

Sclerotherapy Complications and Contra-Indications

Dr Paul Thibault

Phlebologist

Newcastle NSW

Sclerotherapy

Complications and Contraindications

- *“World-wide clinical experience has proved beyond all possible doubt that embolism is a complication not to be feared and its interest is almost purely theoretical.*

Properly conducted, the injection treatment of varicose veins can be carried out without the slightest risk, and if reasonable care be exercised, ulceration at the site of injection becomes a complication of extreme rarity”

Dr Victor Coppleson 1929

Patient Selection (Contraindications)

- Absolute
 - Acute deep venous thrombosis
 - Allergy to sclerosant



Patient Selection (Contraindications)

- Relative

- Predisposition to thrombosis

- Past history of thrombosis/PE
 - FH venous thrombo-embolism
 - Hypercoagulable state eg polycythaemia rubra vera
 - Long-distance travel
 - Known Thrombophilia
 - Bedrest and inability to ambulate
 - Pregnancy
 - 1st and 3rd generation Ocs
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Sclerotherapy and Thrombosis

- *“World-wide clinical experience has proved beyond all possible doubt that embolism is a complication not to be feared and its interest is almost purely theoretical.”*
 - Dr Victor Coppleson 1929
 - *“The reported incidence has been less than seen in the general population”*
 - Dr Craig Feied Semin Dermatol 1993
 - *Anecdotal reports that asymptomatic DVTs are more common than recognised*
 - Parsi and Myers
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Sclerotherapy and Thrombosis

- Large veins are probably at no greater risk than small veins
 - STS and POL are anticoagulants at concentrations $>0.3\%$
- There is no evidence that patients with known thrombophilia are more predisposed to post-sclerotherapy VTE than others
- Medico-Legal Indication for Prophylactic Clexane
 - Significant thrombophilia
 - Past History DVT or PE if no significant contributory cause



Sclerotherapy and Thrombosis

● Diagnosis

- Asymptomatic
- Painful swollen leg
- Pulmonary embolus (chest pain, dyspnoea)



Sclerotherapy and Thrombosis

Complications	STS <i>N</i> =2,686	POL <i>N</i> =16,804
Death	0	0
Anaphylactoid	4 (0.15%)	0
Urticaria	2 (0.07%)	12 (0.07%)
Rash	1	2
Migraine	3	3
Deep vein thrombosis	0	3 (0.02%)

Sclerosant Effects on Clotting Tests (*Parsi*)

- STS prolongs all clotting tests at concentrations **higher than 0.3%** demonstrating potent **anticoagulant** activity.
 - STS shortens XACT and NAPTT at concentrations **less than 0.3%** demonstrating **pro-coagulant** activity.
 - STS behaves as a procoagulant phospholipid at concentrations lower than 0.3%.
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Sclerosant Effects on Clotting Tests (*K Parsi*)

- POL has less dramatic effect on clotting time
 - POL has **more procoagulant activity at low concentrations** when compared with STS with increased exposure time.
 - STS is a **more potent anticoagulant at high concentrations** when compared with POL.
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Sclerotherapy and Thrombosis Prevention

- Selection of patients
 - Post-treatment graduated compression stocking
 - 3-4 days day and night
 - 10 – 20 days during day
 - Walk immediately after treatment 15-20 minutes
 - Daily walking 45 minutes
 - Prophylactic Clexane for high-risk patients
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Patient Selection (Contraindications)

- Relative

- Predisposition to thrombosis
 - **Defer or avoid Rx or consider prophylactic LMWH**
 - Past history of thrombosis/PE
 - FH venous thrombo-embolism
 - Hypercoagulable state eg polycythaemia rubra vera
 - Long-distance travel
 - Known Thrombophilia
 - Bedrest and inability to ambulate
 - Pregnancy
 - 1st and 3rd generation Ocs
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Allergy and Anaphylactic Reactions

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Allergy and Anaphylactic Reactions

- More likely with STS
- More likely with stronger solutions ($>1\%$)
- Most commonly pseudoanaphylactic (anaphylactoid)
- Anaphylactic reactions can occur also



Anaphylactic Reactions

- Type 1 hypersensitivity reaction
 - Requires previous exposure
 - always caused directly by degranulation of mast cells or basophils mediated by IgE
 - Time of onset varies from minutes to hours after exposure
 - Biphaseic anaphylaxis (relapse within 72 hrs) occurs in 1 – 20% of cases
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Anaphylaxis - Symptoms

- **Skin** involvement including generalized hives, itchiness, flushing, and swelling of the lips, tongue or throat. (90%)
 - **Respiratory** symptoms may include shortness of breath, wheezes or stridor, and low oxygen. (70%)
 - **Gastrointestinal** symptoms may include crampy abdominal pain, diarrhea, and vomiting (45%)
 - **Cardiovascular** Hypotension can cause fainting & LOC. Due to histamine release, coronary artery spasm may occur with subsequent myocardial infarction or dysrhythmia (45%)
 - **CNS** - loss of bladder control and muscle tone
- feeling of anxiety and “impending doom”
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Pseudoanaphylaxis (Anaphylactoid)

- does not involve an allergic reaction but is due to direct mast cell degranulation
 - most common trigger for this mechanism is an intravenous infusion of an iodine-containing radiological contrast medium
 - exact mechanism with sclerosants is unknown but may be related to degranulation (release of mediators) from endothelial cells
 - occurs on 1st exposure to sclerosant and within minutes of injection
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Allergy and Anaphylactic Reactions

- Management
 - Awareness and early detection
 - Adrenaline (sc or im)
 - I-V line and airway (O₂)
 - corticosteroids
 - ?admission to hospital
- Avoidance
 - Use foam rather than 1-3% STS solution as direct degranulation is dose/time -related

Patient Selection (Contraindications)

- Relative

- Current other systemic disease
- Lymphoedema
- Deep venous obstruction (only about 10% of cases)
- Patent foramen ovale - avoid foam

Local Complications

- Postsclerotherapy pigmentation
 - Telangiectatic Matting
 - Cutaneous Ulceration
 - Superficial Thrombophlebitis and Acute Chemical Phlebitis
 - Infection
 - Hypertrichosis
 - Skin reactions to tape, compression pads and stockings
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Skin Reactions

- Allergy to tape
- Friction (cleavage) reaction to compression pads
- Allergy to stocking fibres



Postsclerotherapy Pigmentation and Telangiectatic Matting

- Most common adverse sequelae
- Commonly occur together
- Probably have common aetiology
 - disruption of endothelium
 - thrombus formation
 - inflammation

