TABLE I.
Age Incidence of Leukemia in Strain F Mice on High and Low Fat Diets.

	High fat (48 mice)			Low fat (48 mice)		
Age in days.	Leukemic	Non-leukemic	Living	Leukemic	Non-leukemic	Living
0-100						
101-200						
201-300	6			2	1	
301-400	6			6	1	
401-500	9	2	3	9	1	2
501-600	6	3	8	12	1	12
601-700		2	3		1	
Totals	27	7	14	29	5	14

ferences in caloric intake and growth of the two experimental groups could account for the results obtained.

In order to test the effect of dietary fat on the carcinogenic induction of leukemia, methylcholanthrene (0.5% solution in benzene) was applied percutaneously twice weekly to 49 strain Dba mice (subline 12) fed the high fat diet, and to 48 mice of the same strain and subline which were fed the low fat diet Mice of this subline are susceptible to the induction of leukemia with this treatment.[12] Administration of carcinogen was begun when mice were 8 weeks of age and had been fed their respective diets for 4 weeks. Of those mice on the high fat diet, 16 of 22 males and 14 of 27 virgin females developed leukemia after average latent periods of 143 and 135 days respectively. Of those on the low fat diet, 21 of 33 males and 12 of 15 virgin females were leukemic after average induction periods of 116 and 113 days. Food intake was approximately isocaloric and there was no appreciable difference in the weights of the high and low fat animals. Frequency distribution curves plotted from induction periods of individual mice on the two diets indicate that the delay in onset of carcinogen-induced leukemia of mice on the high-fat diet was statistically significant.

It would seem that although the leukemia incidence was the same for both dietary groups, the induction of the disease was significantly delayed when the diet contained a relatively high per cent of fat. The oil on the fur of the high fat animals might have retarded absorption of carcinogen, thus prolonging the induction period.

[12] Kirschbaum, A., Lawrason, F. D., Kaplan, H. S., and Bittner, J. J., PROC. SOC. EXP. BIOL. AND MED., 1944, in press.

14568 P

Prevention of Neuroma Formation by Encasement of the Severed Nerve End in Rigid Tubes.

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Neuromata characteristically form at the proximal end of sectioned peripheral nerves. These tumors are frequently painful and represent a most troublesome problem in the handling of amputation stumps. The methods recommended in the Neurosurgical section of the Military Surgical Manual[1] to prevent such neuroma formation are: (a) the alcohol injection of the stump as suggested by Huber and

[1] National Research Council: Military Surgical Manual, Vol. 6, W. B. Saunders Co., Phila., 1943.

Lewis,[2] (b) the excision of a wedge of the nerve end and suture of the flaps (Corner[3]), and (c) a combination of the two procedures as proposed by Stookey.[4] Kirk[5] described a procedure in which the end of the nerve is cauterized with the high frequency current. Boldrey[6] reported an experimental method for overcoming neuroma formation by burying the stump of the nerve in bone. While these procedures frequently reduce the size to which neuromata grow, they have not been universally effective in preventing the development of painful amputation stumps.

In an attempt to completely inhibit neuroma formation, by controlling the growth of the nerve fibers and connective tissue, the cut ends of the sciatic nerve of the dog were placed in snugly fitting, rigid tubes of various materials: silver, cellophane, vitalium, or glass.

This experimental procedure has been carried out on 24 animals. After as long as 102 days the fibers of the sciatic nerve encased in a snugly fitting glass tube are straight and orderly and there is no tendency towards neuroma formation. All control experiments in which a portion of nerve was resected showed neuroma formation.

Conclusion. When the cut end of the sciatic nerve of the dog is supported by a closely fitting, rigid tube, there is no tendency to disorderly overgrowth of the nerve fibers and connective tissue characteristic of neuroma formation.

2 Huber, G. C., and Lewis, D., *Arch. Surg.*, 1920, **1**, 85.

3 Corner, E. M., *Proc. Roy. Soc. Med.*, **11**, 7, April 9, 1918.

4 Stookey, B., *Surgical and Mechanical Treatment of Peripheral Nerves*, p. 461, chap. 21, W. B. Saunders Co., Phila., 1922.

5 Kirk, N. T., Amputations: Dean Lewis, *Practice of Surgery*, Vol. III, chap. 10, 1943.

6 Boldrey, E., *Ann. Surg.*, 1943, **118**, 1056.

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In vitro Action of Penicillin Alone, and in Combination with Sulfathiazole, on *Brucella* Organisms.

TSUN T'UNG. (Introduced by Martin Frobisher, Jr.)

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Information concerning the antibacterial effect of penicillin on gram-negative organisms is meager and it is a general belief that most gram-negative bacilli, including *Brucella*,[1] are relatively resistant to penicillin. This has led to possibly undue pessimism with regard to the therapeutic use of available chemotherapy in brucellosis. The present communication presents *in vitro* studies of the action of penicillin alone, and in combination with sulfathiazole, on a number of strains of *Brucella* organisms.

Materials and Methods. Penicillin was prepared from a Fleming strain of *Pen. notatum* cultivated on modified Czapek-Dok medium[2] to which 1% of casamino acid was added. The potency of the crude penicillin on the 7th day when tested by the serial dilution method was around 1-640 against a test strain of *Staphylococcus aureus* H (3R9674). The pH of the culture fluid on the 7th day was about 7.5. Extraction with amyl acetate and buffer was carried out according to the procedure of Abraham *et al.*[3] Extraction was repeated once to concentrate the penicillin. The extracted material was shaken with 2% animal charcoal

1 Abraham, E. P., Chain, E., Fletcher, C. M., Gardner, A. D., Heatley, N. G., Jennings, M. A., and Florey, H. W., *Lancet*, 1941, **11**, 177.

2 Hobby, Gladys L., and Meyer, Karl, PROC. SOC. EXP. BIOL. AND MED., 1943, **50**, 227.

3 Abraham, E. P., and Chain, E., *Brit. J. Exp. Path.*, 1942, **23**, 103.