of acid gastric juice with a minor erosion resulted in the formation of a peptic ulcer. The application of strong acid to the inflamed and eroded mucosa did not produce pain, although it might have done so with more intense inflammation and a further lowering of the pain threshold.

In the final formulation of the work with its clinical implications the authors consider the objective in the management of patients with gastritis and ulcer to be clearly that of preventing or controlling gastric hyperfunction, the problem thus resolving itself into the care of the man rather than his stomach. "It is to be hoped that factors of emotional conflict, on the one hand, and security on the other will be more directly dealt with in the future. . . . Dealing actively with the patient's life situation and his reactions to it may then be adequately judged as a means for the control of 'dyspepsia,' gastritis and peptic ulcer." The reviewer does not question the importance of this approach, but he is not persuaded that adequate care of the diseased stomach can be regularly accomplished thereby. Complete emotional tranquility often seems a utopian dream rather than a possible achievement in a real world with its inevitable problems, frustrations and insecurities. Nevertheless dreams may provide goals. The authors have established relationships and mechanisms; they have indeed pointed beyond the horizon.

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SPECIAL ARTICLES

TRANSMISSION TO RODENTS OF LANSING TYPE POLIOMYELITIS VIRUS ORIGINATING IN THE MIDDLE EAST[1]

DURING 1941–42 a number of cases diagnosed as poliomyelitis or encephalitis were reported[2] in the Middle East Forces of the British Army. Other neurological syndromes observed among the troops in the same area at that time were tentatively designated as "lymphocytic choriomeningitis," "polioencephalitis," "brachial neuritis," "wasting" of various groups of muscles. A number of specimens derived from these cases were made available to us for investigation.[3]

The materials consisted of fragments of human and monkey spinal cord and brain, immersed in glycerolated saline solution, and many of them had been in transit for several weeks at different seasons of the year. The monkey tissues were from Abyssinian grivets and baboons (*C. griseoviridis* and *P. hamadryas*) which had developed paralysis after inoculation of human materials by Major Van Rooyen.

Transfers to monkeys: Of tissue specimens derived from seven cases, three proved to be infectious for *rhesus* monkeys.

(a) Strain MEF1: The material received was labeled "monkey and baboon cords, 1st passage." Bacterial culture yielded no growth; intracerebral inoculation of a 10 per cent. suspension in broth into rabbits, guinea pigs and mice, and injection into embryonated eggs gave negative results. Intracerebral inoculation into two monkeys led to characteristic flaccid paralysis on the 12th day in one, while the other one was sacrificed on the 9th day when showing tremor and weakness of the hind limbs. Typical lesions of experimental poliomyelitis were present in the spinal cord and brain of this animal. Nevertheless, four monkeys inoculated with a suspension of this cord remained well.

A second isolation of virus from the original material was made and this time the virus could be maintained through another passage. Two monkeys inoculated with a mixture of the original material and pooled poliomyelitis (MV and Philadelphia strains) monkey antiserum remained well.

(b) Strain MEF2: The specimen received was baboon cord. Culture yielded no bacterial growth. Transfer into mice, rabbits, guinea pigs and chick embryos failed. Two monkeys, inoculated intracerebrally with a 10 per cent. suspension in broth, developed characteristic paralysis and in one instance histological study revealed typical poliomyelitic lesions. In the same test, one monkey paralyzed following infection with Strain MEF1 remained unaffected by Strain MEF2, while a second one which, after injection of Strain MEF1, had had only fever for 5 days, came down with quadriplegia. A mixture of virus and anti-MV-Philadelphia-poliomyelitis monkey serum was injected into two monkeys. One of these developed paralytic poliomyelitis, while the other one may have had an abortive attack (fever and weakness on the 6th day after inoculation).

(c) Strain MEF6: The original material was human cord tissue heavily contaminated with bacteria. All

[1] This investigation was made in collaboration with the Commission on Neurotropic Virus Diseases, Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army.

[2] C. E. Van Rooyen and A. D. Morgan (in press).

[3] Numerous human sera and specimens of nervous tissues were sent by Major C. E. Van Rooyen, R.A.M.C., to Captain T. M. Rivers, M.C., U.S.N.R., whose guidance and encouragement made this work possible. We also wish to thank Major Van Rooyen for his enthusiastic cooperation under difficult conditions, and Colonel J. S. K. Boyd, R.A.M.C., for his gracious permission to publish the results here presented.

ten mice, one of three guinea pigs, and three of four rabbits injected intracerebrally with centrifuged (twice at 10,000 r.p.m.) suspension succumbed to bacterial meningoencephalitis. The survivors remained well for several months. Through two passages monkeys developed characteristic poliomyelitis in absence of bacterial contamination. One monkey with residual hind-limb paralysis following infection with Strain MEF1 became completely paralyzed when reinoculated with Strain MEF6.