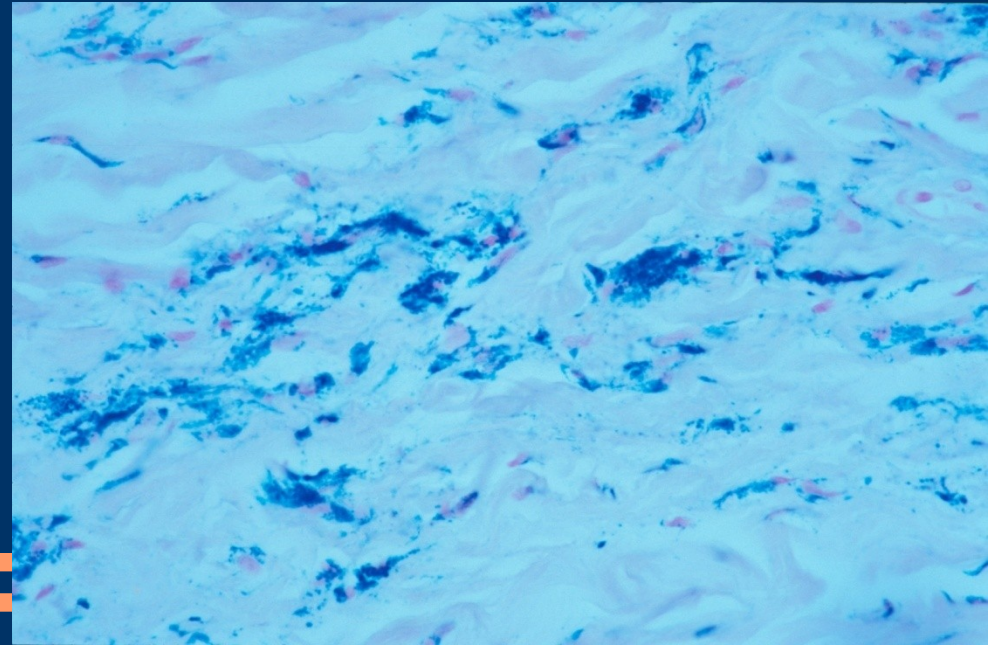


Post-sclerotherapy Pigmentation: What is it?

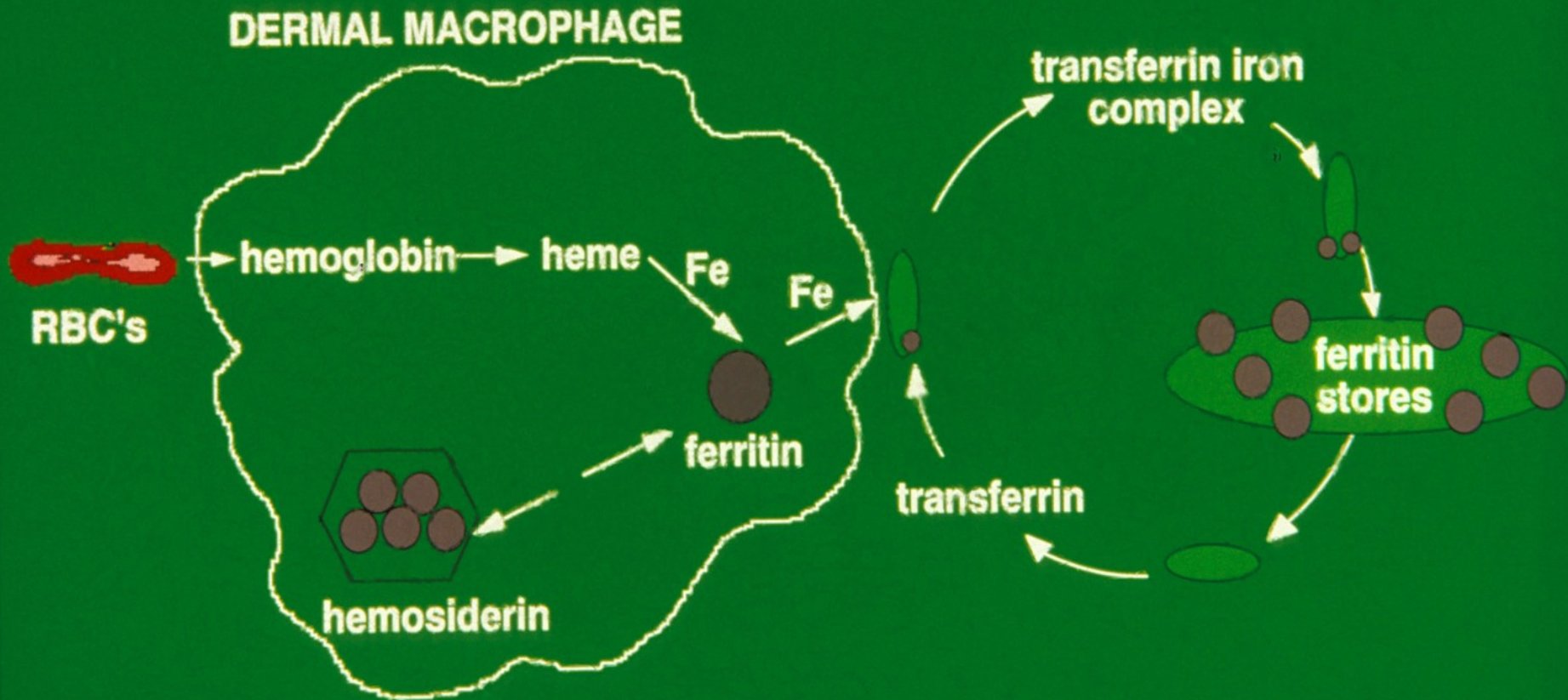


Perl's stain

Haemosiderin found
around dermal vessels



CELLULAR MECHANISM OF PSHP



Postsclerotherapy Pigmentation: Aetiologic Factors

- Proportional to quantity of haem (Fe) in tissues
- Inversely proportional to rate of removal of Fe from tissues



Postsclerotherapy Pigmentation: Aetiologic Factors

- Sclerosing solution type and concentration
 - Sclerotherapy technique
 - Persistent proximal venous incompetence
 - Vessel fragility (age)
 - Vessel diameter
 - Total body iron stores/transport mechanism
 - Post-sclerotherapy management (compression)
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Post-sclerotherapy Pigmentation: Causes

