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Protective and Complement-binding Bodies in the Serum of Human Yellow Fever Convalescents.

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We have had the opportunity to make a serological study of 5 cases of yellow fever due to laboratory infections. The sera of 4 of these were examined for protective and complement-fixing bodies throughout convalescence so far as was practicable, while the serum of the other case was examined for the first property from the seventh week and for the second property from the eighth month after the onset of illness.

Protective antibodies: The technic of testing for the protective property was that usually employed, namely, the intraperitoneal inoculation of monkeys with serum, followed after 6 hours by the subcutaneous injection of fresh monkey blood containing virus. The table giving the results of these tests indicates that antibodies protecting monkeys against massive doses of virus appeared about the fifth day of illness in 4 cases. In the one instance in which serum was examined for the first time 7 weeks after illness, this protective property was present and repeatedly demonstrated thereafter.

Complement-binding bodies: The technic we employed in the complement-fixation tests was as described in a recent publication,[1] where the literature is briefly reviewed. It consisted essentially in the use as antigen of pooled infectious monkey serum preserved in a dry state, in the employment of a series of antigen dilutions against a constant dilution of serum, and in overnight fixation at 5°C. The usual controls were always included and in the experiments reported here were satisfactory.

A positive reaction appeared in one case in the eighth week after the onset of illness, and in another in the ninth week. The sera of 2 individuals gave positive tests at their first examination, 4 and 8 months after onset, while the reaction with the serum of one person has been consistently negative during the 8 months of observation after the acute attack. The same antigen preparation was used throughout these tests conducted for more than a year.

[1] Hudson, N. Paul, in press.

TABLE I.

Serological reactions of human yellow fever convalescents.

Protective antibodies									
Days after onset of illness	2	3	4	5	6	7	8	Remarks
Case No. Before illness								
1 (W. A. S.) not tested	—	—	—	—	—	—	—	Repeatedly positive from 7 weeks after illness.
2 (K. S.) not tested	—	neg.	neg.	pos.	—	—	pos.	Repeatedly positive later
3 (N. P. H.) negative	—	neg.	—	—	pos.	—	pos.	
4 (G. C.) not tested	neg.	—	neg.	—	—	pos.	—	
5 (S. F. K.) negative	—	neg.	—	pos.	—	pos.	—	

Complement-fixation										
Weeks after onset of illness	2	5	8	9	10	12	13	19		
Case No. Before illness										
1 (W. A. S.) not tested	—	—	—	—	—	—	—	—	Positive from 8 to 21* mo. after illness.	
2 (K. S.) not tested	neg.	neg.	—	pos.	pos.	—	—	—	Positive for 9* mo. after illness	
3 (N. P. H.) negative	—	—	—	neg.	—	neg.	—	—	Negative for 8* mo. after illness.	
4 (G. C.) negative	neg.	—	pos.	—	—	—	—	—	Repeated positive tests on the one specimen.	
5 (S. F. K.) negative	—	—	—	—	—	—	weak pos.	pos.	The only specimens examined.	

* = the time limit of experimentation.

Further laboratory and clinical studies on these cases are to be reported by Berry and Kitchen,[2] and the numbers of the cases given in the accompanying table are made to conform to their case numbers. Our findings in regard to the protective and complement-fixing properties cannot be correlated with the severity of illness of the patients.

The appearance of positive fixation reactions later than protective antibodies is probably due either to the crudeness of the complement-fixation test as conducted, or to the result of persistence

[2] Berry, G. P., and Kitchen, S. F., to be published.

of the virus in the host, or to the combination of both factors. When a positive fixation reaction occurred, it has persisted to the time limit of experimentation, in one instance to 21 months after onset of illness.

The animal test for protective antibodies as a means of diagnosis is expensive and not without danger to the worker. If the complement-fixation reaction is developed so as to give a high proportion of reliable results without false positives, it appears essential to apply it not before several weeks after the suspected illness.

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The Cardiac Output in Hyperventilation.*

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Henderson[1] described the depression of blood pressure in acapnia, advanced the hypothesis of a venopressor mechanism, and still believes that the decrease of the venous return to the right heart is a principal factor in the production of this effect.[2] Dale and Evans[3] showed by cardiometer experiments that the cardiac output is not significantly altered in hyperventilation, and thought the effect due to depression of the vasomotor centres of the bulb and spinal cord. Voluntary over-breathing in man results inconstantly in a reduction of the blood pressure; and McDowell[4] reported that in some dogs anesthetized with chloralose no fall, or even a rise, of blood pressure occurred with acapnia, while in all animals anesthetized with ether a fall occurred. These observations were explained by the hypothesis that acapnia has a dual effect on the circulation, (a) vasodilatation due to a central action, and (b) constriction of smaller and more peripheral vessels due to a local action.

In studying the effects of acapnia, it was desired to measure by the Fick method the cardiac output before and during hyperventila-

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[1] Henderson, Y., *Proc. Am. Physiol. Soc.*, Dec., 1905.

[2] Henderson, Y., *J. Am. Med. Assn.*, 1930, **95**, 572.

[3] Dale, H. H., and Evans, C. L., *J. Physiol.*, 1922, **56**, 125.

[4] McDowell, R. J. S., *J. Physiol.*, 1930, **70**, 301.