Comment: Identification of all three strains as poliomyelitis virus was based on (1) host range, (2) typical disease produced in *rhesus* monkeys, and (3) lesions in the central nervous system (CNS) of infected monkeys. In addition, one strain (MEF1) was neutralized by a standard anti-poliomyelitis monkey serum. The tests in which monkeys convalescent from MEF1 infection were reinoculated with the other two strains permit no definite conclusion as to the relationship between the agents. The frequent occurrence of "cross-immunity" on consecutive intracerebral inoculation of serologically unrelated poliomyelitis viruses has been described by others.[4]

Transfers to cotton rats, hamsters and mice: Cotton rats (*Sigmodon hispidus hispidus*) remained well after intracerebral inoculation of Strain MEF2, original baboon cord, and Strain MEF6, human or *rhesus* cord; these were infectious for monkeys. Moreover, with Strain MEF6, three intracerebral blind passages at 4-day intervals in cotton rats were ineffective.

Two of the three cotton rats inoculated with Strain MEF1-infected *rhesus* tissue developed flaccid hind-limb paralysis on the 14th and 15th days, respectively. The same material, with addition of a small portion of the MEF2 baboon cord, which in itself was non-infectious for cotton rats, was given intracerebrally to six hamsters and five cotton rats. Two consecutive blind passages were made at 4-day intervals in both species, using two animals as source of transfer material. All hamsters thus inoculated survived. The CNS tissue of those sacrificed showed no lesions. Eleven of twelve cotton rats left for observation developed flaccid paralysis after 11 to 22 days' incubation. Subsequently six more cotton rat to cotton rat passages were carried out (Fig. 1).

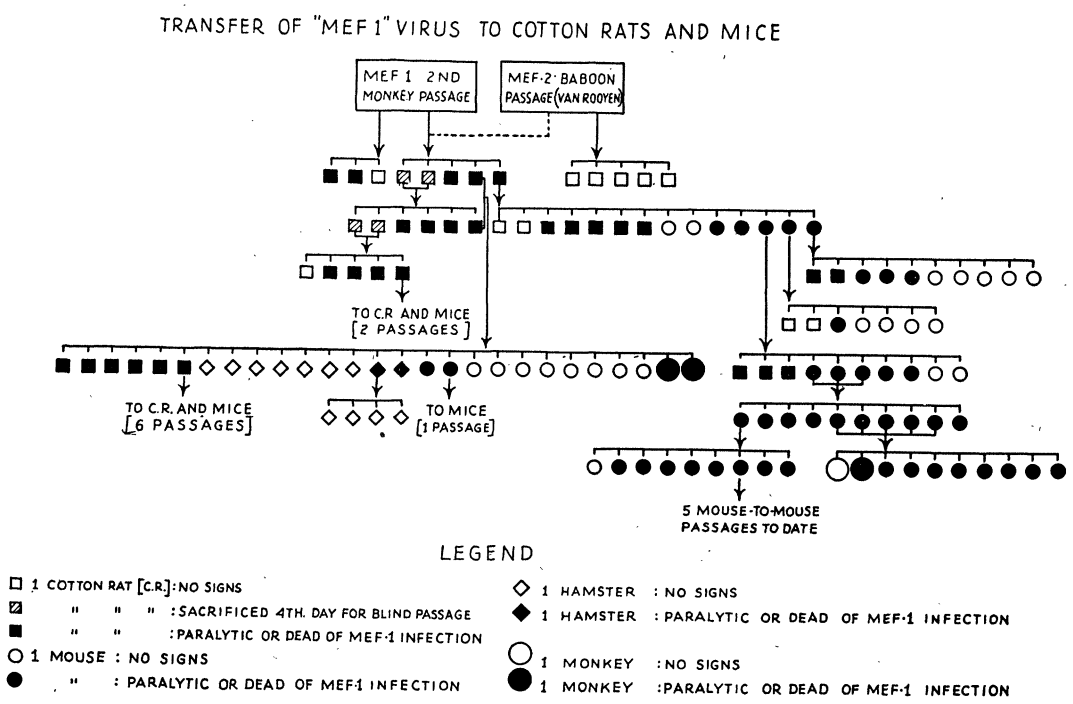

FIG. 1.

Nine hamsters received intracerebrally a pool of brain and cord from two paralyzed cotton rats. On the 16th day, one was found dead; another one had flaccid fore-limb paralysis. Passage from this one into four others was unsuccessful. Further tests in hamsters remain to be done.

Four of seven mice inoculated intracerebrally with the CNS tissue derived from the first infected cotton rat became paralyzed after 5 to 18 days. Four other cotton rat to mouse passages and subsequently five mouse to cotton rat passages transmitted the agent. In addition, fourteen mouse to mouse transfers have been carried out thus far. Two monkeys inoculated

[4] J. F. Kessel and F. D. Stimpert, *Jour. Immunol.*, 40: 61, 1941.

with pooled CNS tissue from cotton rats of the first two passages developed characteristic flaccid paralysis; so did one of two monkeys injected with mouse brain from the fourth mouse passage.

