reaction, involving 2 mechanisms, namely, encapsulation and phagocytosis.

Although infection of the dog with human strains of *S. stercoralis* is maintained only for a period of weeks or months, in contrast to years in man, the natural host, it is unlikely that man remains infected indefinitely without outside exposure or hyperinfection. Suspected patients should never be pronounced "negative" for *Strongyloides* until prolonged intensive fecal examinations have been made. Even then the patient may be harboring tens or hundreds of post-productive females, which may account for characteristic symptoms of chronic strongyloidosis. Likewise the number of larvae recovered from the feces is not necessarily an index of the degree of infection in the intestinal wall.

7432 P

Filarial Periodicity in the Dog Heartworm, Dirofilaria Immitis, After Blood Transfusion.

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Lane[1, 2] believes that the mechanism of filarial periodicity in *filariasis bancrofti* in man is due to a simultaneous daily parturition of the females, involving the destruction daily of as many microfilariae as are produced. O'Connor[3, 4] has found histological evidence to support Lane's hypothesis, and concludes that this occurs about midday in Puerto Rico. On the other hand, Low and Manson-Bahr[5] criticize the theory, because of the effects of absorption of these millions of dead microfilariae in the body, and because microfilariae have been kept alive outside the host for a week. Murgatroyd,[6] however, injected citrated blood containing 720,000

[1] Lane, C., *Lancet*, 1929, 1291.

[2] Lane, C., *Lancet*, 1933, 399.

[3] O'Connor, F. W., *Porto Rico J. Pub. Health and Trop. Med.*, 1931, **6**, 263.

[4] O'Connor, F. W., and Hulse, C. R., *Trans. Roy. Soc. Trop. Med. and Hyg.*, 1932, **25**, 445.

[5] Low, G. C., and Manson-Bahr, P. H., *Lancet*, 1933, 455.

[6] Murgatroyd, F., *Lancet*, 1933, 610.

microfilariae into a human volunteer 2 hours after withdrawing the donor's blood, but never recovered microfilariae from the recipient's blood. Rao[7] kept microfilariae alive *in vitro* at 4°C. under aseptic conditions for 4 to 6 weeks. He concludes from collateral evidence that the duration of the life of the embryos in the human body is about 70 days.

Dirofilaria immitis is an infection of the dog, the adult worms occurring chiefly in the right ventricle and pulmonary arteries and their microfilariae exhibiting a nocturnal periodicity, although not so marked as in *Wuchereria bancrofti.* Fülleborn[8] injected 6.25 cc. of blood, containing approximately 450,000 microfilariae, from an infected dog into a young puppy. These microfilariae persisted in the blood of the recipient for 9 months but showed no periodicity and were present in only about one-third of the expected number. He believed that the majority of the larvae withdrew to the lungs and only a fraction got into the circulation. Unfortunately the possibility of natural infection by mosquitoes or *via* the placenta of the mother was not entirely excluded.

The present writers transfused heavily parasitized blood at 5 P. M., March 29, 1934, into an uninfected 12-pound dog, shipped from Chicago (well outside the endemic area). The animal was kept in a doubly screened cage before and after the experiment. One hundred forty cc. of blood containing 27,475 microfilariae per cc. were transfused directly into the femoral vein, using the Gillentine-DeBakey syringe[9] after previous removal of an equal amount of blood. This latter blood was citrated and injected into the donor which weighed 10 pounds. Estimating one-twelfth of the body weight of the dog to be blood, the recipient had lost and later received approximately one-third of its blood. Therefore the number of microfilariae which might be expected to appear in its blood, if an even distribution resulted, would be about 9000 per cc. Actually only 8% of this number ever appeared in the peripheral bloodstream.

Counts of microfilariae were made upon the donor and recipient every 2 hours for a 24-hour period before and after the transfusion (Table I). Daily counts were then made at 5 P. M. for a week and subsequently, weekly counts (Table II). These figures

[7] Rao, S. S., *Indian Med. Gaz.*, 1933, **68**, 3; *Abst. in Trop. Dis. Bull.*, 1933, **30**, 705.