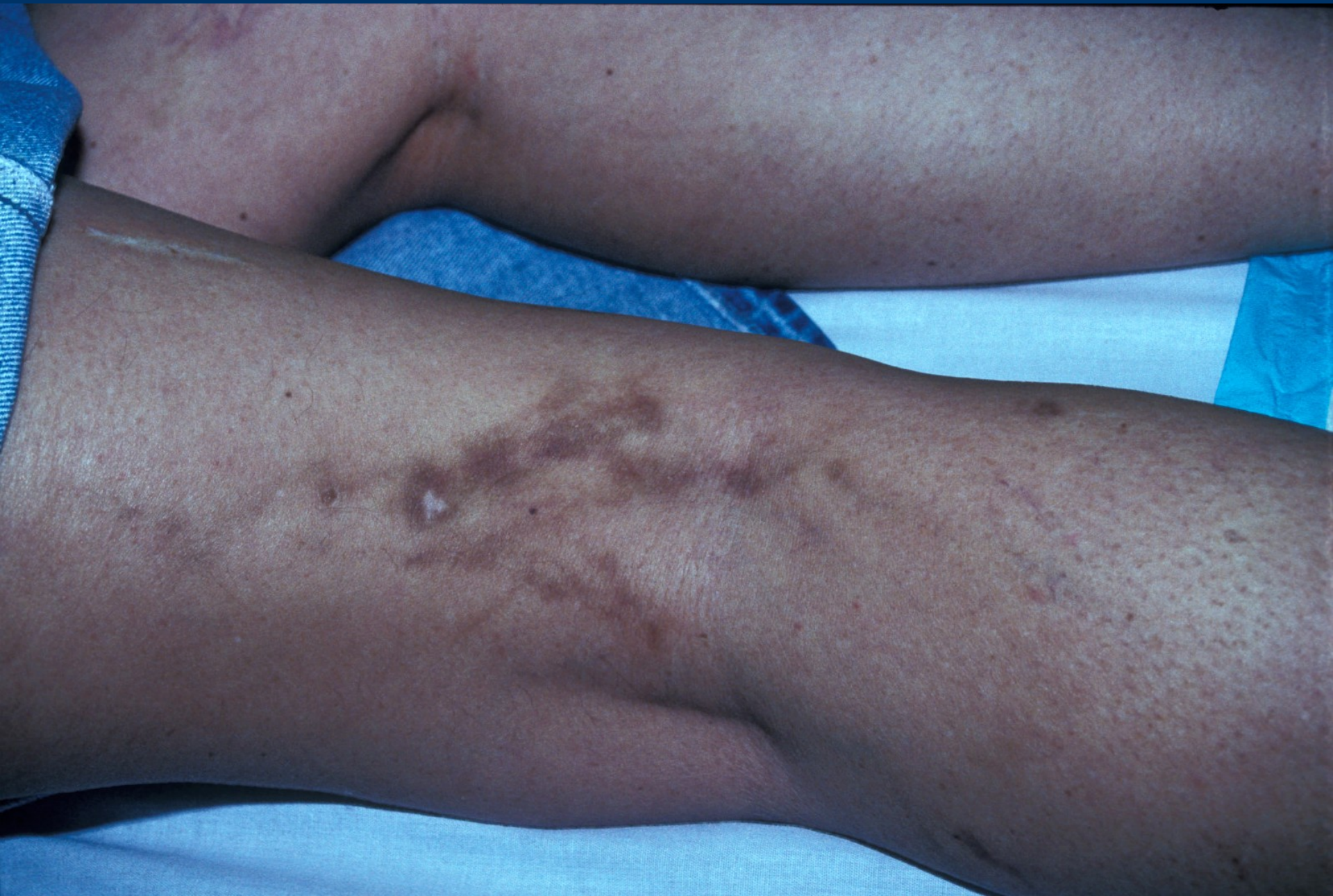
Solution Type and Concentration

- Excessive endothelial destruction predisposes to pigmentation
 - Potent sclerosants (eg STS,POL,Iodide)
 - Less with weak sclerosants (HS and chromated glycerin)
 - Concentration dependent (STS and POL)
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Local Complications

Complication	STS <i>N</i> =2,686	POL <i>N</i> =16,804
Ulceration	4 (0.15%)	32 (0.2%)
Significant or severe pigmentation	5 (0.2%)	30 (0.2%)
Telangiectatic matting	4 (0.15%)	7 (0.04%)
Superficial thrombophlebitis	3 (0.1%)	14 (0.08%)

Excessive Sclerosant Concentration



Postsclerotherapy Pigmentation: Aetiologic Factors

- Sclerosing solution type and concentration
- Technique



Postsclerotherapy Pigmentation: Technique

- ✗ Failure to treat proximal point of reflux first
 - incompetent junctions
 - incompetent trunks
 - larger varices
 - reticular veins
 - ✗ Excessive injection pressure
 - 2-3ml syringes therefore better than 1ml
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Failure to treat proximal reflux

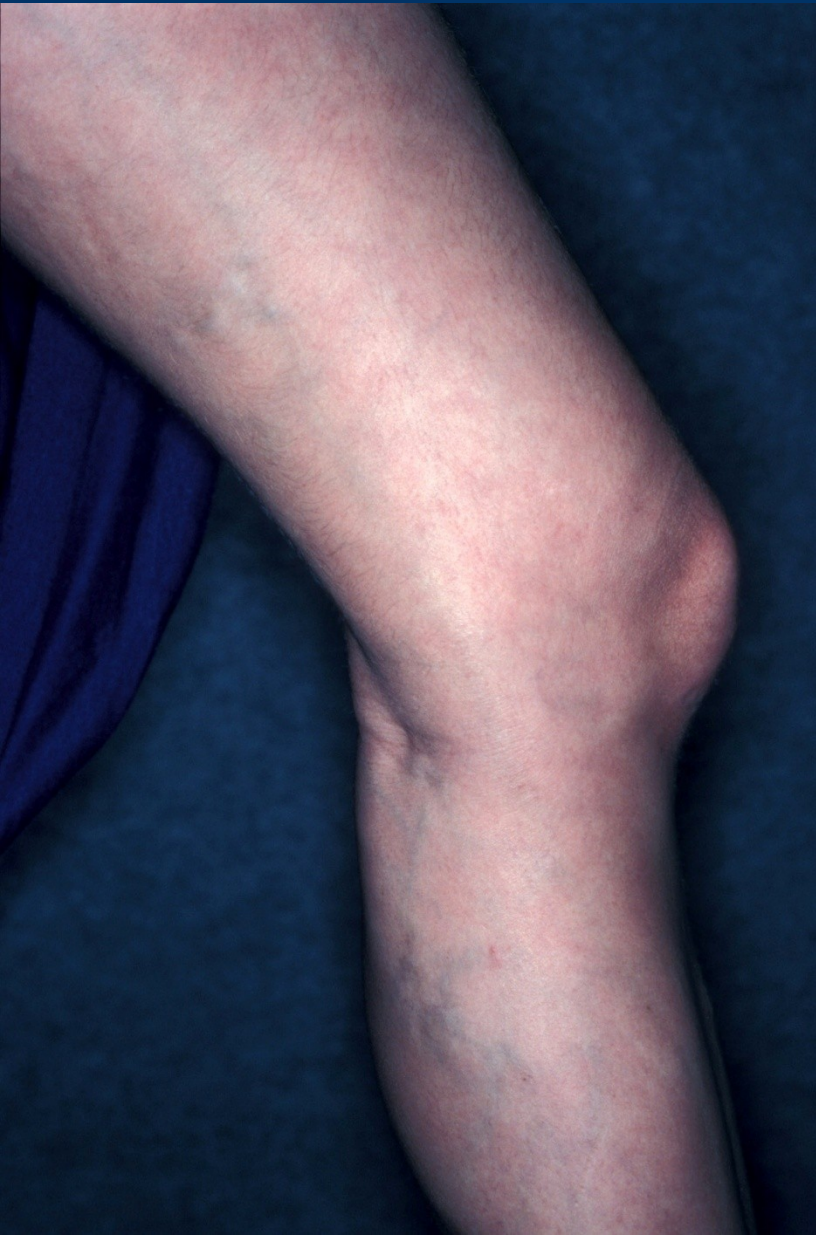


Postsclerotherapy Pigmentation: Aetiologic Factors

- Sclerosing solution type and concentration
- Technique
- Predisposition to pigmentation



Size of Vein and Sclerosant Concentration



Postsclerotherapy Pigmentation:

Predisposition to pigmentation

- Unrelated to skin or hair colour
 - Increased vessel fragility
 - post-menopausal
 - menstruation
 - Minomycin - blue-grey
 - Total body iron stores
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Minomycin staining



