The Wound Module

Traditionally, electrology experts define overtreatment as "treatment of a hair follicle beyond necessity."[1] Too much treatment, they say, damages surface tissues and produces "unwanted" skin reactions.

Although ambiguous, the traditional definition is proper because we must always try to minimize treatment reactions. However, some electrologists see normal post-treatment skin reactions as "overtreatment." Thus, fear of overtreatment persuades some colleagues to use insufficient current—they tweeze.

Skilled professionals recognize the normal wound-healing process. Furthermore, they know that even carefully performed electrolysis sometimes causes "unwanted" skin reactions, but that most of these reactions are temporary and have no lasting consequences. They are able to visually differentiate between immediate inflammatory skin reactions, and later-forming primary and secondary lesions.

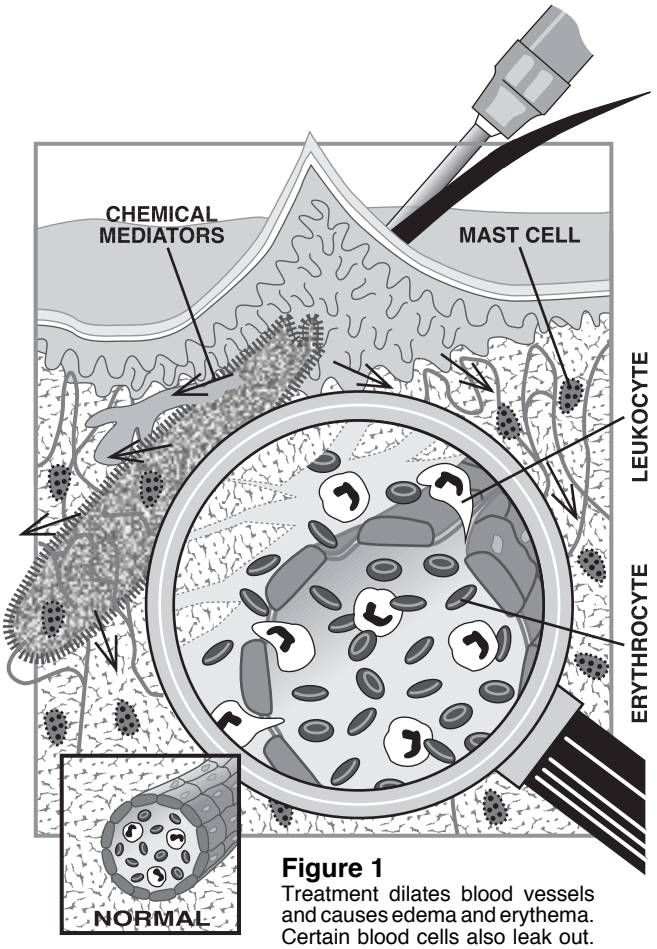


Figure 1
Treatment dilates blood vessels and causes edema and erythema. Certain blood cells also leak out.

THE WOUND-HEALING PROCESS

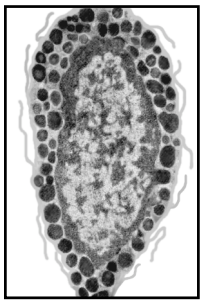
Electrolysis injury, both heat and chemical, causes dermal and epidermal cell membranes to release chemical mediators. These substances activate key responders, such as mast cells, that initiate a cascade of events called inflammation.[2] *Inflammation turns-on the entire wound-healing process!*

Inflammation: You can see and feel inflammation, because this elaborate biochemical process causes tiny blood vessels to dilate and carry more blood. Thus, the skin displays erythema (redness) and feels warm to the touch.[3] The affected blood vessels also become more permeable.[4] As a result, plasma, the liquid part of the blood, leaks out into the spaces between the skin cells.[5] The area swells, and develops a so-called edema (Figure 1).

Mast cells appear responsible for the onset of inflammation and play a vital role in the healing process. These large egg-shaped cells are located throughout the dermis, and are often found assembled around small blood vessels. Uniquely, mast cells are filled with granules that hold various bioactive inflammatory chemicals.

1 Jane Riddle: "International Hair Route." Nov. 1991, p. 9.
2 **Inflammation**: redness, warmth, swelling and pain.
3 Increased blood flow cools the coagulated tissues.