[8] Fülleborn, F., *Beihefte Arch. f. Schiffs-u. Tropenhyg.*, 1908, **12**, 6.

[9] Gillentine, W. H., and DeBakey, M. E., *New Orleans M. and S. J.*, 1933, **86**, 100.

are averages of counts on 2 samples of 8 cmm. each for the donor and of 20 cmm. each for the recipient.

TABLE I.
Transfusion.
No. microfilariae per cc.

Time	Donor		Recipient	
	Before	After	Before	After
5 P.M.	21,400	19,875	0	563
7 "	25,190	18,250		750
9 "	26,440	8,000		275
11 "	41,875	8,000		250
1 A.M.	44,625	9,063		375
3 "	47,000	9,425		175
5 "	31,500	8,875		300
7 "	16,325	6,000		350
9 "	17,625	13,750		375
11 "	17,750	9,350		425
1 P.M.	21,550	8,750		250
3 "	27,475	10,037		200

TABLE II.
No. microfilariae per cc.

Date—1934	Donor	Recipient
3/31— 5 P.M.	6,875	400
4/2 — 5 "	10,475	450
4/3 — 5 "	10,125	400
4/4 — 5 "	10,000	550
4/5 — 5 "	15,600	550
4/13— 5 "	21,650	750
—11 A.M.	35,875	75
4/19— 5 P.M.	38,750	100
—11 "	61,000	100
4/20— 5 A.M.	34,250	75
4/27— 5 P.M.	43,075	200
5/4 — 5 "	44,500	50

A study of Tables I and II indicates that the filarial count of the donor was markedly reduced by the removal of approximately one-third of the dog's blood, and remained lowered for 6 days, when it began to rise and had returned to almost the original level in 2 weeks (April 13). Counts done at 6-hour intervals on April 19-20 revealed that a greater number of microfilariae was present than before transfusion and that the usual periodicity reappeared. (Injection of sodium citrate was shown to produce no apparent reduction in numbers of microfilariae.) Daily production and destruction of the microfilariae would have returned the count to normal levels within 24 hours. Active microfilariae were found in the recipient's blood for 10 weeks, with only a slight variation during the first 2 weeks. The fact that the majority of embryos disappeared almost immediately after transfusion into the recipient may

be due to their filtration by the viscera, particularly the lungs, as suggested by Fülleborn.[8]

It is apparent that, in the case of the dog heartworm-infection, periodicity cannot be explained on the basis of cyclical parturition and daily destruction of the larvae.

7433 P

Observations upon the Complement Titre in Experimental Leukopenia and Leucocytosis.*

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In connection with the causation of the Schultz syndrome or so-called agranulocytic angina, allergy has been considered as a factor.[1, 2] In observations upon allergy Deutsch and Weiss[3] have shown that the complement titre was markedly lowered and in anaphylactic shock, apparently absent.

During certain studies upon the production of granulocytopenic leucopenia with bacterial toxins[4, 5] we considered that it would be of interest to make observations upon the complement content of animals in which these cells had been depressed. As another factor in the production of granulocytopenic leucopenia we added the use of benzene inoculations. In addition to the animals wherein the leucocytes were depressed, others were employed in which experimental leucocytosis had been provoked.

In the present experiments 36 guinea pigs and 18 rabbits were employed. The complement titre of all rabbits used was found to be approximately 1/10 the strength of that of the guinea pigs. Seventeen guinea pigs in which granulocytopenic leukopenia was produced with *in vivo* prepared toxic filtrates of *B. enteritidis* and *B. typhosus* were bled from the heart. Eight rabbits were injected

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1 Pepper, O. H. P., *Calif. and Western Med.*, 1931, **35**, 173.

2 Editorial, *J. Am. Med. Assn.*, 1933, **101**, 368.

3 Deutsch, F., and Weiss, E., *Med. Klin.*, Berlin, 1933, **29**, 1402.

4 Harris, W. H., and Schattenberg, H. J., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 843.

5 Schattenberg, H. J., and Harris, W. H., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 847.