the individual. Most authors consider an amputation of the thumb, of multiple digits, of a partial or a whole hand, or any amputation in a child, to be a good indication for replantation (Blomgren et al 1988, Earley & Watson 1984, Jones et al 1982, Tamai et al 1983). Many factors such as multiple trauma where prolonged surgery for replantation may hazard the patient's life, severe damage to the amputated part, extensive mutilation of the hand and arm from which the part has been amputated (Lister 1991), have been suggested as relative or total contraindications for replantation surgery. Single digit amputation, prolonged ischemia, advanced age, avulsion amputation, previous injury to the part, psychological disturbance and the assumed higher risk for development of PTCI after replantation have also at one time or another been named among the contraindications to replant. (Aasko-Seljavaara et al 1988, Tamai et al 1983, Vlastou & Earle 1987, Weiland & Raskin 1990).

A common misunderstanding about reconstructive surgery concerns the cost-benefit relations of replantation as opposed to stump revision and closure. Even in absolute values, the cost for replantation seems not to exceed that of less sophisticated reconstruction (Ekeroth et al 1986).

The functional gain of digital replantation is probably of little importance in most cases, and does not correspond with the often very high degree of patient satisfaction (Brown 1982, Bunke 1990, Goldner et al 1990, Nyström et al 1991, Tamai 1982). Yet, few authors have discussed in any depth the psychological aspects of replantation surgery. Brown (1982) pointed out that the motivation of the patient is more important for hand function, than the absence of a digit, and Grunert et al (1988a and 1988b) demonstrated the psychological impact
that comes with major hand injury. Others have mentioned the psychological benefits of replantation and it is well known that patients, especially women, tend to be pleased also with a replanted digit of poor function, as long as the cosmetic appearance is satisfying (Blomgren et al 1988, Nyström et al 1991, Russell et al 1984, Scott et al 1981). On the other hand, patients whose amputated parts have not been replanted, commonly refer to the fact that a part of their body is missing as a major source of unhappiness (Schweitzler et al 1986). It is therefore essential that replantation is not excluded as an option because of the surgeon's erroneous opinion that function would be more important than psychological factors (Aasko-Seljavaara et al 1988, Kleinert et al 1980, Nylander et al 1984).

Replantation of an amputated part is indicated whenever the patient expresses a strong wish after thorough information concerning the functional and cosmetic outcome as estimated by the surgeon responsible for reconstruction.

**B. COLD INDUCED VASOSPASM**

It has been assumed and largely accepted that a low environmental temperature may jeopardize the survival of a replanted part during the first postoperative days or weeks (Phelps et al 1979, Urbaniak 1988). It is possible that this opinion has come from amalgamating Wirchoff's triad, and the observation that cold exposure may cause peripheral vasospasm (Acland 1972, Hayhurst 1976). As shown in our study cold induced arterial spasm seems not to exist in the replanted finger during the first postoperative weeks if long enough grafts have been used for reconstruction of the artery, peripheral spasm is a highly unlikely reason for circulatory failure in a replanted digit. Measures to relieve a supposed vasospasm are therefore of no value, unless possibly in cases where no grafts or short grafts have been used to reconstruct the artery. A spasm could then theoretically affect the innervated segment of artery proximal to the anastomosis.

Nunley's et al (1990) concept for investigating the vasoregulation of the replanted finger, is different from the one adopted in our studies. While we have investigated the immediate effect of cold on the tone of digital vessels, Nunley's Isolated Cold Stress Testing (ICST) monitors
the rewarming process after digital cooling. At present it is not possible to explain why the
initially deranged function of rewarming tends to normalize within a year from replantation,
while a once established tendency towards cold related arterial spasm does not resolve.