4 **Permeable**: substances able to pass through.
5 Plasma penetrates into the non-cellular spaces in the skin tissues. This is called an interstitial infiltration of plasma.



MAST CELL

When mast cells are stimulated, the granules in the cells disintegrate and release their bioactive chemicals. One chemical, heparin, prevents blood from clotting and speeds the removal of lipids. Serotonin enhances nerve transmission and is a vasoconstrictor. Histamine, a key chemical in wound-healing, promotes blood vessel dilation and permeability. It also stimulates vital elements of the immune system, the body's defense mechanisms, by activating white blood cells called leukocytes.[6]

Leukocytes defend the body from microorganisms that have invaded the tissues or bloodstream. There are several types of leukocytes, each with a specific mission. Certain leukocytes (neutrophils and monocytes) attack, engulf and digest the invading microorganisms with enzymes.[7] All cells that perform this task are labeled phagocytes.[8] The process itself is known as phagocytosis (Figure 2).

As inflammation causes the local blood vessels (capillaries and venules) to stretch, gaps form between the cells of the vessel walls. Leukocytes change shape and squeeze through these tiny openings to enter and defend the tissues (Figure 1).

When skin is injured by electrolysis, microorganisms sometime enter the broken skin barrier. Thus, leukocytes protect the body from infection[9] by harmful microorganisms. However, possible infection is not the only problem, for the body must eliminate the tissues destroyed by electrolysis *and* rebuild the damaged skin.

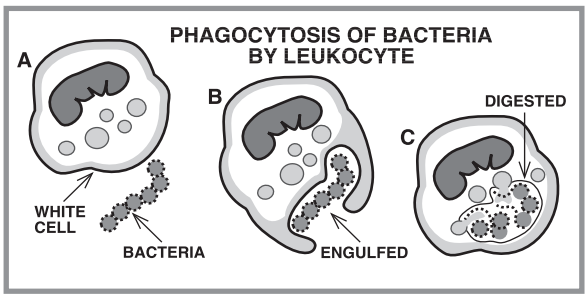
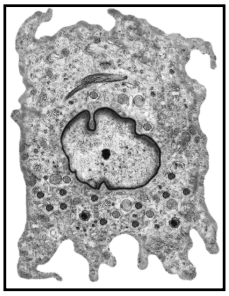


Figure 2
Leukocyte (white blood cell) destroys an invader: A) White cell recognizes bacteria, B) Bacteria surrounded, C) Bacteria destroyed. Cells that perform this process are called phagocytes.

The healing dermis: Although dilated capillaries carry more blood, the blood cannot reach the area destroyed by electrolysis. Vessels surrounding the treated follicle have been coagulated and are thrombosed (clotted). Thus, the denatured mass of cells is deprived of nutrients and oxygen, and is a "dead space."

Miraculously, the surrounding tissues detect the inflammation and lack of oxygen, and wound-healing proceeds. All the repair elements respond together and work as a team to mend the dead space with supple scar tissue.

Initially, leukocytes dominate the wound site. Then, as certain leukocytes, named monocytes, scavenge the tissues, they evolve into super phagocytes called macrophages.[10] Finally, within the second to fifth day, a virtual army of macrophages have amassed around the wound edge of the lifeless follicle.



MACROPHAGE

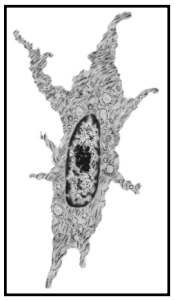
Macrophages are plentiful in the dermis and found throughout the body. These large single-cell phagocytes are mobile and can survive in undernourished

6 Also, lymphocytes activate to carry out immune response.
7 **Neutrophil(s) and monocyte(s)**: leukocytes that protect the body from microorganisms by performing phagocytosis. Such leukocytes, including macrophages, use enzymes to break down organic matter. These enzymes are found in membrane-bound particles called lysosomes, located within the leukocyte.
8 **Phagocyte(s)**: (fag'o-syt) Cells that ingest bacteria, foreign particles and other cells. [Greek: *Phagein:* eating, *kytos:* cell.]

