

Table 2. Spin-spin coupling constants for fluorides of Xe, Sb, and Te.

Compound	Cycles per second		Reference
	J	J'	
Xe ¹²⁹ F ₄	3836	13040	This work
Xe ¹²⁹ OF ₄	1127	3830	This work
Sb ¹²¹ F ₆ ⁻	1937	7610	(5)
Te ¹²⁵ F ₆	3688	10980	(6)

four fluorines in each of the compounds. This is in agreement with the square planar structure reported for XeF₄ (2) and the fourfold-symmetry axis shown by the Raman and infrared spectra of XeOF₄ (3).

The chemical shifts (δ) for the four compounds are in a logical order; the higher numbers indicate a greater degree of shielding of the F¹⁹ nuclei. The δ values seem to bear closest resemblance to those of the iodine fluorides (4).

Table 2 shows spin-spin coupling constants (J) for fluorides of Xe, Sb, and Te (M). The J values have been adjusted whereby $J' = J_{MF} (\gamma_F/\gamma_M)$, to compensate for the difference in magnetogyric ratios (γ) of the nuclei. It is reasonable to expect XeF₄ to have a larger J value than XeF₆, as is true of

other series of fluorine compounds (6). Assuming a J' value for XeF₆ comparable to that for TeF₆, the J' value for XeF₄ does not seem out of line. Data for compounds analogous to XeOF₄ are not available.

The nuclear magnetic resonance spectra observed so far fail to reveal any major difference between the xenon compounds and compounds of their analogous neighbors in the periodic table (7).

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References and Notes

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Burns and Other Skin Lesions: Microcirculatory Responses in Man during Healing

Abstract. Morphological changes in the cutaneous microcirculation during wound healing have been studied by capillaroscopy in man. In experimental and accidental burns, abrasions, and lacerations, in addition to the anticipated revascularization by the deeper dermal vessels, a distinctive pattern of response by the surrounding papillary capillaries was consistently observed. This appears to be a general phenomenon in healing of skin lesions and suggests the possibility that a potent capillary-attracting factor may be produced within the injured tissue.

Mechanical and thermal injury to the skin or any other vascularized tissue results in profound changes in the minute blood vessels supplying the damaged area. To discern the relationship of these changes either to the specific pathogenesis of the lesion or the generalized reparative response of the human cutaneous vasculature, the reaction of the microcirculation to experimentally produced skin lesions was followed during both the acute and healing stage.

The general structure of such changes can easily be observed by capillaroscopy, and this technique was used to study experimental lesions produced by burn, laceration, and abrasion in the forearm skin of volunteers. It was antic-

ipated that major changes would be seen only within the injured area itself; however, striking and unexpected vascular changes occurred during healing in capillaries in the area surrounding the lesions. These changes proved to be a distinctive, reproducible response which was observed during healing of all the experimental lesions. This response has also been detected in a number of other skin lesions.

Lesions were produced on the ventral surface of the forearm. This area is conveniently studied by capillaroscopy, and the structure of its microcirculation is well-known. The keratin was stripped with cellophane tape to afford a clearer view of dermal vessels, and oil and a

cover slip were applied to the lesions to reduce surface reflection. These lesions were observed microscopically and photographed (black and white film) with a 25 mm microtessar lens (1, 2).

Stripped and unstripped areas of the keratin were burned with a heated platinum wire 1 mm in diameter and several millimeters long. To make the degree of burn roughly constant, we waited 5 seconds after the wire ceased to be visibly red hot before inducing the burn. The depth of the burns was approximately constant and on histological examination the burns were entirely within the upper dermis. Lacerations and abrasions, extending into the superficial dermis and resulting in mild bleeding, were produced with a razor. The lesions were observed closely with the capillaroscope and photographed frequently. Accidental burns and lacerations were observed as well.

The response of the microcirculation after acute injury and the time sequence of changes during healing were similar in all experimental lesions as well as in accidental ones.