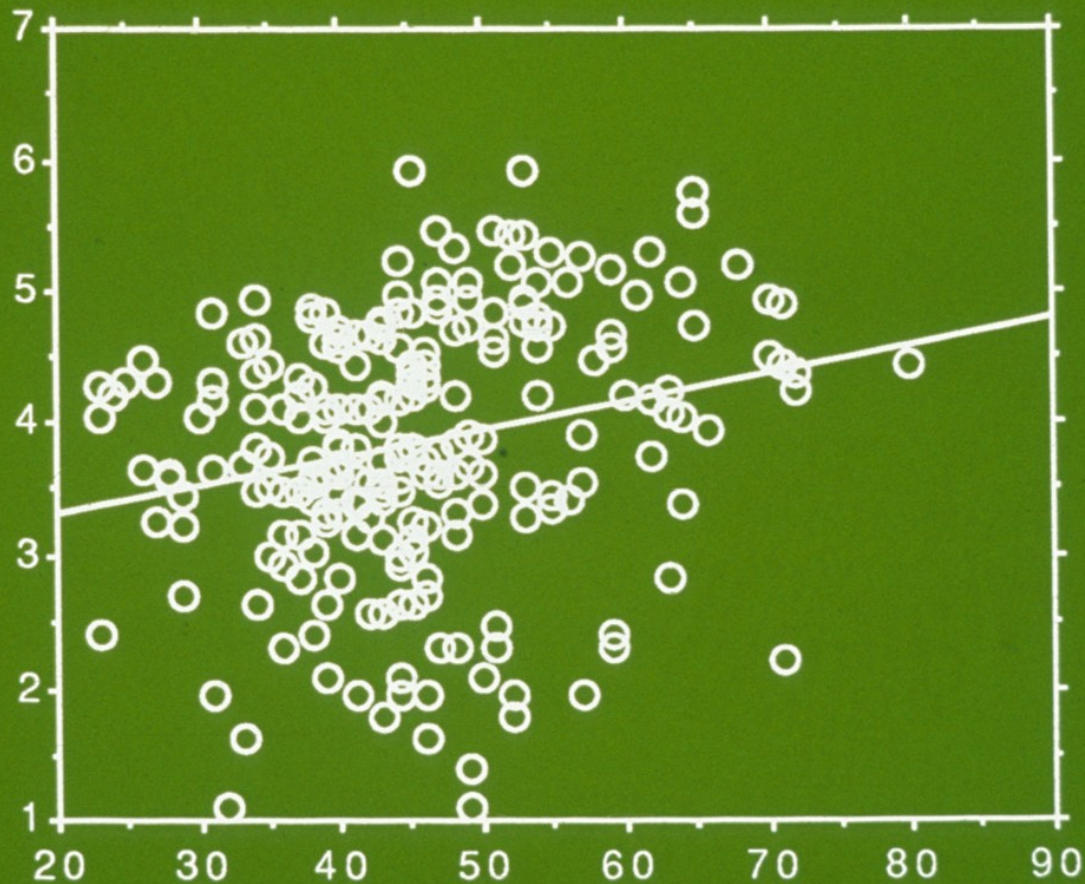
Post Sclerotherapy Pigmentation

The role of Fe

- Prospective study JDSO Oct 1994 (Thibault & Wlodarczyk)
 - 233 patients
 - Serum Ferritin taken prior to treatment
 - Followed at 3, 6, 12 months for PSP
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Serum Ferritin vs Age