9 **Infection**: Invasion and multiplication of parasitic organisms (or microorganisms) within the body.
10 **Macrophage(s)**: Large roving phagocytes found throughout the body. [Greek: large eating cell, *Macro:* large, *phage:* from "phagocyte."] Within hours after electrolysis injury, macrophages are at work breaking down dead tissue and scavenging for bacteria. At the same time they amass alongside the wound-edge perimeter of the destroyed follicle.

and oxygen-deficient environments. In wound-healing, macrophages convert dead matter into useful substances that the cell then excretes. These chemical mediators attract more macrophages and stimulate blood vessel growth.

Macrophage excreted substances also induce perivascular cells[11] in the wound area to change into collagen-making fibroblast cells. These mobile spindle-shaped cells then replicate and assemble alongside the macrophages.[12] Ultimately, the macrophages break down dead follicular matter into reusable amino acids that the fibroblasts synthesize to produce wound-mending collagen.



FIBROBLAST

At first, fibroblasts make thick gel-like collagen that fills the border of the wound, and perhaps the entire follicle. At the same time, clotted blood vessel nubs sprout "buds" that grow and join with other buds to form new capillary loops. The gel-like collagen supports the newly formed blood vessels that, without support, would burst from arterial pressure. (New vascular growth is called angiogenesis.)

Wound module: By the third to fifth day, all repair elements are working as a *single unit*—the so-called "wound module." Macrophages digest dead matter and transform it into substances that "feed" the fibroblasts. Next, fibroblasts produce gel-like collagen to support the new vascular system that, in turn, supplies oxygen and nutrients to all the repair cells. As blood vessels start bridging the wound gap, fibroblasts resynthesize the gel collagen, and produce fibrous collagen that gives structural support to the new skin (Figure 3).

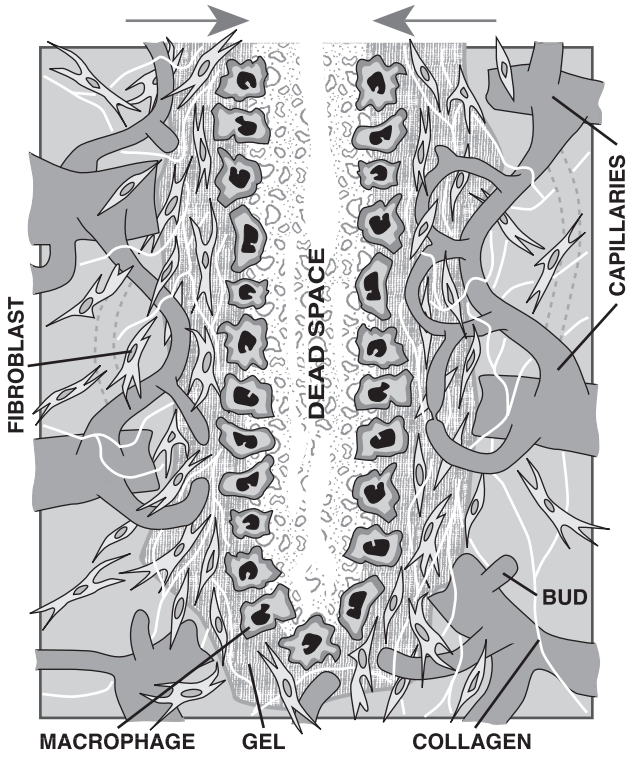
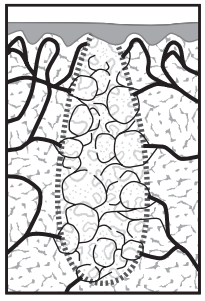


Figure 3
WOUND MODULE: Macrophages and fibroblasts form a phalanx of cells that rebuild the dead space follicle. Vessels bud and form capillary loops that bridge the gap.

The entire wound module progressively moves inward to fill the dead space—all elements depending on each other. The wound module persists until the dead space is bridged, then certain cells are lysed[13] (broken down), while other cells remain in the area.