The adrenergic reinnervation of the vessels, which will occur to some extent within the first
year after replantation, is essential for the development of cold induced vasospasm (Faber
1988). If the vasospastic reaction was due to an increased sensitivity of the arteries of the
replanted digit to catecolamines or other circulating factors, it would be expected to appear
also after segmental digital sympathectomy of the common digital artery and the proximal part
of the digital artery proper (Flatt 1980). Our results showed a lower mean value of FSP% in
the replanted digits using short grafts, compared to long graft technique and a correlation
between control digits and replanted digits in the short graft group. These facts in addition to
the findings that there was a pathologic vasospasm in four out of five amputation stumps,
indicates that the feeding vessel (the common digital or digital artery proper still intact as to
the adrenergic sympathetic innervation) is the site of pathological vasoconstriction on cold
stimulation.

This theory is supported by the positive results reported after limited or extended
sympathectomy for PTCI, with improved digital vascularity and reduced discomfort from cold

Perivascular sympathectomy is effective only at the site of adventitiectomy (Kaarela 1991,
Kaarela et al 1991, Waris et al 1991). To achieve an adrenergic denervation more distally,
requires division of the digital nerve proper (Waris et al 1988), since the distal sympathetic
fibres travel along the peripheral nerves and each digital artery receives 3-12 twigs from
adjacent digital nerves (Morgan et al 1983, Wilgis 1981). Why cold related symptoms can be
abolished through a more proximal adventitiectomy of the arteries in the distal palm, leaving a
normal distal adrenergic innervation to the digital vessels, is not clear. The explanation can not
be purely mechanical, as suggested by Kaarela (1992), since blood flow through the more
distal tissues depends upon the peripheral, not the central vascular resistance (Nyström et al
1990).
There is a heterogenicity in different vessels' response to cold, and trauma might alter adrenoreceptor function or impair the factors responsible for active vasodilatation of the distal palm arteries. In fact, cold related vasospasm might be less of a defect in adrenergic innervation, than in active vasodilatation.

Disturbed vasoregulation must be considered in the explanation of the PTCI phenomenon but an effective armament for prevention or treatment of the condition can probably not be found until a better understanding of the pathophysiology of PTCI and cold related arterial spasm, and their interactions, is gained. There is a need for a technique allowing the assessment of threshold temperatures for cold induced discomfort or pain, to facilitate the study of PTCI in order to define its etiology and pathophysiology and provide means to evaluate various principles for its treatment or prevention.

A vasospastic tendency in a replanted finger reflects an imbalance between vasoconstricting and dilating factors. It is unlikely that the finger's adrenergic innervation would be more dense after the replantation than it was before the amputation. Fields of particular interest for study in the future might therefore concern individual factors responsible for vasomotor control, with a focus on the dilating factors such as neuropeptides, endothelium derived factors and other related substances especially in the arteries of the distal palm.
1. **Cold related arterial spasticity** does not affect arterial grafts in the hand during the first postoperative weeks. Cold exposure is therefore not a risk factor in replantation surgery if the vessels of the replanted part have been anastomosed to a sufficiently large donor artery.

2. **Cold related arterial spasticity** is common in replanted fingers. It is found in approximately 50% of cases operated with long grafts for arterial reconstruction.

3. **Cold related arterial spasticity** is not more common after digital replantation, than after stump revision and closure.

4. **PTCI** affects close to 100% of digital replantation patients, regardless of the methods for vascular repair.

5. **PTCI** is likely to reach a peak level within the first years of the trauma. Approximately half of the afflicted patients will eventually consider their cold related handicap less prominent.

6. **PTCI** does not correlate with variations in digital blood flow as determined by measurements of FSP, but in cases of cold induced vasospasm it is likely to be a more severe.

7. Improved success rates and possibly less prominent PTCI are given, using long grafts when **replanting** avulsed digits.
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Lewis T. Observations upon the reactions of the vessels of the human skin to cold. Heart 1930; 15: 177-208.


Schlenker JD, Kleinert HE, Tsai T-M. Methods and results of replantation following traumatic amputation of the thumb in sixty-four patients. J Hand Surg 1980; 5: 63-70.