The disease in mice and cotton rats was characterized by flaccid paralysis of fore- and hind-limbs with rapidly progressing muscular atrophy. In some, death occurred overnight, before paralysis was observed. The incubation period ranged from 4 days to 3 weeks; occasionally to 40 to 57 days. Infected mouse brain, diluted 1/1000, was infective for mice. In these characteristics and in its nonpathogenicity in adult mice by peripheral routes, and also in regard to the type of lesion produced in the CNS of mice and cotton rats, the agent is indistinguishable from the Lansing strain of poliomyelitis virus.[5] Its pathogenicity appears to be quite different from that described by Jungeblut and Sanders[6] for their rodent-adapted SK-strain. The relation to the cotton rat-acclimated poliomyelitis strains of Toomey and Takacs[7] is uncertain.

Identification by neutralization tests: Mixtures of serum and varying dilutions of Lansing virus- or Strain MEF1-infected mouse brains were incubated for 1 or 2 hours at room temperature or 37° C. and injected intracerebrally into mice (Rockefeller Institute strain). The results, as summarized in Table 1,

TABLE 1

RESULT OF INTRACEREBRAL NEUTRALIZATION TESTS IN MICE

Animal	Source of serum: Convalescent after infection with	Virus (infected mouse brain): MEF1	Lansing
Rhesus monkeys	MEF1—Monkey CNS	+	+
	MEF1 + 2—Monkey CNS	+	+
	MEF2—Monkey CNS	+	+
	MEF6—Monkey CNS (Rh. No. 3710)	0	0
	MEF6—Monkey CNS (Rh. No. 3711)	N.T.	0
	MV—Philadelphia	+	N.T.
	MEF1—Mouse CNS	+	+
	Lansing—Cotton rat CNS*	+	+
Cotton rats (Pool)	MEF1 { Mouse CNS / Cotton rat CNS	+	N.T.
Mice (Pool)	MEF1 { Mouse CNS / Cotton rat CNS	+	N.T.

+ = Neutralization.
0 = No neutralization.
N.T. = Not tested.
* = Lansing antiserum supplied by Dr. Max Theiler.

indicate cross-neutralization between the MEF1 strain (before and after cotton rat and mouse passage) and Lansing virus. The MEF1 strain was also inactivated by MV-Philadelphia poliomyelitis monkey antiserum. Theiler's (mouse encephalomyelitis) virus was not neutralized by Strain MEF1 monkey antiserum. Serum from Strain MEF2-convalescent monkeys neutralized Strain MEF1 as well as Lansing virus.

Strain MEF6 failed to induce antibody against either Lansing or MEF1 virus. The simultaneous presence of at least two serologically unrelated poliomyelitis viruses in one epidemic area would limit the usefulness of the mouse neutralization test for an epidemiological study of human sera from convalescents and contacts. Nevertheless, a variety of human convalescent sera received from Major Van Rooyen have been tested against Lansing virus. The significance of the results would depend on the outcome of similar tests planned with sera from healthy individuals of the Middle East Forces.

Attempts to transfer virus from other cases into rodents are being made.

Summary: Three strains of poliomyelitis virus were isolated by monkey passage from cases occurring among the Middle East Forces of the British Army. One was indistinguishable from the Lansing strain, since it was transmitted to rodents and was serologically identical. The second was also serologically of the Lansing type, but thus far transfer to rodents has failed. The third was apparently not related to the Lansing or the first strain and passage to rodents has been unsuccessful. Thus, two apparently unrelated poliomyelitis viruses were isolated from the same epidemic area.

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[5] C. Armstrong, *Pub. Health Rep.*, 54: 1719, 1939; *Pub. Health Rep.*, 54: 2302, 1939; R. D. Lillie and C. Armstrong, *Pub. Health Rep.*, 55: 718, 1940.

[6] C. W. Jungeblut and M. Sanders, *Jour. Exp. Med.*, 72: 407, 1940.

[7] J. A. Toomey and W. S. Takacs, *Proc. Soc. Exp. Biol. and Med.*, 45: 364, 1940; 46: 22; 319, 1941; 47: 123, 1941.

THE MECHANISM OF AUXIN ACTION

A NUMBER of studies on the relation of plant auxins to enzyme activity[1,2,3] have been carried out in this laboratory. It has been reported that the activity of certain dehydrogenases from *Avena* coleoptile tissue is not accelerated when synthetic auxins are added to the functioning enzyme systems. Commoner and Thimann[4] have reported stimulation by auxin of the total oxygen uptake of excised, living coleoptile segments. A different approach to the problem, though less direct than the first mentioned, has yielded promising results, and since the work has had to be discontinued, we are reporting preliminary findings now.

[1] J. Berger and G. S. Avery, Jr., *Am. Jour. Bot.*, 30: 290–297, 1943.

[2] Ibid., 30: 297–302, 1943.

[3] Ibid., in press.

[4] B. Commoner and K. V. Thimann, *Jour. Gen. Physiol.*, 24: 279–296, 1941.