CAPILLARY LOOPS

Angiogenesis: Blood vessels bridge the dead follicle by sprouting buds that join to form tiny capillary loops. From these loops, new buds form and push into the dead space; again building loops. Thus, the freshly healed follicle is densely populated

11 Specifically, connective tissue cells around blood vessels. There are also resident fibroblasts in the area.
12 Normal process of cell replication is called **mitosis**: the cell divides and produces two identical cells. [Greek: *mitos,* threat.]

13 **Lyse**: To cause lysis. To break up and disintegrate. Common dissolving process used in the body to remove tissues. (Electro-lysis: to disintegrate and break up with DC electricity. Thermo-lysis: to disintegrate and break up with HF heat.)

with minute blood vessels. As wound-healing progresses, the new vascular system continually refines itself. Some blood vessels enlarge to carry more blood, and others disappear. Eventually, the blood vessels are indistinguishable from those in the surrounding tissues.

Sometimes, such as on the fingers or toes, the highly vascularized healing follicles appear as tiny red dots, several days after treatment. As the vascular system becomes remodeled, the red dots disappear; usually in a few weeks. (See photo of fingers on page 197.)

Mending: Fibrous collagen is the principle structural protein of the body, and forms the tough base of the skin. It is also a primary component of bones, tendons, cartilage and *scar tissue*. Collagen is plentiful in the reticular (lower) layer of the dermis. The papillary (upper) layer of the dermis has abundant blood vessels and nerves. Microscopically, collagen fibers are composed of large stiff elongated rod-shaped protein molecules that overlap each other and form long spiraling fibrous strands.

As early as the second day, gel-like collagen fills the dead space follicle, and then is progressively replaced by fibrous collagen. The highest rate of fibrous collagen formation occurs between the fifth and seventh day. This "wound collagen," however, is highly disorganized and structurally inferior to collagen in the uninjured skin (Figure 4).

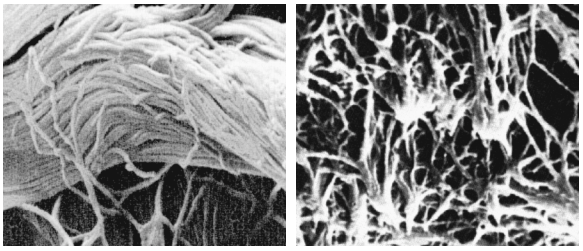


Figure 4
COLLAGEN: **Top**: Dead follicle filled with wound collagen. **Left photo**: Normal skin collagen. **Right photo**: Wound collagen after 10 days. (Photos are courtesy of Thomas K. Hunt, MD.)

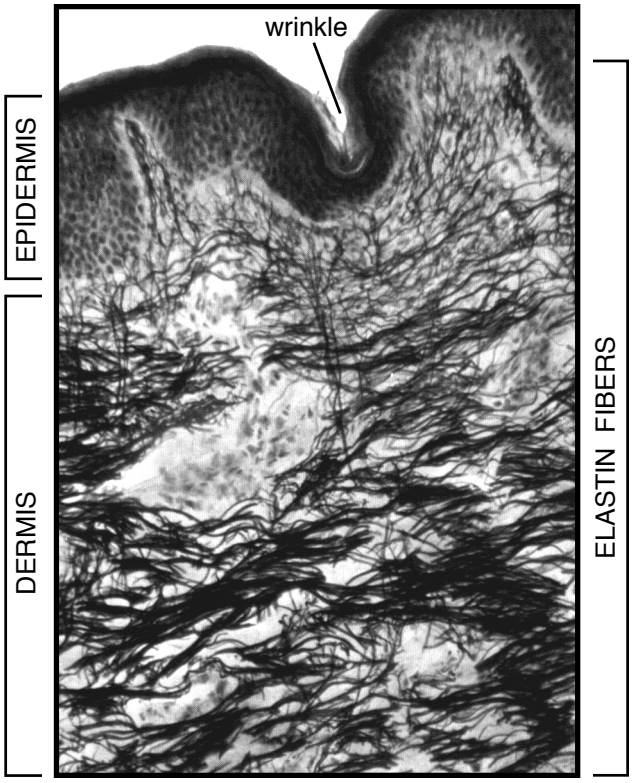


Figure 5
ELASTIN FIBERS are produced by fibroblasts and found throughout the dermis. Although not significant in wound-healing, these rubber-band-like fibers give flexibility to the skin and anchor the epidermis. Elastin fibers also extend into the subdermis and help anchor the skin to the body. They are often entwined with collagen fibers, and add structural support to skin. (Photo courtesy of Wm. Montagna, Ph.D.)