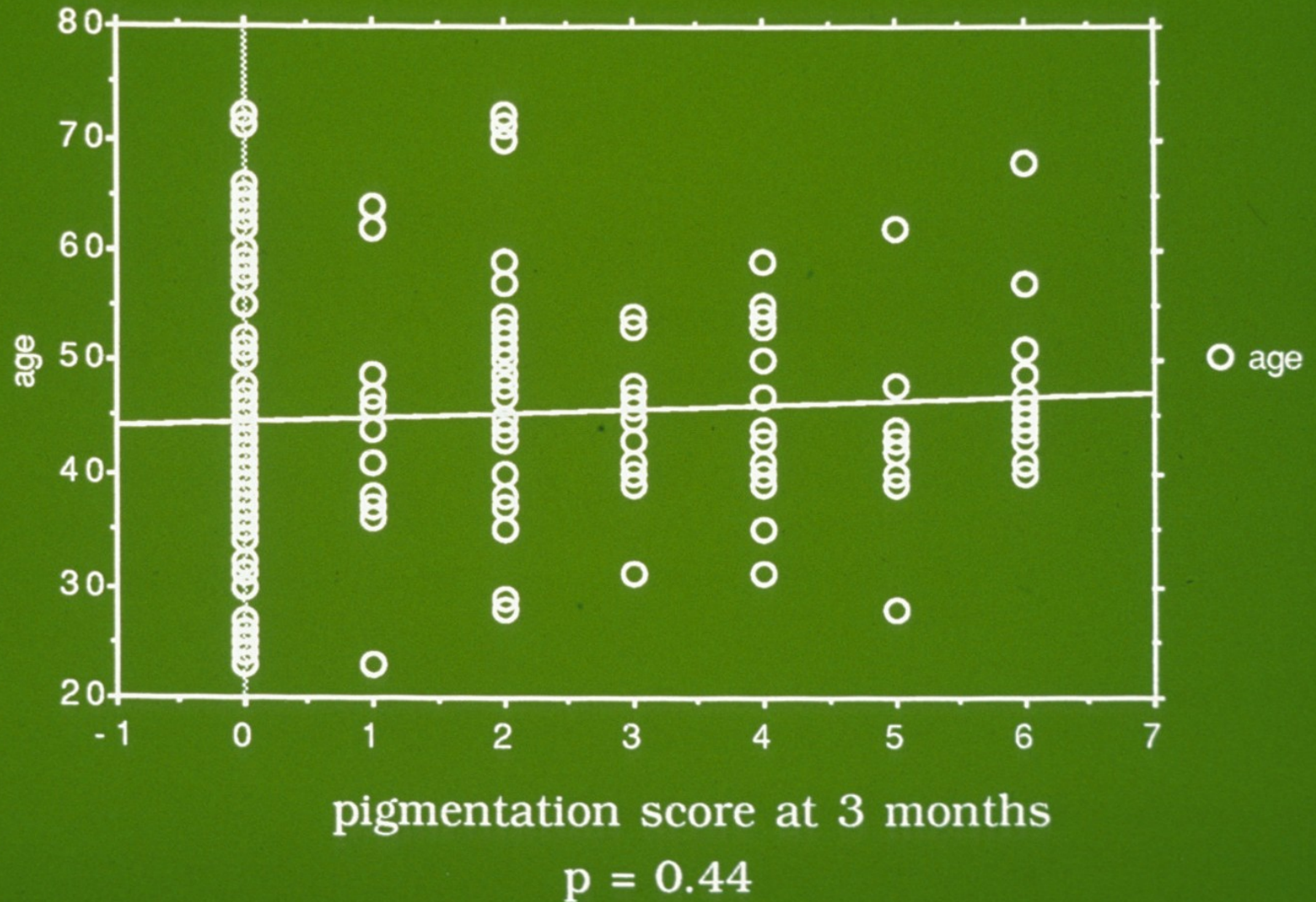
Index of
Serum
Ferritin



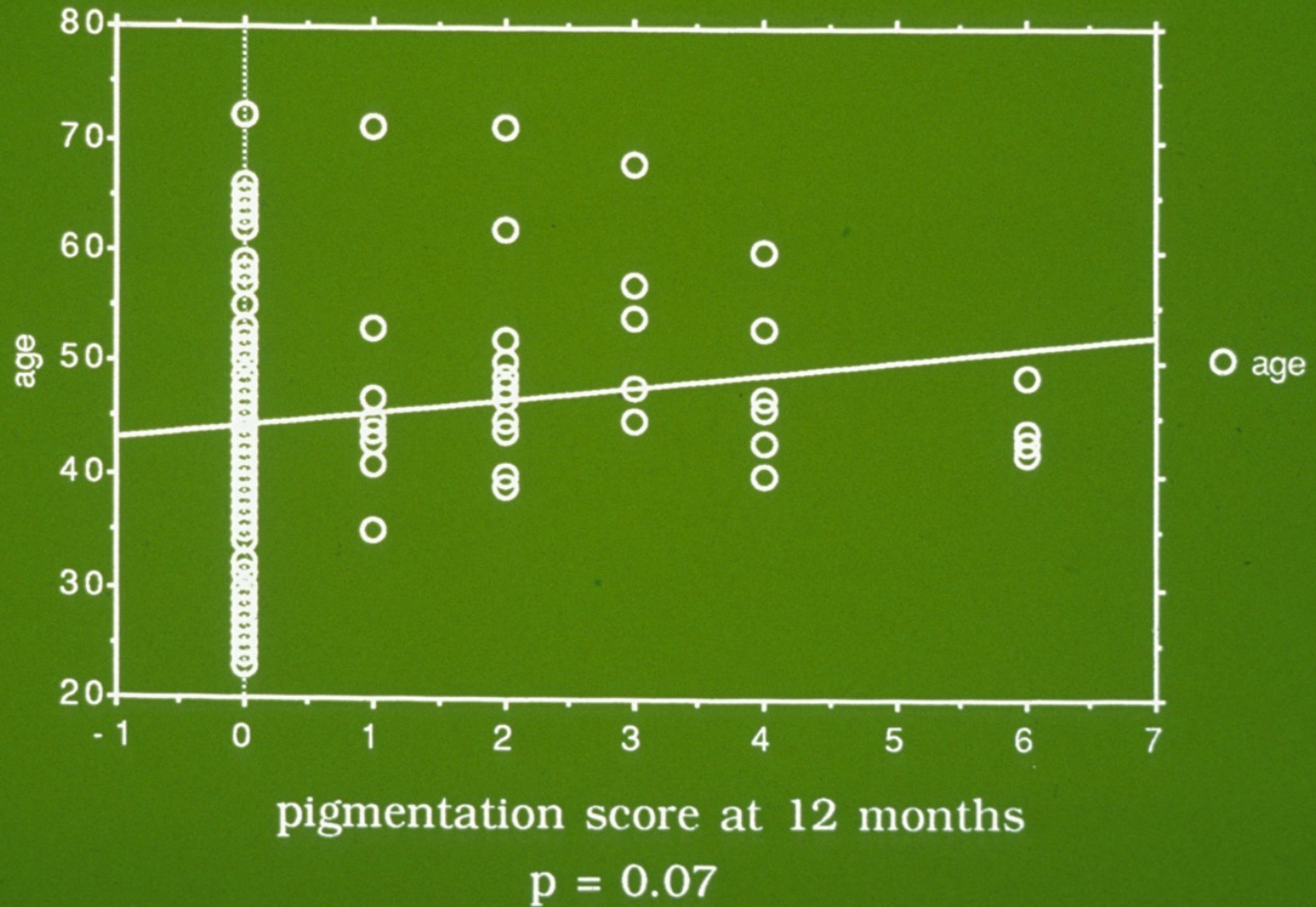
○ $\ln(x)$ of se Fe...

