was diminished. 7. Blockade of the primary blood pressure rise elicited by faradization of the afferent vagus occurred; the secondary rise was essentially unaltered. 8. The hypotensive responses to acetylcholine, histamine and efferent vagal faradization were not altered.

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Presence in Human Sera of Complement Fixing Antibodies to Virus of Sporadic Bovine Encephalomyelitis. (20920)

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The Department of Public Health, School of Veterinary Medicine, of the University of California routinely obtains blood samples from the personnel and students in the School. The sera are tested by complement fixation or agglutination for antibodies to various etiological agents of disease that are transmissible from animals to man. This program is expanded at times to encompass other agents that have not as yet been reported as causing infection in man. This paper is a preliminary report of the result of one such investigation involving the virus of sporadic bovine encephalomyelitis (SBE). The data reported here indicts SBE as yet another disease of animals whose causative agent may infect man. There has been little in the literature to indicate its role in animal disease until the past few years and no reports of infection in man. The disease in animals was first recognized in Iowa and reported by McNutt (1). SBE has been reported from only 11 states, but Menges, Harshfield, and Wenner (2) suggest that it is much more widespread.

Materials and methods. Seven sera from personnel in the School, were tested for complement-fixing (C-F) or agglutinating antibodies with 9 different antigens. In addition, a total of 481 human sera were tested for C-F antibodies to the SBE virus and to the Henzerling strain of *Coxiella burneti*. These same sera were tested by agglutination for antibodies to *Brucella*. The antigen of primary significance in this report, designated here as LMcN, was received from Dr. Sam C. Wong of Lederle Laboratories. It is an experimental, soluble, C-F antigen prepared from a 10% suspension of the infected yolk sac of embryonated eggs by ether extraction in the same manner as for the preparation of typhus vaccine. The organism used in preparing this antigen was isolated from the pleural exudate of a sick calf by Dr. S. H. McNutt. Subsequent to receiving this antigen from Dr.

TABLE I.* Comparison of Complement-Fixing or Agglutinating Activity of Seven Human Sera to Nine Different Antigens.

History of exposure	Antigens								
	LMcN	6BC	LG	Q	Bruc	LCM	WEE	StL	Jap-B
I	128	Neg	<8	<8	<25	<8	<8	<8	<8
X†	128	16	<8	64	<25	ac	ac	ac	ac
0	64	2	<8	<8	<25	<8	<8	<8	<8
I†	32	4	<8	128	<25	<8	<8	<8	<8
0	32	16	ac	32	<25	ac	ac	ac	ac
X†	8	Neg	<8	64	<25	<8	<8	<8	<8
X†	<8	4	<8	<8	<25	<8	<8	<8	<8

* Titers are reciprocal of highest dilution showing a 3+ or 4+ reaction.
Key: I —Known exposure to psittacosis but no known exposure to SBE.
X—Known exposure to SBE, but no known exposure to psittacosis.
0 —No known exposure to either psittacosis or SBE.
† —Known exposure to Q fever.

Wong it was used to identify 2 isolates from bovines with typical symptomatology and pathology of SBE. These isolations were made in the School of Veterinary Medicine, University of California, and the organism passaged in guinea pigs. The paired sera from these guinea pigs showed a rise from a pre-inoculation C-F titer of less than 1:8 to a post-inoculation blood titer of up to 1:128. The *6BC psittacosis antigen* was a 20% suspension of infected yolk sac material steamed for one-half hour and then phenolized. The second "Psittacosis" antigen, designated LG, was Squibb's "Lygranum". The *Q Fever antigen* was Lederle's commercial Q Fever (Italian strain Henzerling) C-F antigen. The Brucella antigens were (a) prepared from the standard NIH strain and (b) the U.S. Bureau of Animal Industry Strain 19. The *C-F antigens* used to test the sera for antibodies to the viruses causing lymphocytic choriomeningitis, Western equine encephalomyelitis, St. Louis encephalitis, and Japanese B. encephalitis were standard diagnostic antigens.

Results. The C-F or agglutination titers of the 7 human sera to the 9 antigens are tabulated in Table I.* The C-F titers of the 481 human sera to LMcN SBE and to Q Fever antigens are presented in Table II. C-F titers of the 481 human sera to LMcN SBE antigen and the agglutination titers to Brucella are compared in Table III.