By the third week, the dead space follicle has attained its greatest mass of collagen. Then, the somewhat rigid scar becomes refined. The dense new collagen is continually broken down (lysed) into amino acids and resynthesized into longer, better-shaped collagen strands. Thus, the scar becomes lighter, stronger and more flexible. This ongoing remodeling process, called collagen-turnover, takes place for 6 to 18 months.[14]

14 The collagen formation process is called **collagen synthesis**. The breaking-down process is called **collagen lysis**. Fatty tissues form between fibers and add suppleness to the scar.

The healing epidermis: The epidermis is squamous epithelium: layers of scale-like cells that have no blood vessels.[15] Unlike the dermis that mends with a collagen scar, the epidermis actually *regenerates*.

Clinically, epidermal regeneration is known as epithelization. The process is very efficient for such common wounds as abrasions. Epithelial cells encircling such wounds grow inward, while epithelial cells that form the lining of hair follicles, sebaceous and eccrine glands, generate cells that push upward to rebuild the epidermis. If electrolysis damages the epidermis, epithelization begins within a few hours after injury.

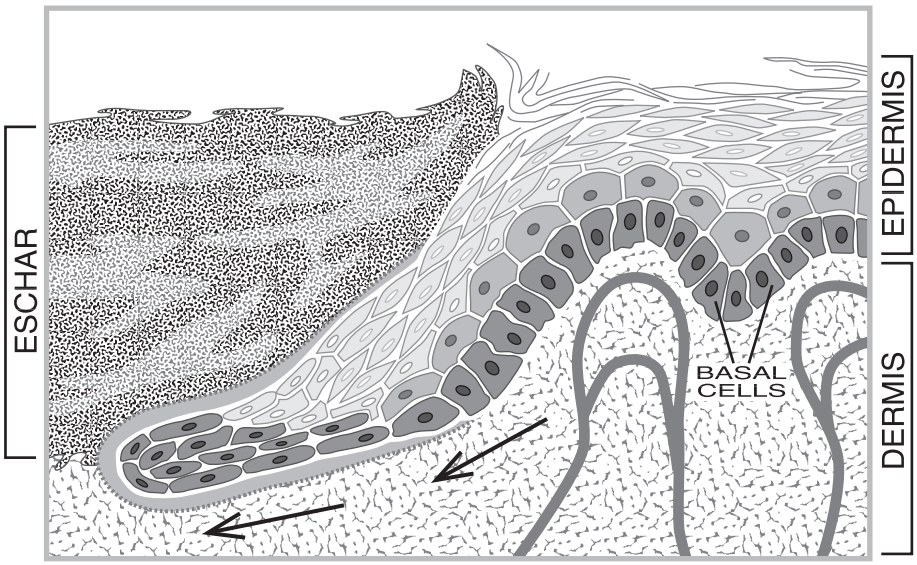


Figure 6
EPITHELIZATION: Basal cells form a wedge or "tongue" that tunnels under the nonviable crust. Enzymes are produced at the leading edge of the wedge that allow the advancing cells to dissolve through the dermis and crust. (Crusts usually form with body-technic. Face-technic on the body rarely produces crusts. Using face-technic, crusts should never appear on the face.)

Electrolysis injury probably nullifies follicle-generated epithelization. Nonetheless, basal cells,[16] at the perimeter of the wound, flatten and point inward toward the coagulated area. As the eschar[17] congeals, the basal cells replicate and climb over each other to form a circular "tongue" of epithelium that actually burrows under the eschar (Figure 6).

Because the epidermis has no independent blood supply, it must fasten to the blood-rich dermis. Thus, the advancing tongue of epithelium releases enzymes that dissolve through the eschar and dermis. In this way, the new epidermis cleaves into the tissues to find the dermal blood supply. In electrology, of course, the dot-like eschar overlies a column of coagulated dermis. Thus, the new epithelium grows inward *with* the healing dermis. However, because the wound gap from electrolysis injury is minuscule, an attenuated layer of epithelium probably spans the wound gap in a day or so. In this case the epithelium utilizes oxygen directly from the atmosphere.

