3. Peptone shock has long been known to resemble anaphylactic shock closely, and the inference has generally been drawn that the latter is due to the production and circulation of peptone-like bodies. But the transfusion experiments above described do not bear out this theory. The suggestion is made that these two syndromes coincide for the reason that both alike result from a reaction of the liver. Phosphorus and chloroform, both hepatic poisons, also produce entirely similar changes in the chemistry and coagulability of the blood. Peptone is known to protect sensitized animals against anaphylactic shock. I have found that phosphorus or chloroform poisoning exerts a similar effect. The conclusion is drawn that the liver may be partially exhausted by any of these methods, and will not then react as acutely in anaphylactic shock.

4. Anaphylaxis in dogs is a cellular phenomenon, due chiefly, if not wholly, to the reaction of the sensitized liver cells.

5. It is suggested that the fall of blood pressure in anaphylactic shock may indirectly be due to the liver. That organ is found to be enormously congested, and it is conceivable that the animal is "bled to death" into its own liver. Or, the drop in pressure may be a vaso-motor reflex, comparable to the Goltz phenomenon, initiated by the acute hepatic shock of the reaction.

70 (1248)

The response of the respiratory mechanism to rapid changes in the reaction of the blood.

By JOHN P. PETERS, JR., M.D. (*by invitation*).

[*From the Medical Clinic, Presbyterian Hospital, and the Coolidge Fellowship for Medical Research, Columbia University, New York.*]

For the past year we have been studying the reaction of the blood by two parallel methods: the Fridericia method for alveolar CO_2 and the Van Slyke determination of the carbon dioxide combining power of the blood. We believe that this combination offers a simple method for the study of the state and the reaction of the respiratory mechanism.

In no normal person have we found a disagreement of over 10 per cent. in the ratio of alveolar/plasma CO_2. Discrepancies observed seem to fall into two large classes: (1) Those due to a mechanical interference with the gaseous exchange, and (2) those due to changes in the control of the respiratory mechanism.

The first class is best illustrated by patients with cardiac dyspnea or very great diminution of the pulmonary capacity. In these the alveolar is invariably lower than the plasma reading. If compensation is established the two again come into agreement. This, we believe, is due to an impairment of the lungs that renders the excretion of CO_2 more difficult. The body overcomes this, in response to stimulation of the respiratory center by the retained CO_2, with a greater pulmonary ventilation. In this way a pressure difference is established between the carbon dioxide tension in the blood and in the alveoli, sufficient to compensate for the impairment in the lungs. The increased minute volume, the intolerance to carbon dioxide in the inspired air, the dyspnea and the disproportionate response to exercise are all expressions of the same thing.

The second class is best illustrated by the discrepancies observed after rapid changes in the reaction of the blood. The alveolar air tends to lag behind the plasma in its response. This is susceptible of an easy explanation during a sudden increase of the H-ion concentration of the blood, when the demand on the respiratory mechanism is overwhelming and the lungs are flooded with CO_2. We have been unable to study any patients who have developed spontaneously a persistent acidosis, to determine the time relations of the change. We have found that adrenaline given intramuscularly to normal and diabetic persons produces an acidosis, the curve of which is somewhat more rapid than that of the hyperglycemia. After adrenaline and after exercise the return to normal is very rapid. In none of our cases did the alveolar CO_2 fall as far as the plasma CO_2.

A similar retardation of the alveolar response is found after rapid alkalinization of the blood. This was first noted after the administration of carbonates for therapeutic purposes, but has since been determined in several cases of diabetic acidosis of various degrees that have recovered without bicarbonate. In no

such case have we failed to find a discrepancy. The abnormal alveolar/plasma ratio persists for some days. This would make it seem unlikely that the disturbance was due to the fixation of CO_2 by the accession of alkali to the blood. It persists after all obvious hyperpnea is gone, so that an increase of the sensibility of the respiratory center can not be offered as a definite explanation as yet. The fault is not in the choice of the Fridericia method as it has also been observed with the Plesch-Higgins method. It seems unlikely that it is dependent on personal factors because of the absolute constancy of its relation to rapid changes in blood reaction and its absence at all other times. We are continuing studies to determine the cause of the phenomenon.

We believe that alveolar methods do not indicate accurately the reaction of the blood after recovery from acidosis. We also suggest that the value of respiratory quotients at such times may be questionable.

71 (1249)

Active immunization with sensitized and non-sensitized bacteria.

By HOMER F. SWIFT, M.D. and RALPH A. KINSELLA, M.D.

[*From the Medical Clinic, Presbyterian Hospital, Columbia University, New York.*]

In a previous communication[1] we reported that the serum of rabbits which had been injected with sensitized vaccine or living cultures of *Streptococcus viridans*, did not contain agglutinins, complement fixing antibodies or protective antibodies. Later similar results were obtained with sensitized vaccines of pneumococci, with the exception that the serum of animals inoculated with sensitized living pneumococci showed a rapid production of these antibodies. We then attempted to determine whether there was evidence of active immunity even though no antibodies could be demonstrated in the serum. It has been found impossible to immunize mice with green streptococci. Rats may be immunized with green streptococci, but the virulence of these organisms is so low that it is impossible to compare the results of immunity

[1] Homer F. Swift and Ralph A. Kinsella, PROC. SOC. EXP. BIOL. AND MED., 1915-16, XIII, 103.