

casionally, however, hemorrhagic spots were observed in the peripheral nerves especially in the sciatic.

Histologically, the most striking change was in the ganglion cells which showed pycnosis of nuclei, karyorrhexis and subsequent lysis of the cytoplasm. Hemorrhagic suffusions were characteristically located around the damaged ganglion cells (Figure) and a small degree of leucocyte infiltration was also noted. Sympathetic ganglia appeared normal. Other morphological lesions present in the same animals were located respectively at the site of the subcutaneous injection, in the liver, kidney, heart, testes and ovaries.

Preliminary results indicate that the same ganglion lesion can be reproduced in the guinea-pig and the hamster. In similar experiments performed in the rat with the chlorides of mercury, thallium, lead and indium, the ganglionic changes described above were not observed.

Morphological lesions produced by cadmium salts have been described in organs such as the liver, kidney² and

testes³. At present, in relation to our work we have found in the literature only one reference concerning the action of this ion on nervous conduction⁴.

Cadmium is known as a strong inhibitor of sulfhydryl enzymes⁵ and several symptoms of poisoning are consistent with the theory that its toxic effects are due to this property; it seems difficult, however, to explain the selective effect on nervous ganglia on this basis and further studies will be needed to clarify the mechanism of this toxic action.

Résumé. Le chlorure de cadmium produit chez le rat, une nécrose hémorragique sélective du ganglion de Gasser et des ganglions sensitifs spinaux. Dans ces organes, on observe une pycnose des noyaux et une lyse du cytoplasme des cellules nerveuses qui sont entourées d'effusions hémorragiques.

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November 12, 1965.*

Action of CdCl₂ on sensory ganglia

Group	Dose of CdCl ₂ (mg)	% of animals with ganglion necrosis	% mortality
1	5.5	100	100
2	3.5	100	100
3	2.0	100	0
4	0.5	65	0

L. PRODAN, J. ind. Hyg. Toxicol. 14, 174 (1932).

³ J. PARIZEK and Z. ZAHOR, Nature 177, 1036 (1956).

⁴ CH. S. KOSTOJANC, Physiologia bohemoslov. 3, 382 (1954).

⁵ F. P. SIMON, A. M. POTTS, and R. W. GERARD, Archs Biochem. 12, 283 (1947).

PRO EXPERIMENTIS

Infrared Emission Spectra of in vivo Human Skin

The recent observation of IR-emission spectra of various solid surfaces¹ suggested the extension of the emission techniques to the study of biological systems. Exploratory experiments were therefore made to observe the IR-emission spectra of human skin in vivo. As conventional spectrometers were unsuitable, because the amount of radiation emitted at body temperatures was small, an interference spectrometer was used. Such an instrument separates the component frequencies of incident radiation by interference rather than by dispersion. As narrow entrance slits are not required and all of the radiation of interest is incident on the detector simultaneously, the light-gathering power and the signal-to-noise ratio of the interference spectrometer are relatively high. This permits the observation of faint sources.

The Block Model I-4T interferometer spectrometer was used². The instrument and its operation are described elsewhere³⁻⁵. The detector temperature was about 30°C. The spectral region from 2000 cm⁻¹ to 700 cm⁻¹ was scanned repetitiously at a rate of 120 scans per min. The signal of each scan was added digitally and stored in the memory of a Block Coadder, a coherent information adding device. After the accumulation of the individual signals of 360 consecutive scans, the cumulative signal was fed from the Coadder to a wave analyzer coupled with a potentiometer recorder, to result in traces such as those

shown in the Figures 1, 2, and 3. The resolution was 20 cm⁻¹, indicated in each Figure at R. The ordinates of relative spectral emission are arbitrary, and have been shifted to avoid overlapping of spectra. All subjects were fair-skinned Caucasians. The aperture of the spectrometer was situated 2-3 cm from the skin of each subject, so that a skin area of about 3 cm diameter was observed.

Plot A of Figure 1 shows the IR-emission of normal skin near the right elbow of a girl, 'as is', i.e. there was no attempt to clean or especially treat the patch of skin in some way. Spectrum B was then recorded after the same area of skin had been swabbed with acetone. Spectrum B of the whitened, fat-free skin is seen to show different detail than spectrum A. Spectra C-G were obtained from one area of skin near the left elbow of a man. Spectrum C was obtained from the untreated skin. A small amount of oleic acid was then applied, and spectrum D was recorded. The area was then swabbed with acetone, and the skin was abraded with sandpaper. Spectrum E of

¹ M. J. D. Low and H. INOUE, Analyt. Chem. 36, 2397 (1964); Can. J. Chem. 43, 2047 (1965).

² Block Engineering Inc., Cambridge, Mass. 02139, USA.