As the wound is bridged, the flat basal cells at the perimeter take on their normal cube-like shape. Then, these cells divide and push replicated cells outward to reconstruct all the layers of the epidermis. The new epidermis is identical to the surrounding epidermis, except it is usually somewhat thinner.

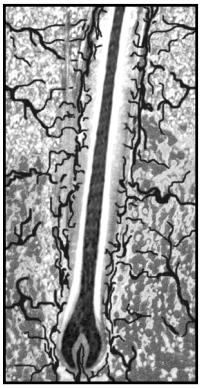
Clinical research shows that although the eschar initially protects the wound from outside infection, it ultimately impedes epidermal healing. The eschar physically retards cell migration and reduces surface oxygen needed for epithelial respiration. Therefore, if the wound is kept clean and moist so that no eschar forms, the epidermis heals rapidly.

15 **Squamous**: Covered with scales. **Epithelium**: Purely cellular with no vascular system. Outer covering of all body surfaces.
16 **Basal Cells**: Cells of the lowest layer of epidermis (basal layer), responsible for growth (germination) of the epidermis.

17 **Eschar**: A crust or scab that develops following thermal or chemical coagulation or desiccation of the skin. [Greek: *eschara.*] Eschar formation indicates cellular destruction of the epidermis. Crusting may also denote damage to the lower tissues.

INFLAMMATION / PRIMARY LESIONS

Various short-term manifestations result from electrolysis. These changes in the skin may simply be part of the inflammation process, or actual injury-caused primary lesions.[18]



CAPILLARY PLEXUS

Micro-hemorrhage: A rich plexus[19] of capillaries lies within the dermal sheath that envelops and feeds the follicle. An inaccurate insertion sometimes pokes through the dermal sheath and ruptures small blood vessels, thus causing a so-called micro-hemorrhage.[20] A tiny droplet of blood may appear at the follicle orifice as the needle path fills with blood. If larger vessels are ruptured, a small bruise may form that indicates subcutaneous bleeding is taking place. These primary lesions are always transient.

A blood droplet denotes a less serious injury, usually caused by poking through the middle or upper follicle. Should you see this injury, withdraw the needle and mop-up the droplet with cotton or tissue. Then, correctly reinsert, treat and epilate the hair, and finish the treatment. Afterward, there should be *no evidence* of the micro-hemorrhage.

A bruise indicates more extensive injury: blood is flowing into the tissues. Larger faster-flowing vessels have been impaled. Bruising is caused by inserting through the follicle wall or by premature tugging on the hair that plunges the needle through the bottom of the follicle. Most needle-stick bruises are less than 5 millimeters in diameter and disappear in 4 to 10 days. Bruising is possible on the eyebrows and other thin-skinned areas that are richly supplied with blood vessels.

Should you see a bruise developing, *immediately stop the treatment* and withdraw the needle. Quickly apply pressure with your finger, for a minute or so, to minimize the size of the bruise. Pressure collapses the blood vessels and reduces the micro-hemorrhage flow while the blood clots.

After the bruise appears stable, do not reinsert or work in the immediate area until the next appointment. Reworking and stretching the skin can open the clotted vessels and enlarge the bruise. Some experts recommend using ice to stop bruise formation. However, by the time you get ice from the freezer, the bruise has already formed. Pressure works much better—if you are quick.

Erythema: The elaborate combination of skin reactions called inflammation, is signified by erythema[21] (red flush). As blood vessels dilate from electrolysis injury, they carry more erythrocytes[22] (red blood cells). Consequently, the affected skin appears florid.

Obviously, erythema is more pronounced in patients with very white skin: the increased blood volume shows through the transparent epidermis. (Red-heads and people that easily blush, often display splendid erythema.) By contrast, patients with darker skin exhibit less erythema. Black patients show no erythema at all, because blood cannot be seen through the dark pigmented epidermis.

18 **Lesion**: A disease or injury-caused change in the skin.
Primary Lesion: The first manifestation seen after injury.
19 **Plexus**: A complex network. (Nerves or blood vessels.)
20 **Micro-hemorrhage**: Minor bleeding, an escape of blood from the vessels. [Greek: *micro:* small, *haima:* blood, *rhegnymi:* to burst forth.]

