this unit as a guide. Because of the known variation of the normal complement curve, all series of animals including controls were bled and tested at the same time. The time of titration of the different series, after withdrawal of blood, varied from 4 to 18 hours.

Table I demonstrates the results obtained for representative animals of the different series and controls. In general it may be stated that no marked or clear cut difference in the complement content is manifested in animals showing leukopenia or leucocytosis as compared to the controls. Upon closer analysis there is an indication that leukopenic animals have a slightly higher complement titre than either the normal animals or those showing leucocytosis. In certain of the animals wherein benzene injections had been employed, the complement titre was high even just prior to death. The complement content of the sera of the animals with leucocytosis was quite similar to that of the controls.

While complement may be reduced or absent in allergy and this latter factor may play a rôle in the causation of the Schultz syndrome, our preliminary results do not show any reduction of the alexin of the sera of animals in which experimental leukopenia was produced.

7434 P

Barbiturate-Strychnine Antagonism in the Spinal Cat.

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Barbiturates are generally thought to act chiefly on the brain,[1] strychnine to exert its convulsant effects by an action on the cord.[2] If this were an absolute difference between the two, it would be difficult to see how the barbiturates could act as effective antagonists to strychnine, as numerous observers[3] have shown.

To test whether this antagonism does exist in the cord, the writers have made use of 9 spinal (decapitate) cats, prepared by the Sher-

[1] Sollman, T., Manual of Pharmacology, 4th Ed., 1932.

[2] Dusser de Barenne, J. G., *Physiol. Rev.*, 1933, **13**, 325.

[3] Zerfas, L. G., and McCallum, J. T. C., *Curr. Res. Anesth. and Analg.*, 1929, **8**, 349. Dawson, W. T., and Taft, C. H., Jr., PROC. SOC. EXP. BIOL. AND MED., 1931, **28**, 917. Llopis Llorente, R., *Cron. Med. Valencia*, 1932, **36**, 58. Swanson, E. E., *J. Lab. and Clin. Med.*, 1932, **17**, 325. Haggard, H. W., and Greenberg,

rington method.[4] Strychnine sulphate was administered subcutaneously in doses of 0.75 or 1 mg. per kg. Violent convulsions followed in all cases, marked by typical repeated rigid extensor tetanus. When this stage was reached, a barbiturate solution was injected intravenously. This injection was followed in each case instantly by muscular relaxation. Threatened or actual recurrence of tetanus was met by further doses of barbiturate. The animals survived for varying lengths of time from 24 minutes up to several hours after tetanus appeared. Three control animals receiving strychnine alone showed recurrent tetanus for 20 to 80 minutes after its onset. All doses were based on weight of intact cat.

The results are summarized in Tables I and II.

TABLE I.
Barbiturate-Strychnine-Antagonism in Spinal Cat.

Strych. Sulph. Cryst. mg./kg.	Min. to tetanus	Barbiturate, Initial dose in mg./kg.	Further tetani and procedure	Further doses Barbiturate X size of each in mg./kg.	Min. survival after onset of tetani
0.75	32	Na pento-barbital, 2%; 10	1 tetanus; 10 mg./kg. repeated	0	95+
0.75	40	Na amytal 10%; 10	0	0	68-81
1.00	39	Na pheno-barbital, 10%; 40	0	1 x 40	43-48
1.00	42	Alurate, 10%; 33	0	2 x 16	92+
1.00	39	Dial, 10%; 10	0	11 x 5	69+
1.00	9	Pernocton, 1% (Na salt); 1	1 weak tetanus	0	24-37

TABLE II.
Strychnine Subcutaneously in Spinal Cat (Strych. Sulph. Cryst. 1.0 mg. per kg.).

Cat No.	1	2	3
Min. to onset of tetanus	5	14	19
Time tetani continued to occur	20	60	80
Time strong tetani continued to occur	12	38	55
No. of strong tetani	7	28	10
" " weak "	2	8	5
Min. survival after onset of tetanus	43-59	86-91	80-83

L. A., *J. Am. Med. Assn.*, 1932, **98**, 1133. Simon, I., *Arch. di farmacol. sper.*, 1932, **54**, 55. Barlow, O. W., *J. Am. Med. Assn.*, 1932, **98**, 1980. Wheelock, M. C., *J. Am. Med. Assn.*, 1932, **99**, 1862. Kempf, G. F., McCallum, J. T. C., and Zerfas, L. G., *J. Am. Med. Assn.*, 1933, **100**, 548. Swanson, E. E., *J. Lab. and Clin. Med.*, 1933, **18**, 933. Stalberg, S., and Davidson, H. S., *J. Am. Med. Assn.*, 1933, **101**, 102. Maloney, A. H., *J. Pharmacol. Exp. Therap.*, 1933, **49**, 133. Fenton, B. C., *J. Am. Med. Assn.*, 1933, **101**, 1333. Koumans, A. K. J., *Klin. Woch.*, 1934, **13**, 103.

[4] Liddell, E. G. T., and Sherrington, Sir Charles, Practical Mammalian Physiology, 1929.

Comment on Tables. "Survival"—disappearance of reflexes and of heart beat were usually nearly simultaneous, the survival period being between limits given. *Notes on individual experiments.* Na pentobarbital—recurrence of tetanus after 26 minutes shows that the strychnine dosage used is capable of causing a prolonged tetanic stage; observation was interrupted 70 minutes after the second tetanus, the preparation was left on artificial respiration and was in good condition 4½ hours later. Na amytal—it will be noted that the abolition of tetani may result from even very moderate dosage with barbiturate, but that this does not necessarily restore to the spinal cat preparation its normal viability of many hours. Alurate in diethylamine solution—92 minutes after onset of tetanus the preparation was in very good condition; observation terminated. Dial, in urethane-monoethyl urea solution, these bases present in amounts claimed not greatly to affect dial action[5]—the preparation was left on artificial respiration and found in good condition 2 hours later. Pernocton—a weak tetanus occurred 14 minutes after the first, and the reactivity then spontaneously and rapidly diminished to zero.

The present work is, of course, purely preliminary. It indicates, nevertheless, the existence of a barbiturate-strychnine antagonism in the cord animal. Experiments of a more quantitative nature are in progress.

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Electrocardiographic Changes in Acute and Chronic Pericarditis in the Goat.

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Clinical electrocardiographic experiences emphasized to us the similarity of the subjective, physical and electrocardiographic findings in hemopericardium and acute fulminating pericarditis, but we also noted electrocardiographic changes in the subacute or chronic healing stages of pericardial inflammation.[1] In order to get more

[5] New and Non-official Remedies, 1934.

[1] Herrmann, G., and Schwab, E. H., *Trans. Assn. Am. Phys.*, 1934, **49**, in press.