$p = 0.0004$

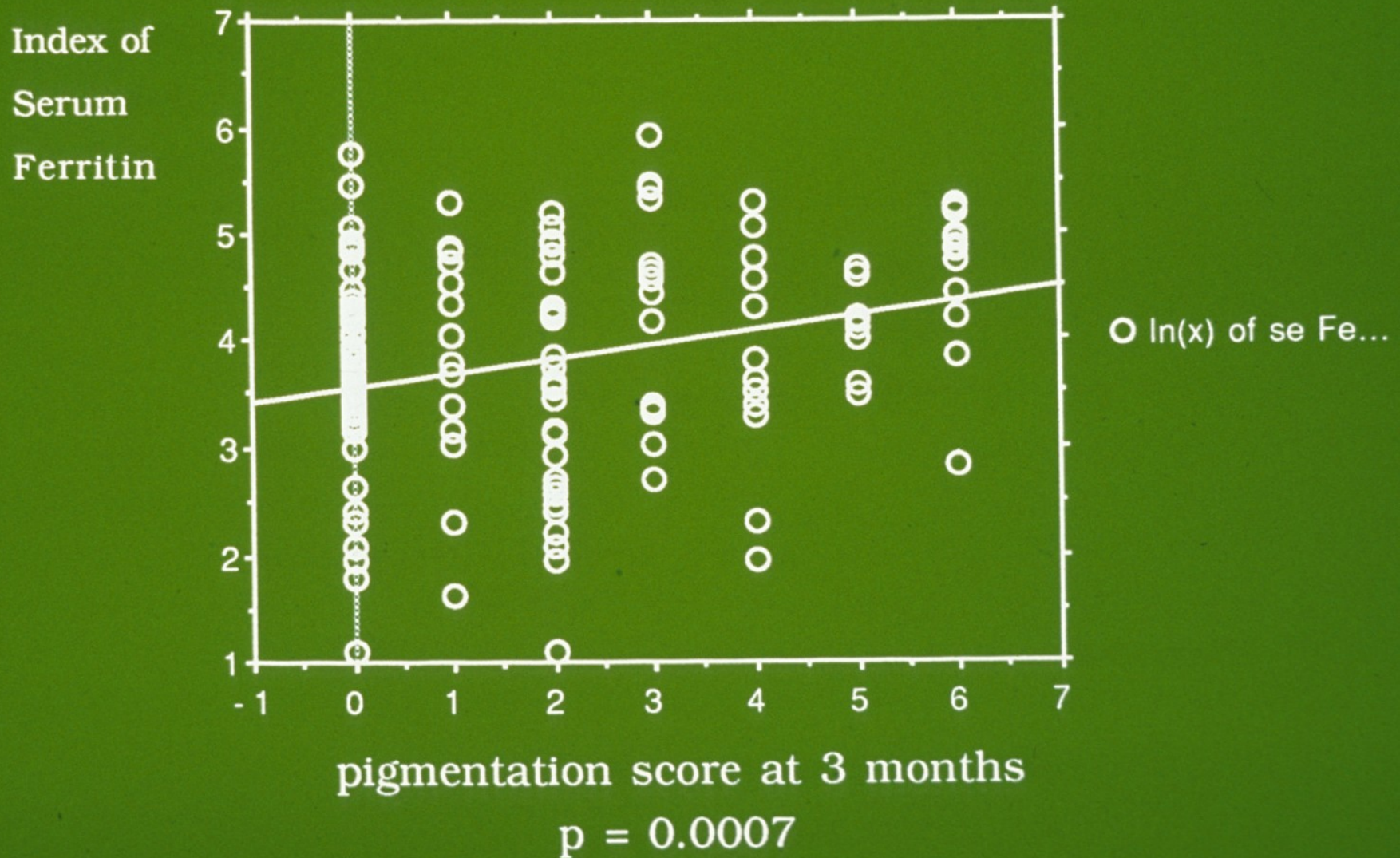
Age vs Pigmentation at 3 months



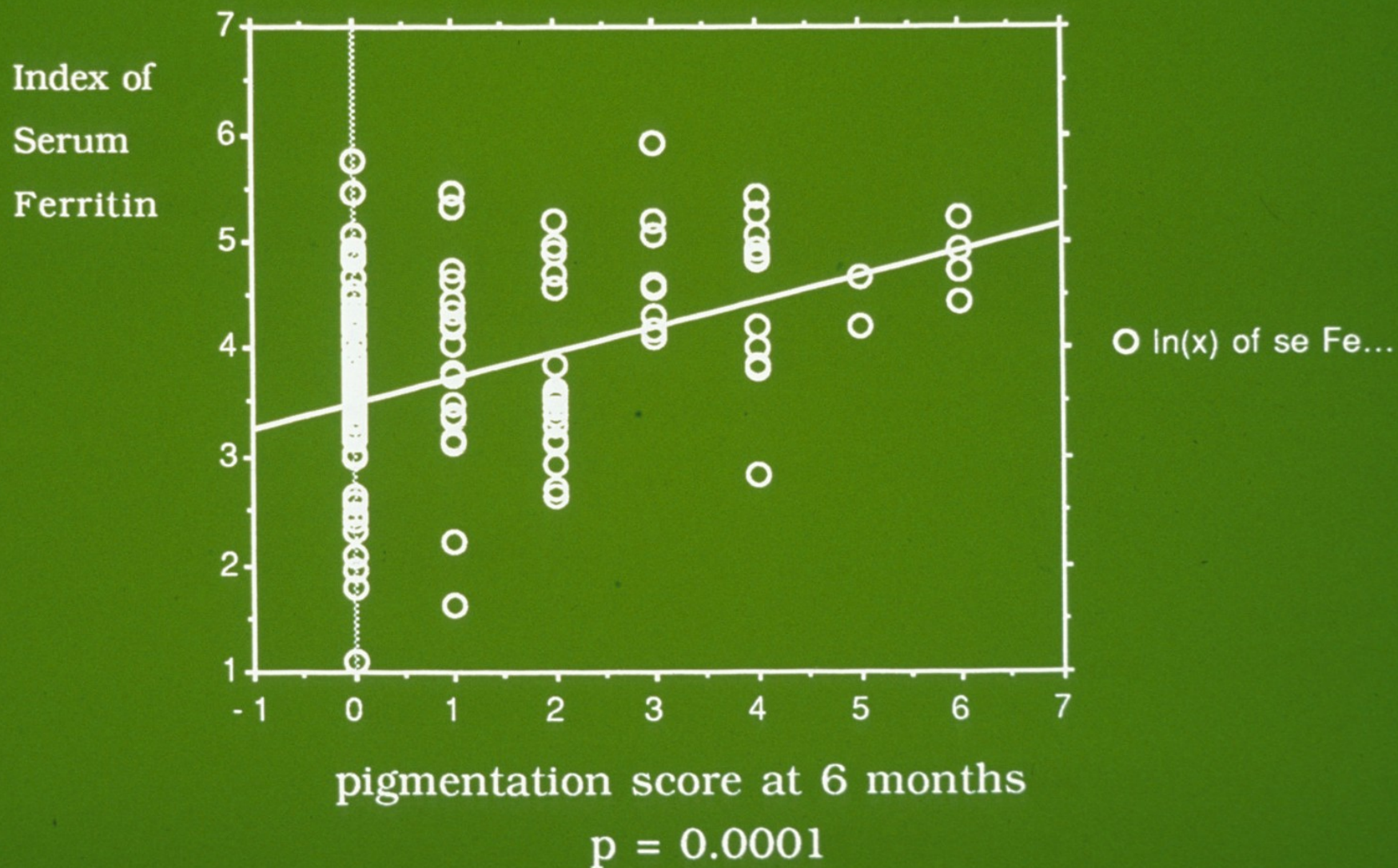
Age vs Pigmentation at 12 months



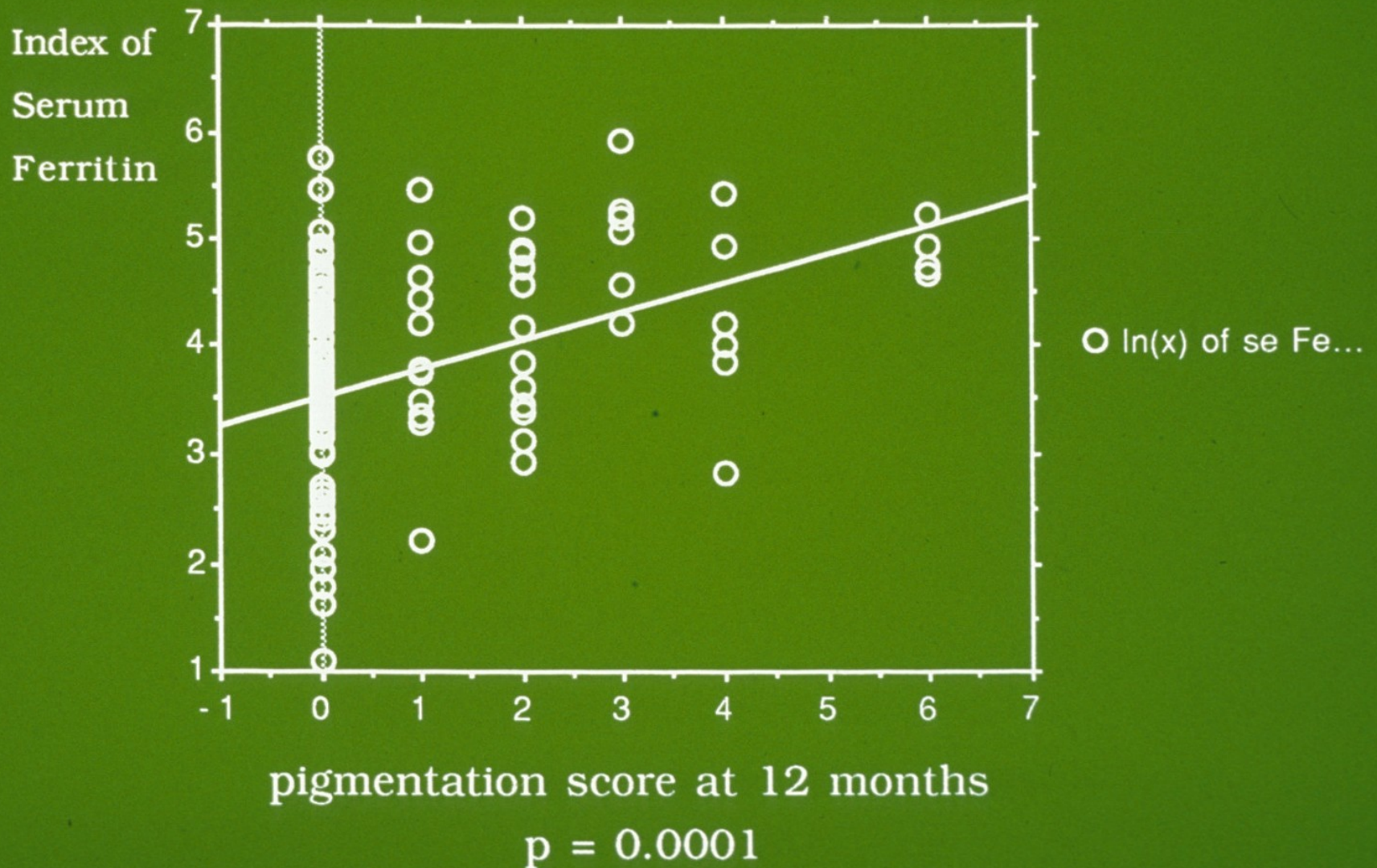
Serum Ferritin vs Pigmentation at 3 months



Serum Ferritin vs Pigmentation at 6 months



Serum Ferritin vs Pigmentation at 12 months



Post Sclerotherapy Pigmentation

The role of Fe

- CONCLUSION: The higher the serum ferritin level, the greater the risk of persistent post-sclerotherapy pigmentation



How to remove Fe from the Skin?



How to remove Fe from the Skin?