³ M. J. PERSKY, Atmospheric Infrared Optics-Flux Measurements (AFCRL, 1963), p. 63.

⁴ L. C. BLOCK and A. S. ZACHOR, Appl. Optics 3, 209 (1964).

⁵ M. J. D. Low and I. COLEMAN, Spectrochim. Acta, in press.

the abraded skin was then recorded. About 12 h after the abrasion, numerous small and tightly-adhering crusts about $50\ \mu$ in diameter had formed and dotted the rough and slightly inflamed skin. Spectrum F records the emission of that skin. The sensitive skin was then swabbed with acetone, and spectrum G was recorded. The subject

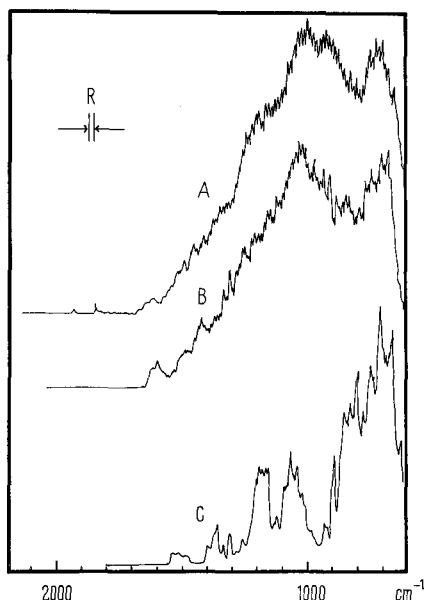


Fig. 1. Emission spectra of normal skin. (Ordinate: relative spectral emission; no abscissa.) A, normal skin of a girl, as is; B, the same skin after washing with acetone; C, normal skin of man I.

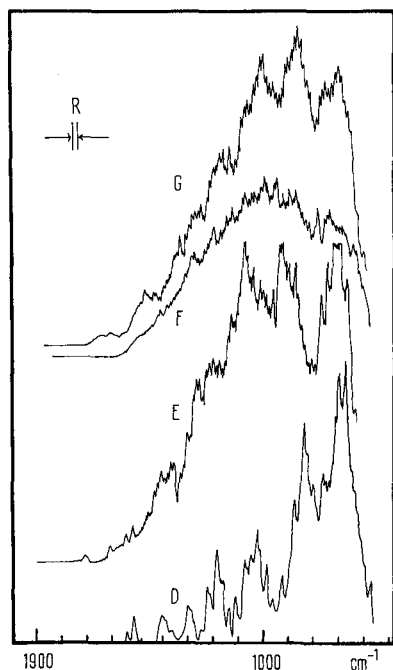


Fig. 2. Emission spectra of damaged skin. (Ordinate: relative spectral emission; no abscissa.) D, skin of man I after application of oleic acid; E, after washing with acetone and abrading; F, 12 h after abrasion; G, after washing abraded area with acetone.

of the spectra of Figure 3 was a man afflicted with acute psoriasis. Spectrum H was obtained from an area of unaffected skin inside the right forearm. Spectra J, K, and L were taken from one area on the back of the right hand, which was severely affected by psoriasis. The skin was taut, shiny, apparently dry and without scabs or scales, and was a livid scarlet. The subject had applied a petroleum jelly ointment to the hand some 2 h prior to the recording of the first spectrum J. The latter shows the emission of the otherwise untreated skin affected by psoriasis. After spectrum J had been measured, the hand was washed with soap, was well flushed with water, and dried. Spectrum K was then measured. A small amount of the same ointment was then rubbed onto the skin, and spectrum L was obtained.

The resolution shown is relatively poor, but this is more than offset by the ability to observe the spectra. Resolution with the present instrumentation is more than adequate to indicate that marked differences in band positions and relative band intensities are observable in the spectra of different samples of skin. There are differences in the spectra of normal skins of different subjects, shown by comparison of plots A and B with C and H. Also, marked structural variations are shown by the spectra of normal and damaged skin of the same subject, as indicated by the sequences C to G and H to L. The band structures of the various spectra have not been interpreted, and it is not known what contribution to the emission were made by the skin per se and by natural secretions or foreign substances on the skin surface. Also, it is not known to what extent emission intensities were affected by differences in skin texture. The spectra are thus qualitative. These exploratory results do show, however, the feasibility of the present approach to measuring the spectral distribution of IR-radiation from human skin in vivo by means of multiple-scan interferometry. This, and the versatility and sensitivity of the

