matrix components and the denaturation of collagen.[9,10] Closer examination of the literature, however, reveals no agreed-on method for the histologic assessment of burn depth. Research workers have simply classified second-degree burns as "deep" or "superficial" without any reference to the percentage of the dermis involved or exactly which factors have been used to determine the extent of dermal destruction. Chvapil et al.[11] reported that burn depth could be assessed by the presence of heat-denatured collagen staining red (instead of blue) after Masson's trichrome staining. However, we have observed that the thermal energy necessary to denature collagen far exceeds that which may cause vascular occlusion or irreversible cellular damage.[12] Because microvascular flow relates directly to the final depth of the burn, histologic assessment of vascular patency might seem to provide a more reliable index. Vessel damage with subsequent blockage is one of the most obvious signs of dermal injury in burns,[13] and Kahn et al.[14] intimated the importance of assessing the precise level of vascular patency. Future histologic studies should, therefore, devise accurate methods of assessing burn depth based on vascular patency.

We endorse the need for accurate burn depth evaluation to enable optimal treatment of the burn wound. Laser doppler techniques seem to have a high predictive power regarding the need for surgical intervention. Newer scanning laser Doppler technology provides a rapid, noncontact method of assessing large areas of variable burn depth to provide a "surgical map" of the burn wound. Further studies are required to support its emerging use as the new standard for burn depth estimation.

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REFERENCES

1. Boykin, J. V., Eriksson, E., and Pittman, R. N. In vivo microcirculation of a scald burn and the progression of postburn dermal ischemia. *Plast. Reconstr. Surg.* 66: 191, 1980.
2. Zawacki, B. E. Reversal of capillary stasis and prevention of necrosis in burns. *Ann. Surg.* 180: 98, 1974.
3. Jackson, D. M. The diagnosis of the depth of burning. *Br. J. Surg.* 40: 588, 1953.
4. Tyler, M. P. H., Watts, A. M. I., Perry, M. E., Roberts, A. H. N., and McGrouther, D. A. The cellular inflammatory response to cutaneous burns: An insight into the function of the dermal microvascular anatomy. (submitted).
5. Heimbach, D. M., Afromowitz, M. A., Engrav, L. H., Marvin, J. A., and Perry, B. Burn depth assessment—Man or machine. *J. Trauma* 24: 373, 1984.
6. Yeong, E. K., Mann, R., Goldberg, M., Engrav, L., and Heimbach, D. Improved accuracy of burn wound assessment using laser Doppler. *J. Trauma* 40: 956, 1996.
7. Niazi, Z. B., Essex, T. J. H., Papini, R., Scott, D., Mclean, N. R., and Black, M. J. M. New laser doppler scanner, a valuable adjunct in burn depth assessment. *Burns* 19: 485, 1993.
8. Heimbach, D. M., Engrav, L., Grube, B., and Marvin, J. Burn depth: A review. *World J. Surg.* 16: 10, 1992.
9. Sevitt, S. *Burns Pathology and Therapeutic Applications.* London: Butterworth, 1957.
10. de Camara, D. L., Raine, T. J., London, M. D., Robson, M. C., and Heggers, J. P. Progression of thermal injury: A morphological study. *Plast. Reconstr. Surg.* 69: 491, 1982.
11. Chvapil, M., Speer, D. P., Owen, J. A., and Chvapil, R. A. Identification of the depth of burn injury by collagen stainability. *Plast. Reconstr. Surg.* 73: 438, 1984.
12. Watts, A. M. I., Tyler, M. P. H., Perry, M. E., Roberts, A. H. N., and McGrouther, D. A. Burn depth assessment. A new histological method. (submitted).
13. Del Beccaro, E. J., Heggers, J. P., and Robson, M. C. Preventing the prostaglandin effect on dermal ischemia in the burn wound. *Surg. Forum* 24: 603, 1978.
14. Kahn, A. M., McCrady, V. L., and Rosen, V. J. Burn wound biopsy—Multiple uses in patient management. *Scand. J. Plast. Reconstr. Surg.* 13: 53, 1979.

REPLY

Sir:

I would like to thank Dr. Banwell and colleagues for their comments. Requiring multiple "point" readings to assess burned areas correctly cannot be overemphasized. I agree that newer scanning laser Doppler technology using laser Doppler imaging made in England probably provides a rapid, noncontact method of assessing large areas of variable burn depth to provide a "surgical map" of the burn wound. However, I have no idea if laser Doppler imaging can show the exact predictive laser Doppler value for discriminating between the superficial second-degree burn and the deep second-degree burn or between the deep second-degree burn and the third-degree burn.

In our studies, my colleagues and I used laser Doppler flowmetry, made in Sweden (model Periflux 4001, Master, Perimed Company, Sweden), instead of laser Doppler imaging, made in England (Moors Instruments, Axminster, Devon, U.K.), with very satisfactory results. Regarding the biopsy and histology, I agree that biopsy and histology would be the most accurate techniques. However, biopsies leave permanent scars and are expensive, and it is extremely difficult for an experienced pathologist to tell live from denatured collagen and cells. We also believe biopsy and histology have their limitations and should be used as research tools only.