It is quite evident from Table I that no correlation is demonstrated between the titer to the LMcN SBE antigen and the titers to any of the other 8 antigens used. The lack of correlation between titers to the LMcN SBE antigen and to either the 6BC Psittacosis or the "Lygranum" antigens is of particular significance inasmuch as the viruses of psittacosis and SBE are reported to be serologically related(2,3).

Of the 481 human sera tested, 155 had C-F titers to the antigens of LMcN SBE and/or *C. burneti.* These titers are compared in Table II and show no correlation between the 2 antigens.

Of the 481 sera tested, 126 had titers to LMcN SBE and/or brucella. These titers, presented in Table III, also demonstrate no correlation between the titers to LMcN SBE and the titer to brucella.

Discussion. It would seem from these findings that 6 of the 7 individuals reported in

TABLE II.* Comparison of the Complement-Fixing Activity of 481 Human Sera to the Antigens of SBE and Q Fever.

Titer to LMcN SBE	Titer to Q fever					
	<8	1:8	1:16	1:32	1:64	1:128
<1:8	326	21	19	28	18	7
1:8	4	6	0	0	4	0
1:16	4	0	1	2	2	0
1:32	12	0	0	5	2	1
1:64	7	2	1	0	2	0
1:128	4	1	1	0	0	1

* Numbers in table refer to numbers of individuals. One serum reported here is not reported in Table III.

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TABLE III.* Comparison of Complement-Fixing and Agglutinating Activity of 481 Human Sera to the LMcN SBE Antigen and Brucella Antigen Respectively.

C.F. titer to LMcN SBE	Brucella agglutination titers: Neg	20	40	80	160	320	640	1280
<8	355	18	21	9	7	3	4	1
8	13	0	0	1	1	0	0	0
16	6	0	0	0	2	0	1	0
32	18	1	1	0	0	0	0	0
64	9	0	2	1	0	0	0	0
128	7	0	0	0	0	0	0	0

* Numbers in table refer to numbers of individuals. One serum reported here is not reported in Table II.

Table I had C-F antibodies in their sera which were specific to the soluble antigen prepared from McNutt's strain of SBE virus. Furthermore, it would seem that the soluble antigen of the McNutt strain of the virus of SBE is distinct from the above described antigens prepared from members of the psittacosis lymphogranuloma venereum group of viruses. If the comparison of the titers of these 7 sera to the 9 antigens may be considered indicative of the specificity of the LMcN SBE antigen and the results tabulated in Tables II and III are accepted as further substantiation of this specificity, then it is evident that out of 488 human sera tested, 75 (15%) had a titer of 1:8 or higher and 51 of the 75 had a titer of 1:32 or higher to the virus of sporadic bovine encephalomyelitis. Further studies are in progress to determine the significance of these data.

Summary. The presence of complement-fixing antibodies to the virus of sporadic bovine encephalomyelitis has been demonstrated in 15% of the 488 human sera tested, with 51 of these 75 sera showing relatively high titers. The apparent specificity of the C-F antibodies to the virus of SBE has been demonstrated. The suggestion is made that SBE is yet another disease of animals whose causative agent is transmissible to man.

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Production of Lipemia Clearing Factor During Anaphylactoid Shock. (20921)

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It has been known for many years that, in dogs, the intravenous injection of peptone produces profound shock associated with incoagulable blood. Later it was shown that the clotting defect is due to the presence of heparin and that the mast cells of the liver of shocked animals are degranulated(1). Shortly thereafter it was demonstrated that protamine reverses the coagulation defect resulting from anaphylactic shock in dogs(2) and subsequently heparin was isolated from the blood of such animals(3). The source of the heparin is apparently the liver, for no heparin could be found in the blood of shocked, hepatectomized dogs(2). Recently many organic bases, termed "histamine liberators," have been found to produce shock with some of the characteristics of anaphylaxis (4). This also has been accompanied by the release of a heparin-like substance into the blood of dogs. In other mammals it has usually not been possible to demonstrate the presence of heparin in the blood during shock produced by these methods(5). Mast cell degranulation and disruption, however, has been found in rats following the administration of histamine liberators(6). It is well established that in many mammals the intra-