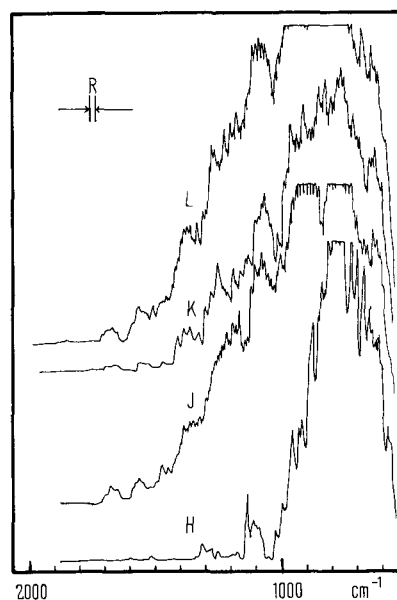


Fig. 3. Emission spectra of psoriasis-infected skin. (Ordinate: relative spectral emission; no abscissa.) H, unaffected area of skin of man II; J, skin affected by psoriasis, as is; K, after washing; L, after ointment application.

instrument, suggest that the development of the technique for the non-destructive examination of skin would be fruitful for studies of pigmentation, the nature of infected areas, the presence or absence of secretions, and similar effects⁶.

⁶ This work was supported by Public Health Service Grant No. APO0211-01A2. The cooperation of the personnel of Block Engineering Inc. is gratefully acknowledged.

Zusammenfassung. Es wird ein Instrument auf Basis der Interferenzmethode, das häufig wiederholte IR-Spektren summieren kann, entwickelt und zur Spektralbeobachtung der IR-Emission der menschlichen Haut benutzt.

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A Technique of Integumental Grafting for Aging Studies

Skin transplantation as a tool in aging studies has been employed in the past^{1,2}. The inherent disadvantages in the conventional skin grafting methods are: a temporary state of ischemia prior to vascularization of the transplant, an upper limit in the size of the graft in order to avoid necrosis, and the requirement for a careful preparation of the graft bed³. The result is that grafts large enough for multiple biochemical analyses are difficult to obtain and the effect of temporary ischemia on their chemical composition enters as a variable.

We are reporting on a technique, which allows an exchange of integumental grafts between old and young rats and in which the above-mentioned problems with the more conventional grafting techniques can be avoided. It consists of a 2-step procedure, the first being the establishment of a parabiotic union which is later followed by separation.

Female rats of the Fischer strain were used. The grafting process was usually carried out between a young (6 months old) and an old (18 months old) animal. Surgery was performed under chloral hydrate anesthesia (270 mg/kg i.p.). Following the removal of the hair a lateral incision was made in both participants and corresponding edges were united with surgical clips. The more intimate union of the conventional parabiotic techniques proved definitely detrimental and should be used only if rotation of the animals and the resulting tension on the skin is to be avoided. The success of this method depends on the administration of antibiotics. Each animal received 60,000 U of long-acting penicillin (Longicil - Fort Dodge Laboratories) by i.m. injection. Clips were usually removed after 12-14 days and the animals were thereafter left to themselves up to 2 months. While separation can be accomplished earlier, delaying this step seems to increase the chances for the survival of larger grafts. Figure 1 shows a parabiotic pair to be separated.

In the second step a pair of incisions are made under chloral hydrate anesthesia in the dorsal and ventral layers of the skin connecting the animals. The incisions curve away from each other and from the original suture line in the direction of parabionts. Each animal is thereby left with an open surface to be covered with the now completely healed flap derived from its parabiotic pair. As a result one of the animals is covered ventrally and the other dorsally, with respect to the original suture line. In this step the cut surfaces are brought into apposition and the wound is closed with individual stitches. This gives a larger intact graft area than closure with clips would allow.

Figure 2 shows the appearance of the previous pair after separation. At this stage the same dose of antibiotic as before is administered again. Stitches are either removed later or are lost spontaneously during the healing process. The final appearance of a separated and completely healed pair is shown in Figure 3.

An outline of this method omitting experimental details appeared in 1964 as a news release⁴. Some time later a Russian team⁵ reported on a technique very similar in



Fig. 1. Parabiotic pair made ready for separation.

¹ G. BRAND, P. J. BAKER, W. L. BEAM, and P. HAGEMAN, *Fedn. Proc. Am. Soc. exp. Biol.* 2, 200 (1964).

² P. L. KROHN, *Proc. R. Soc., London* 157, 128 (1962).

³ R. E. BILLINGHAM, in *Transplantation of Tissues and Cells* (Eds., R. E. BILLINGHAM and W. K. SILVERS; The Wistar Institute Press, Philadelphia 1961), p. 9.

⁴ G. C. RING, *Med. Trib.* 5, 11 (1964).

⁵ A. G. LAPCHINSKY, G. V. MEDVEDEVA, I. D. GADALINA, V. I. SUSLIKOV, and A. G. EINGHORN, *Ann. N.Y. Acad. Sci.* 120, 435 (1964).