Laser Doppler techniques seem to have a high predictive power regarding the need for surgical intervention. However, even if newer, scanning laser Doppler technology (mentioned above by Banwell et al.) is used, the clinical use of laser Doppler flowmetry remains limited to quiet and collaborative patients, and the cost of laser Doppler is relatively expensive. Further studies are required to develop a more precise, convenient method for burn depth assessment.

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A BURN FROM A PLANT

Sir:

The medical problems of a society change and depend largely on socioeconomic conditions. Although medicine is

an ever-changing science, from time to time, it is possible to observe some applications of folkloric medicine, especially in both underdeveloped and developing countries.

Recently, a patient with a second-degree burn injury on her ankle was admitted to our clinic. We learned that the reason for the burn was a plant. Her neighbors advised her to use it to treat ankle pain. Then suddenly, the patient felt a burning pain even worse than the primary problem and removed the plant dressing. On physical examination, there was a second-degree burn injury located on the right ankle.

FIG. 1. Most of the burn was second degree.

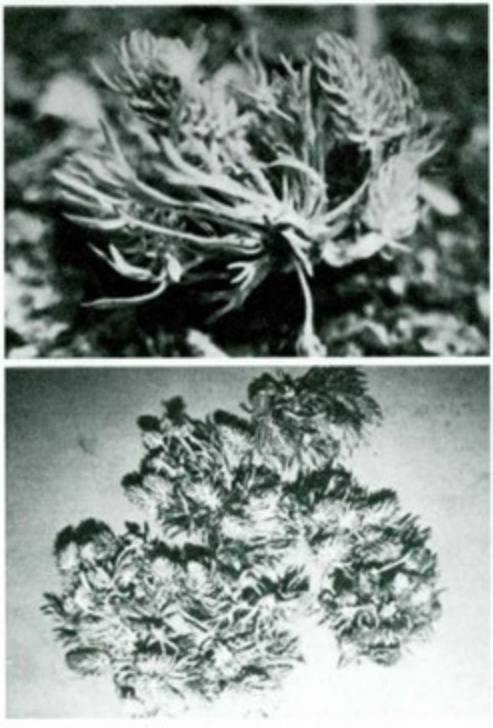

FIG. 2. The plant: *Ceratocephalus falcatus*.

By daily dressing, the burn injury healed completely in 2 weeks (Fig. 1).

When we searched the literature, we realized that the plant used for the ankle pain was *Ceratocephalus falcatus*, a member of the *ranunculaceous* species (Fig. 2). We are unaware of any report in the literature of a burn injury due to a plant that was used as a dressing material.[1–4]

We would like to share this unusual experience with our colleagues.

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REFERENCES

1. Raynaud, J., and Netien, G. The anthocyanic and flavonic glycosides of *Anemone hepatica* L. Ranunculaceae. *Ann. Pharm. Fr.* 29: 449, 1971.
2. Turner, N. J. Counter-irritant and other medicinal uses of plants in Ranunculaceae by native peoples in British Columbia and neighbouring areas. *J. Ethnopharmacol.* 11: 181, 1984.
3. Cammue, B. P., Peeters, B., and Peumans, W. J. Isolation and partial characterization of an N-acetylgalactosamine-specific lectin from winter-aconite (*Eranthis hyemalis*) root tubers. *Biochem. J.* 227: 949, 1985.
4. Nachman, R. J., and Olsen, J. D. Ranunculin: A toxic constituent of the poisonous range plant bur buttercup (*Ceratocephalus testiculatus*). *J. Agric. Food Chem.* 31: 1358, 1983.

IS HEAT SHOCK PROTEIN A POTENTIAL PROTECTIVE MECHANISM AGAINST ISCHEMIA-REPERFUSION INJURY?

Sir:

We read with interest the report by Wang et al. regarding the protective effect of heat-shock protein 72 (HSP72) against ischemia-reperfusion injury in the rat gracilis myocutaneous transplant model (*Plast. Reconstr. Surg.* 101: 776, 1998). We are currently evaluating, using both in vitro and in vivo study models, the protective effect of HSP72 and have some serious reservations about the clinical utility of modulating HSP72 as a protective mechanism against ischemia-reperfusion injury in skeletal muscle.

The ability of HSP72 to provide physiologic protection against ischemia-reperfusion injury has been shown in various organs. The purpose of Wang et al.'s study was to determine if prior induction of HSP72 conferred any degree of myocutaneous protection after ischemia-reperfusion injury.

The authors used a gracilis myocutaneous microsurgical transplant with an ischemic time ranging from 20 to 25 minutes performed 6 hours after heat pretreatment, which resulted in reduced necrosis of the skin paddle, but without any significant difference in muscle viability. First of all, the 25-minute ischemic time used in their study does not accurately reflect ischemic times in replantation surgery, or any free-flap procedures. To induce any protective association with HSP72 in the clinical use of localized hyperthermia based on their experimental findings, we believe, is grounded on a very tenuous foundation.