A typical sequence in one burn is shown in the illustrations. The area to be burned is marked with ink (corners of Fig. 1a). Ten minutes after application of the heated wire, a coagulum has formed in the area (Fig. 1b). The coagulum darkens and enlarges and forms the usual eschar. At day 3 (Fig. 1c) the area seems to have widened, and a horizontal orientation of the surrounding capillaries appears, though in the figure it is difficult to see because of the eschar. By day 6 (Fig. 1d) the eschar loosens and the horizontal inwardly-directed capillaries are more easily seen. The surrounding pattern is more obvious at day 8 (Fig. 1e), and by day 10 the eschar has come off and the vascular tufts growing up from the deeper dermal vessels into the burned area are revealed (Fig. 1f). The edges of the healing burn are supplied by the surrounding undamaged capillaries that have turned horizontally and have grown a short distance into the margin of the burn. Capillaries farther out from the edge also tilt horizontally, but the slant decreases with distance from the site of the burn. Vessels growing up from below into the center of the burned area continue to branch and appear to sprout new capillaries (Fig. 1g, day 20). The surrounding pattern of horizontally oriented capillary loops disappears slowly over a long period as

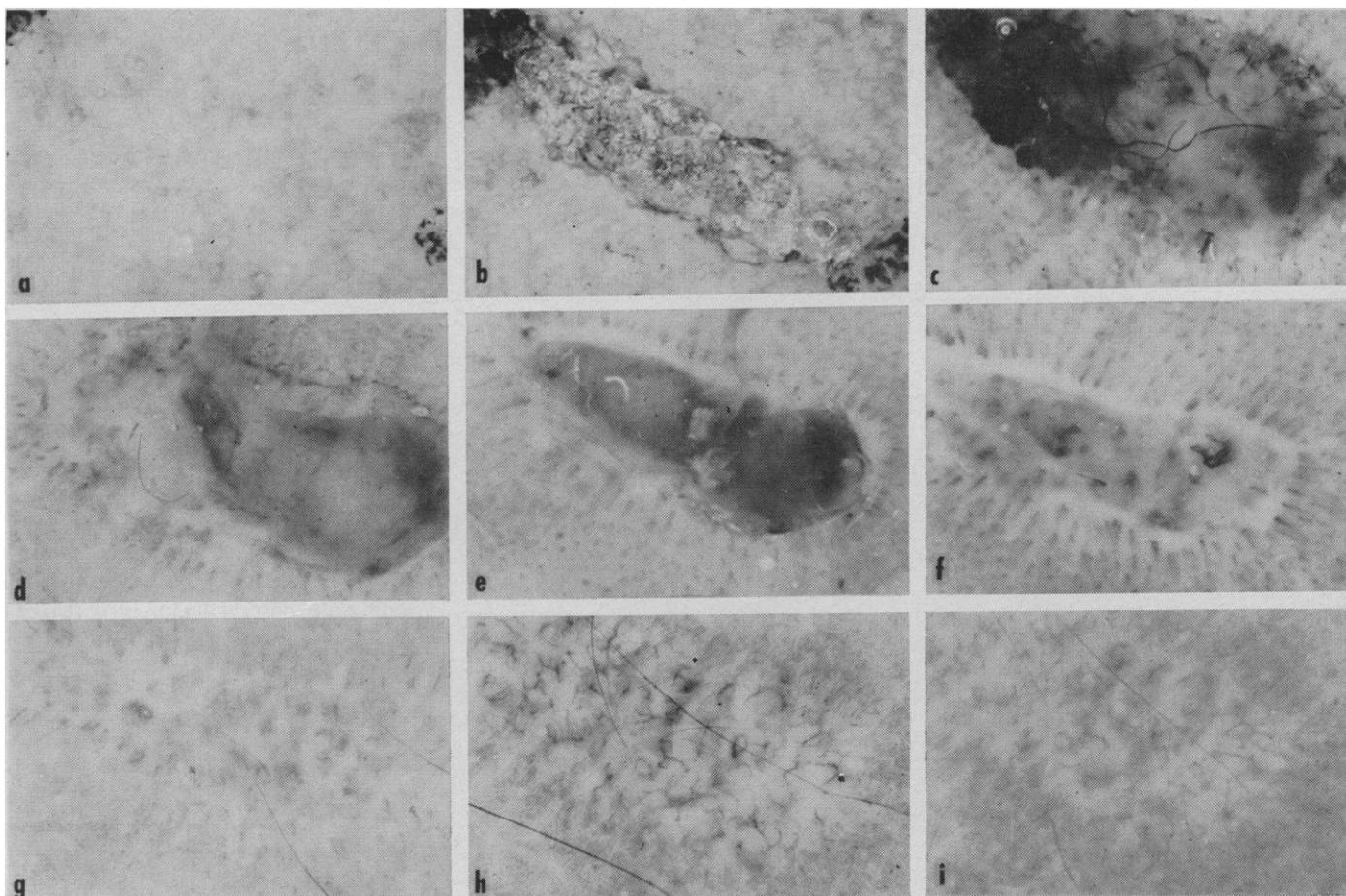
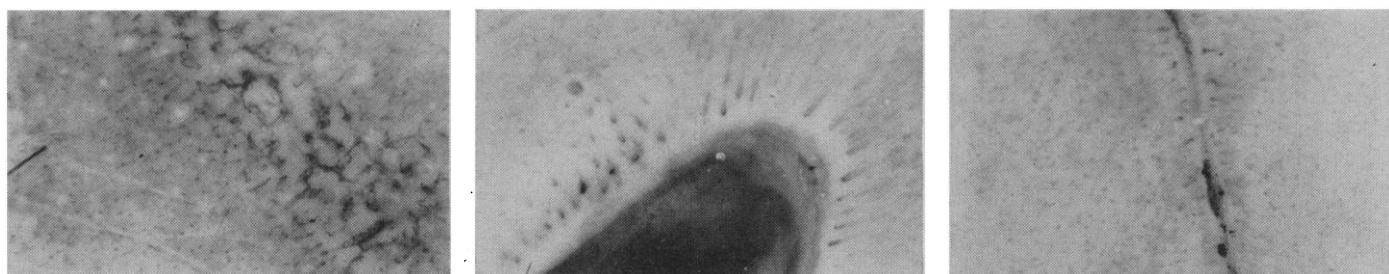