Postsclerotherapy Pigmentation: Prevention

- ✓ Avoid iron supplements before, during and for 3 months after treatment
 - ✓ Meticulous technique
 - ✓ Avoid excessive injection pressures
 - ✓ Select appropriate solution and strength
 - ✓ Treat proximal to distal
 - ✓ Evacuation of retained blood and microthrombi
 - ✓ Adequate postsclerotherapy compression
-
-

Compression Bandaging Vs Graduated Compression Stockings

	no. treated	no. success
Graduated compression stockings ۳۵ - ۴۰ mmHg	۱۵۶	۱۴۴
Elastocrepe bandages	۱۴۷	۱۱۷
p < ۰.۰۰۱		

(Scurr JH, Coleridge-Smith P, 1985)

- Superficial thrombophlebitis less in GCS
- Pigmentation less in GCS

Compression in Treatment of Leg Telangiectasia

	Pigmentation
Compression	28.50%
Non - Compression	40.50%

Study by Goldman, Marley, Butie et al. Patients with symmetrical disease. One leg compressed for 72hrs with Class 2 stocking

- Ankle and calf oedema also reduced
- No increase in effectiveness for vessels <0.5mm diam
- No increase in effectiveness in thigh

Post-sclerotherapy Compression Study of Duration

- Weiss, Sadick, Goldman - Dermatologic Surgery 1999
 - Class 1 GCS during daytime only
 - Reticular and spider veins
 - Effectiveness at 6, 12, and 24 weeks
 - Side effects
 - Post-sclerotherapy pigmentation
 - Telangiectatic matting
 - Oedema
 - Ulceration

Post-sclerotherapy Compression Study of Duration - Conclusions

- Effectiveness

- AT 6 WEEKS

- 3 weeks > 1 week > 3 days > None ($p < 0.004$)

- AT 24 WEEKS

- 3 weeks > 1 week > (3 days, None) ($p < 0.006$)

- Side Effects

- POST-SCLEROTHERAPY PIGMENTATION

- (None, 3 days) > 1 week > 3 weeks

- TELANGIECTATIC MATTING, ULCERATION, OEDEMA

- Not significant

Postsclerotherapy Pigmentation: Treatment

- Chemical exfoliants - non specific
 - trichloroacetic acid, glycolic acid
 - Physical exfoliants
 - CO², liquid nitrogen
 - Chelating agents - topical EDTA
 - Laser treatment
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Laser Therapy

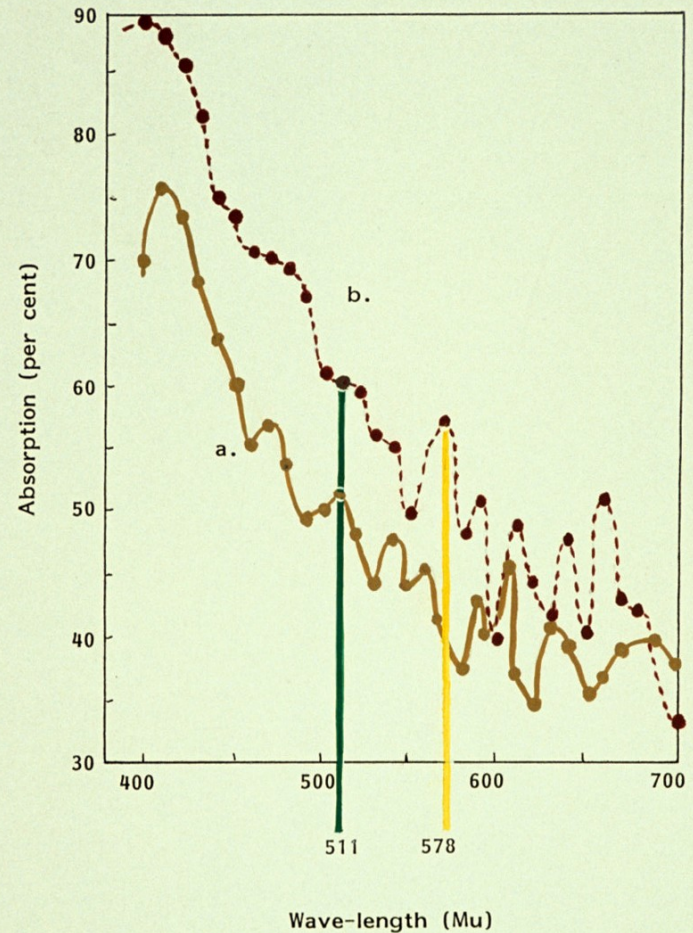


Fig. 3. Absorption spectra of haemosiderin granules.
(a) —●—, Formalin-fixed, lipid-extracted, paraffin-embedded tissue; average of six determinations.
(b) - -●- -, Freshly frozen tissue; average of two determinations.

From NATURE 1962.

Laser Therapy



Chronotherapy or “Flashbulb” Therapy



Telangiectatic Matting

- New vessels < 0.2 mm in diameter
 - Post-sclerotherapy
 - Post-surgical
 - Thigh affected $>$ calf
 - ?Age related
 - ?F $>$ M
-
-

Telangiectatic Matting

- Incidence – 5% - 75% (Goldman)
 - Say 18%
 - 6% will get better spontaneously (inflammatory)
 - 6% will get better with further treatment (related to venous hypertension)
 - 6% is permanent (Duffy)
-
-

Telangiectatic Matting



Telangiectatic Matting

Aetiology

- Patient predisposition
 - Extensive telangiectasias
 - Obesity
 - Oestrogen hormones
- Technique

Telangiectatic Matting: Aetiologic factors

- Angiogenesis - release of angiogenic factors from:
 - damaged endothelial cells (disruption of endothelium)
 - peri-vascular mast cells (peri-vascular inflammation)
 - Arterio-venous anastomoses
 - Persistence of proximal sources of reflux
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Telangiectatic Matting Technique

- Failure to control proximal sources of reflux
- Excessive thrombosis in superficial veins
(Interleukin- 8)



Telangiectatic Matting

Failure to control proximal sources of
reflux



Telangiectatic Matting

Failure to control proximal sources of reflux



Telangiectatic Matting Management

- As for post-sclerotherapy pigmentation
- Look for untreated proximal sources of reflux
- Chronotherapy
- Review and retreat cautiously every 3 months and progress will be made



Telangiectatic Matting: Prevention and Treatment

- ✓ Treat all proximal sources of reflux including reticular veins
 - ✓ Use compression
 - ✓ Withdraw oestrogen therapy if not essential
 - ✓ “chronotherapy” - review every 3 months
 - ✓ Daily walking or other exercise to improve muscle tone
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Post Sclerotherapy Cutaneous Necrosis

