This effect is much the same as that obtained with the bile and sodium α-glycohyodesoxycholate solutions although the bile inhibition occurred somewhat more consistently.

If the stomach happens to be in a quiescent phase, the production of contractions might conceivably explain the prompt hunger sensations and relief of anorexia seen clinically.

The mechanism is not clearly understood as yet. Whether the response occurs because of an irritative phenomenon on the mucosa of the stomach or because of an effect on the intrinsic nerves cannot be stated at present.

11237 P

Attempts to Demonstrate Poliomyelitis Virus in Extraneural Tissues.*

J. EMERSON KEMPF AND MALCOLM H. SOULE.

From the Hygienic Laboratory, University of Michigan, Ann Arbor.

The recovery of the virus of poliomyelitis from nasopharyngeal washings[1] and the recent positive results with sewage,[2] stools of poliomyelitis patients[1, 3, 4] and healthy carriers[5, 6] has again raised the question of virus distribution in tissues other than those of the CNS. Studies with experimental animals have demonstrated the presence of viruses such as rabies[7] and poliomyelitis[8–12] in the extraneural tissues following intracerebral or intravenous injections. In the present work an attempt was made to recover the virus of polio-

* This work was aided by a grant from the Clara Ward Seabury Clinic for Infantile Paralysis.

1 Kramer, S. D., Hoskwith, B., and Grossmann, L. H., *J. Exp. Med.*, 1939, **69**, 49.

2 Paul, J. R., Trask, J. D., and Gard, S., *J. Bact.*, 1940, **39**, 63.

3 Howe, H. A., and Bodian, D., PROC. SOC. EXP. BIOL. AND MED., 1939, **41**, 538.

4 Kempf, J. E., and Soule, M. H., unpublished.

5 Kramer, S. D., Gillian, A. G., and Molner, J. G., *Public Health Reports*, 1939, **54**, 1914.

6 Lépine, P., *Intern. Bull. for Economics and Med. Res. and Pub. Hyg.*, 1939-1940, **A40**, 57.

7 Lee, J. S., Unpub. Thesis, Univ. of Mich., 1938.

8 Landsteiner, K., and Levaditi, C., *Compt. rend. Acad. d. sc.*, 1909, **149**, 1014.

9 Flexner, S., and Amoss, H. L., *J. Exp. Med.*, 1914, **20**, 249.

10 Römer, P. H., and Joseph, K., *München. med. Wchnschr.*, 1910, **57**, 1059.

11 Leiner, C., and von Wiesner, R., *Wien. klin. Wchnschr.*, 1910, **23**, 817.

12 Kling, C., Levaditi, C., and Lépine, P., *Bull. Acad. de med. Paris*, 3.S., 1929, **102**, 158.

TABLE I.
Distribution of Poliomyelitis Virus in Human Tissues.‡

Patient No.	Tissues tested	Neurological signs in monkeys		Pathology in monkeys
		First passage	Second passage	
A-2130	Spleen	Paresis lt. arm and leg—5 days	2 unsuccessful attempts	Small abscess at inoculation site. No microscopic lesion in spinal cord.
	Liver	Negative	*	†
A-2131	Spleen	,,	*	†
	Liver	,,	*	†
A-2136	Spleen	Flaccid paralysis post. extremity 5 days	1 unsuccessful attempt	Small abscess at inoculation site. No microscopic lesion in spinal cord.
	Liver	Negative	*	†
A-2139	Spleen	Brain abscess	*	†
	Liver	Negative	*	†
	Mesenteric nodes	,,	*	†
A-2141	Spleen	,,	*	†
	Liver	,,	*	†
	Mesenteric nodes	,,	*	†
A-2162	Spleen	,,	*	†
	Liver	,,	*	†
	Mesenteric nodes	,,	*	†

* Second passage not attempted.
† No pathological examination made.
‡ We are indebted for autopsy tissues to the Pathology Department of Herman Kiefer Hospital, Detroit, Mich., Joseph A. Kasper, Director.

myelitis from the tissues of a monkey injected intravenously 5 days earlier with the Aycock strain. The animal, which showed no neurological signs, was exsanguinated and perfused with 1 liter of 0.85% saline solution. Lung, liver, spleen, intestine, brain and spinal cord were removed, ground separately, and diluted with saline. Anesthetic ether was added to a final concentration of 20%, and the suspensions were stored overnight at 5°C. The supernatant fluids were decanted, reduced *in vacuo* to one-third their original volume, and 1.5 cc of each extract inoculated intracranially into *Macacus rhesus* monkeys. The liver and spleen extracts were pooled previous to injection. The animals which received this material and the extract of lung developed quadriplegia in 9 and 24 days respectively. The other animals remained normal. The histopathology in both cases was that of acute poliomyelitis. Second transfers were successful.

Before this work could be evaluated, an unusual amount of tissue became available from human autopsies.† This material was subjected to similar treatment and injected intracranially into monkeys (Table I). The monkeys receiving spleen extracts A-2130 and A-2136 developed neurological signs, but the histopathology and monkey transfers were negative. Small pyogenic abscesses were present in both animals at the site of inoculation in the frontal region and may have caused the flaccid paralysis.[13]

Conclusions. The technic described, while similar to that used with fair results for the isolation of the virus from stools and nasopharyngeal washings,[1] failed to detect the agent in extraneural tissues of poliomyelitis patients. Lennette[14] has suggested that the virus may be present outside the CNS, but unidentifiable because of an attachment with antibody. Definite conclusions regarding the presence or absence of virus in extraneural tissues must await the development of more precise methods.

† From the Detroit epidemic, July through October, 1939.

[13] Aring, C. D., *Arch. Neurol. and Psych.*, 1940, **43**, 302.

[14] Lennette, E. H., *J. Exp. Med.*, 1937, **66**, 549.