Fig. 1 (all $\times 22$). (a) Area of ventral forearm (from which keratin has been stripped), marked with ink, before burning. (b) Same area, 10 minutes after burn; a coagulum has formed. (c) Day 3: burned area is covered with an eschar and has widened. (d) Day 6: horizontal capillaries directed inwardly are evident around edges of burn. (e) Day 8: surrounding pattern is more obvious. (f) Day 10: eschar has come off and vascular tufts growing up from the deeper dermal vessels beneath the burned tissue are revealed. Edges are supplied by surrounding capillaries. (g) Day 20: further branching of new vessels can be seen within burned area. (h) Day 56: surrounding pattern of horizontally oriented capillary loops slowly disappears as area becomes fully vascularized. (i) Day 99: appearance of the microcirculation returns toward normal.



Figs. 2-4 (all $\times 22$). Fig. 2 (left). An experimental abrasion on day 21: many small loops "bud" from larger vessels within the injured area. Note typical pattern of surrounding capillaries. Fig. 3 (center). Accidental burn, infected, now healing, day 19 after injury. Fig. 4 (right). Accidental cat scratch, very narrow but showing the typical surrounding capillary orientation pattern.

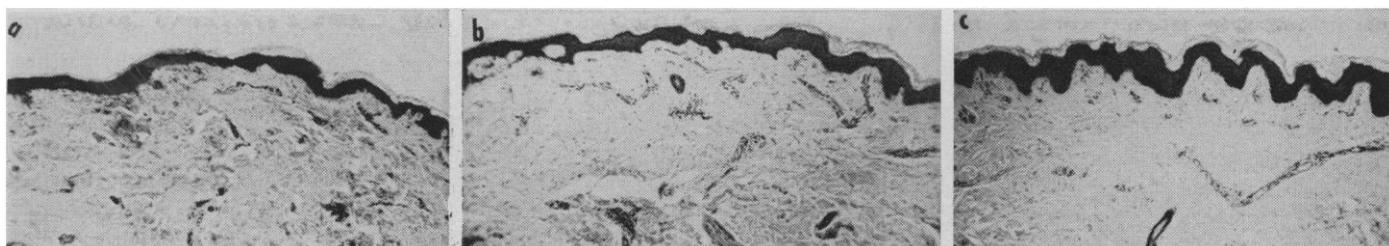


Fig. 5 (a) Histologic section of edge of ventral forearm burn on day 10: small papillary vessels are pointing horizontally toward burned area on left (H and E stain, $\times 27$). (b) Another area at edge of burn, day 10 (H and E stain, $\times 84$). (c) Papillary vessels in normal area of ventral forearm (H and E stain, $\times 84$).

the area becomes fully vascularized from below with a plexiform pattern of small vessels (Fig. 1*h*, the same burn at day 56). Gradually the appearance of the area returns toward normal, but although the vascular pattern is less distinct it is still abnormal at day 99 (Fig. 1*i*). On gross examination after 9 months the area appears as a faint white atrophic scar, yet an abnormality in the microcirculation is evident: the subpapillary vessels are somewhat enlarged and more prominent than in normal skin. Examination of scars from superficial trauma or burns more than 2 years old does not reveal any of these changes which apparently are completely reversible.