21 **Erythema**: (er-eh-the'mah) inflammatory redness of the skin. [Greek: *erythro:* red, *ema:* flush.]
22 **Erythrocyte(s)**: (e-ryth'ro-syt) oxygen-carrying red blood cells, corpuscles. [Greek: *erythro:* red, *kytos:* cell.]

Don't be distressed if the patient's skin flushes red. Erythema is a normal reaction to tissue injury. In most individuals, erythema is brief and lasts only an hour or so after treatment. However, some patients get **persistent erythema** that lasts a day or more. Such cases may be the result of overtreatment, or a peculiarity of the patient. If no other complications are seen, there should be no lasting problems.

You may use cataphoresis or ice to reduce post-treatment redness and swelling, but do not use anti-inflammation creams containing hydrocortisone[23] or other steroids. A few electrologists promote the routine use of such drugs, but these are unnecessary. Remember, inflammation activates the entire healing process. *Without inflammation, the skin would not heal*. Do not use anti-inflammation steroids unless medically prescribed.

Edema: Some minor edema[24] (swelling) is a normal sign of post-treatment inflammation. The inflammatory response produces biochemical changes that allow substances to pass through the blood vessel walls. Thus, blood plasma "leaks out" into the tissue spaces and causes edema. In most cases, edema is nominal and lasts only a few hours.

Certain patients, however, immediately develop wheals.[25] This hive-like reaction to electrolysis suggests a hypersensitive histamine response: too much plasma leaks out. Characteristically, these patients experience transient itching at the start of treatment. Histamine, of course, is known to activate itching.

The degrees of erythema, edema and all post-treatment lesions differ greatly between individual patients. Don't expect all patients to respond the same—*they don't!* Furthermore, you must learn to recognize the normal limits of edema and erythema.

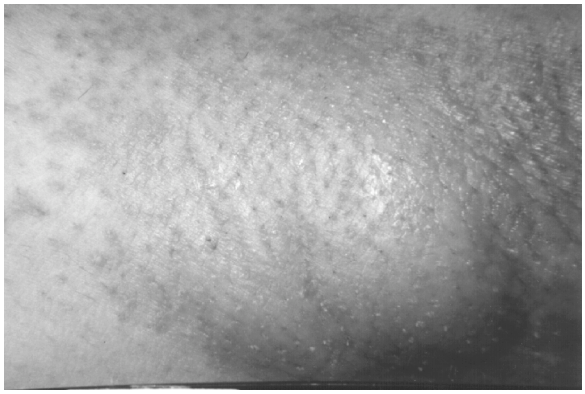


Figure 7
PERSISTENT EDEMA: Two days after treatment, underarm was unusually hard and swollen. There was erythema, but no leaking fluid. Crusting was negligible. Area required one week to recover. Predictably, there were no long-term problems.

Some experts say that DC alone causes this reaction. However, I have found that both DC and HF can cause wheals in sensitive patients. Also, many of these patients report having a history of asthma, various allergies, skin sensitivities and past episodes of hives.

Unfortunately, you cannot minimize wheals in sensitive patients. However, treatment appears to be safe. Patients are seldom concerned, and the edema usually resolves within hours. You may use cataphoresis or ice for marginal reduction of the edema.

In other patients, edema is not immediately seen, but becomes apparent later and lasts for several days. Often, the area is swollen hard, and painful to the touch.[26] Unlike wheals that indicate an unavoidable "hypersensitivity," **persistent edema** often signifies overtreatment. Although unpleasant, there are rarely any lasting complications (Figure 7).

23 Certain creams contain hydrocortisone or other steroids. These drugs inhibit macrophages from entering the wound site. (Such creams may be used immediately after the treatment, but long-term use is not recommended.)
24 **Edema**: Accumulation of excess watery fluid in cells, tissues or cavities. [Greek: *oidema,* a swelling.]
25 **Wheal**: Urticaria, hive, or welt. A circular area of edema of the skin, slightly reddened or lighter in color and causing itching.
26 Sometimes the swollen area is numb to the touch.