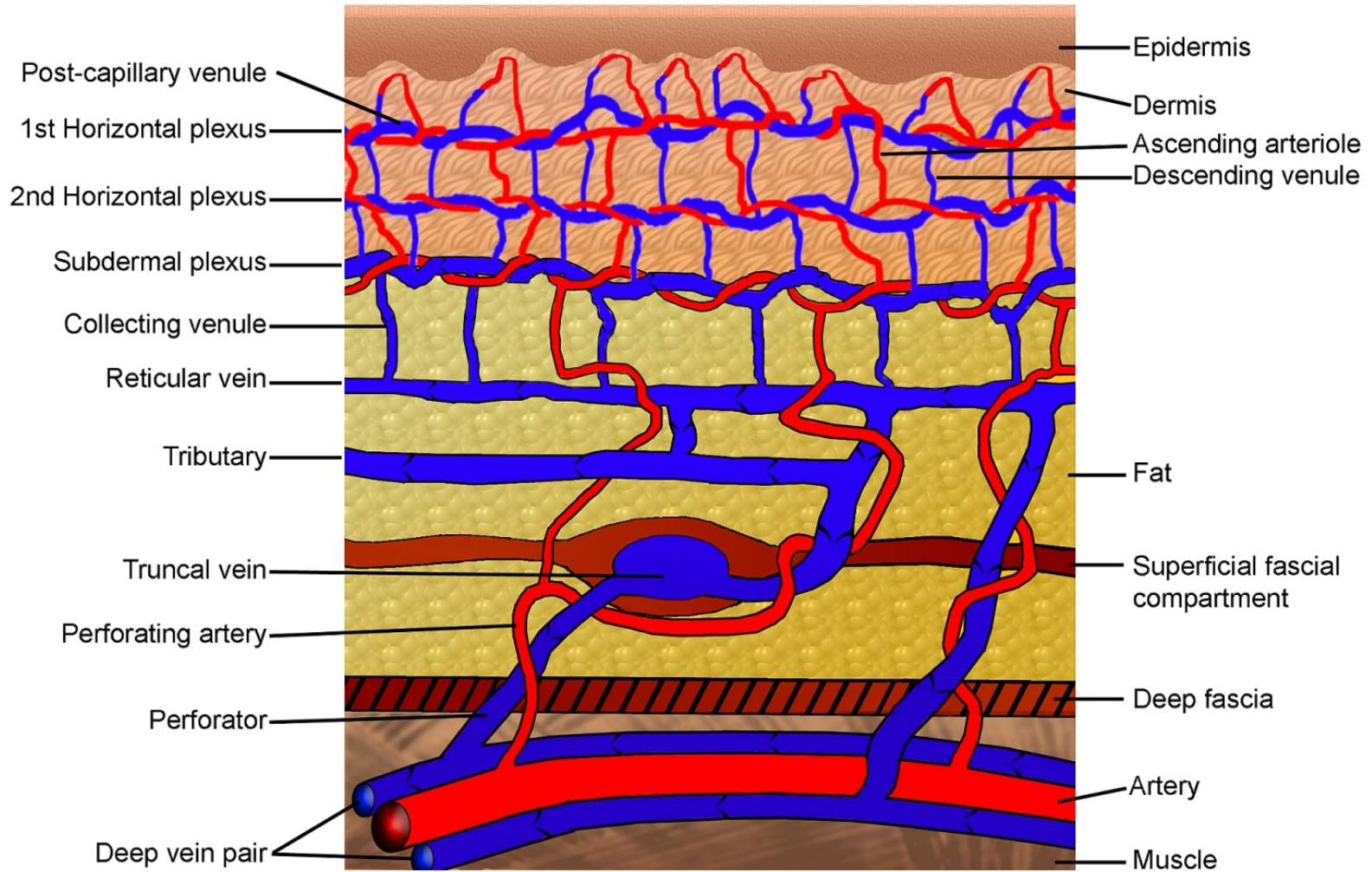


Cutaneous Necrosis

- Most common with hypertonic saline (extravascular injection)
- Less common with detergents – a variety of mechanisms – all ischaemic



Microvasculature



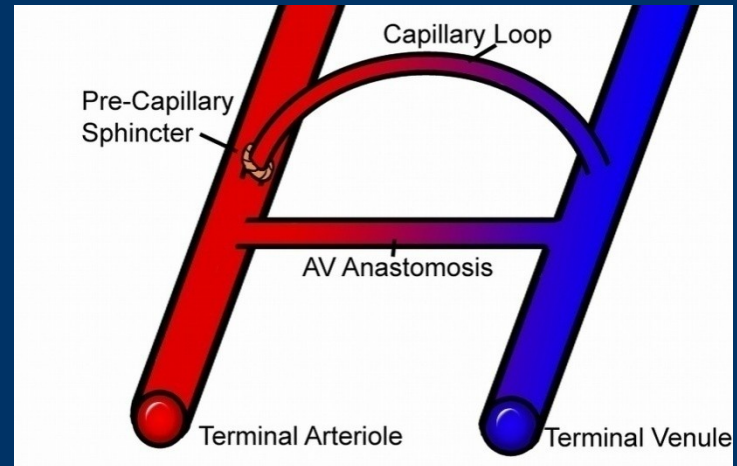
Cutaneous Necrosis

- Direct intra-arterial injection
- Stellate necrosis causing necrosis of large area of skin
- High risk sites
 - Popliteal fossa
 - Medial distal calf (PTPVs)
 - Groin



Veno-arterio Reflex

- Most common cause with detergents
- High venous pressure or sclerosant concentration causes reflex arteriolar spasm
- Cigarette smokers may be at greater risk



Veno-arteriolar Reflex

- Stellate purpura post-sclerotherapy of intersaphenous vein complicated by VAR vasospasm of small saphenous artery



Treatment of Cutaneous Necrosis

- Awareness and early detection
 - Local vasodilator (2% nitroglycerin ointment)
 - Compression
 - Clexane or aspirin
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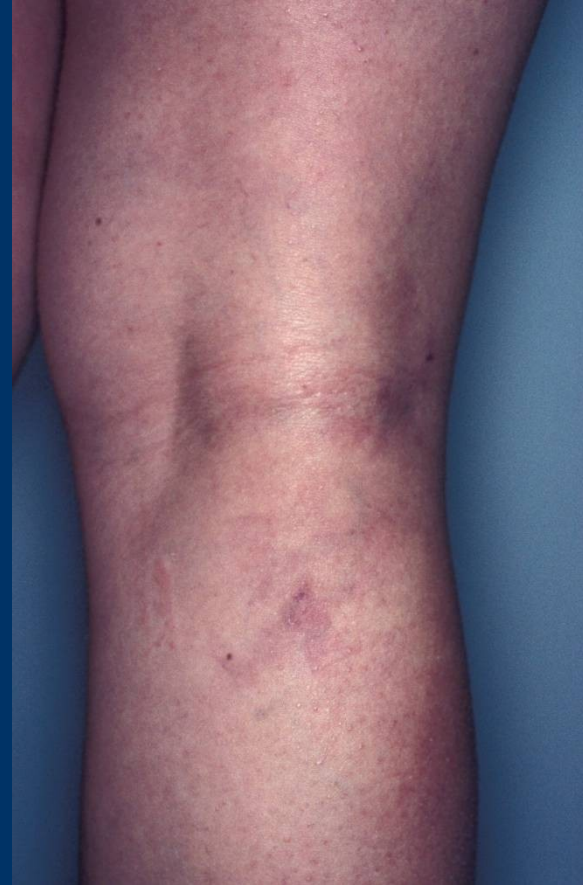
Treatment of Cutaneous Necrosis



Treatment of Cutaneous Necrosis



Treatment of Cutaneous Necrosis



Treatment of Cutaneous Necrosis



Superficial Thrombophlebitis and Chemical Phlebitis

- Common
- Factors
 - Excessive sclerosant concentration
 - Not controlling proximal reflux



Superficial Thrombophlebitis and Chemical Phlebitis Treatment

- Compression
- Walking
- NSAIDs
- Look for proximal reflux and treat



Hypertrichosis

- ?Rare
- Transient
- STS and iodine
- Release of angiogenic cytokines



Infection

- Rare
- May be unusual bacteria such as *Pseudomonas*



Infection

- Differential Diagnosis
 - Thrombophlebitis
 - Intra-arterial injection
- Treatment
 - Appropriate oral or systemic antibiotic