In abrasions the same developments were seen (Fig. 2) 21 days after a thin strip of epidermis was removed with a razor. Many small capillary loops "bud" from large deeper vessels within the injured area.

In accidental lesions horizontal orientation of surrounding capillaries was clearly apparent. Figure 3 shows a burn, infected, and now healing at 19 days, and Fig. 4 shows a cat scratch, which, though very narrow, illustrates the typical pattern of the capillaries surrounding the lesion. The same changes were also seen around actively developing keloids.

Histologically, the changes that occur are those to be expected from the *in vivo* pattern. The edge of the healing burn at 10 days (Fig. 5*a, b*) shows small dermal papillary vessels pointing horizontally toward the burned area. The vessels show evidence of active growth, with some proliferation of the endothelium, and are abnormal when compared with the normal vessels in a nearby area (Fig. 5*c*).

Blood vessels grow into and revascularize all healing tissues. In our study, this is seen in the response of the deeper dermal vessels as they grow into the lesions from below. However, the striking reaction of the papillary capillaries surrounding the injured area, particularly those several millimeters from the wound, was unexpected. Since only the vessels immediately adjacent to the wound revascularize its edges and since the vessels farther away do not provide significant revascularization, even though they are inwardly oriented, the possibility of a potent attracting factor was suggested. Such a factor, conceivably a chemical produced by the injured tissue, apparently attracts the capillaries and causes a progressively

more horizontal orientation and growth toward the wound. The potency of this hypothetical factor can be estimated by its ability to influence capillaries even several millimeters away from the lesion. In general, the size of the wound is related to the distance at which capillary changes occur and suggests a diffusible attracting substance (see Figs. 3 and 4).

In burns the surrounding skin may suffer some thermal injury, however, the possibility of such a direct effect can hardly explain the occurrence of the same phenomenon around the more sharply circumscribed lacerations and abrasions. Furthermore, the surrounding pattern of horizontally oriented capillaries pointing toward the lesion does not appear to be a mechanical result of contraction of the wounds, for these clearly widen during the first few days after experimental injury (see Fig. 1*b* and subsequent figures). Sliding of the epidermis over an injured area to provide re-epithelialization is a well-known occurrence; however it cannot be responsible for the capillary changes observed. The epidermis is avascular and the capillaries that are involved are located in the upper corium; hence the epidermis could not pull the vessels mechanically with it. Movement of the corium *en bloc* toward an area of super-

ficial trauma does not seem likely.

This pattern of horizontal orientation of the cutaneous capillaries occurs around lesions in several skin diseases, for example, erythema multiforme and dermatitis herpetiformis (3); psoriasis and lichen planus (4); dermatomyositis, milium, lichen ruber planus, and erythema induratum (5); and also around healing leg ulcers due to venous insufficiency (6). However, the general nature of these changes has gone unrecognized, the pattern apparently having been attributed to the characteristic pathology of the lesions. Our studies suggest that the horizontal orientation of these capillaries is actually part of a normal general response to injury and an attempt to heal the lesion.

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Reovirus and Wound-Tumor Virus: Serological Cross Reactivity

Abstract: *Reovirus and wound-tumor virus share a common antigen capable of fixing complement. This suggests a relationship between these animal-pathogenic and plant-pathogenic viruses.*

The human-pathogenic reoviruses formerly known under the name of ECHO type 10 (1) are very widely distributed throughout the world and have been found among numerous representatives of the animal kingdom, either directly, or through the presence of specific antibodies (1). The plant-pathogenic wound-tumor virus can infect several species of plants in dozens of families (2) and, in addition, can infect at least three related species of agallian leafhoppers that act as biological insect vectors (3). The wound-tumor virus also infects transovarially a low percentage of the offspring of its insect carrier (4).

Reoviruses and wound-tumor virus are very similar morphologically (5). The structure of the wound-tumor capsid is closely similar to that of the

reoviruses capsid, as shown in separate descriptions in the literature (6). The similarity is emphasized by the appearance of the capsid into which phosphotungstate has penetrated. Both viruses contain ribonucleic acid. According to Black and Markham (7), the wound-tumor ribonucleic acid is double-stranded.

In view of the morphologic similarity, it was of interest to study the possible serologic relationship between reoviruses and wound-tumor virus (8). This problem was informally discussed in Montreal in 1962, during a session of the Virus Subcommittee, eighth International Congress of Microbiology. The discussion centered around similarities in virus morphology, serology, and other characteristics as criteria for groups of related viruses. It then be-