tirely prevented. Since it is not easy to maintain the axial position of the wire in very narrow cells, it is better to use cells as large as possible without so greatly increasing the diameter as to secure troublesome convection currents. If the range of the concentrations to be measured is from 0 to 10% the 1 cm. cell with a wire of 0.005 cm. is satisfactory. For concentrations from 10% upward a cell of about 5 mm. diameter would be preferable.

As moist air has a heat conductivity different from that of dry air, it is necessary to dry the air in the reference cell and to dry the ether-air mixture as it is drawn into the cell. Calcium chloride has been found to be satisfactory for this purpose. It appears not to absorb any ether.

The temperature of the platinum wire carrying 0.2 ampere was calculated to be 68° above room temperature. There is good reason to believe that this temperature is certainly too low to cause ignition of the vapor, and in many hundreds of analyses with various percentages of ether no ignition has occurred. Since, however, the wire may break and a spark be formed, it is safer to disconnect the cell from the reservoir from which the specimen was drawn, before the current is turned on. If the cell is then left open at one end no harm would result from ignition.

5082

Further Note on the Relative Protection by Polymorphonuclear and Mononuclear Cells in Local Streptococcus Infection.

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Our investigations on the protective effect of granulation tissue (Gay and Morrison[1]) (Gay, Clark and Linton[2]) against a highly virulent streptococcus introduced into the pleural cavity of rabbits, led us to attribute a predominating if not exclusive rôle to mononuclears as compared with polymorphonuclear cells. In the acute stage of the inflammatory process when polymorphonuclears predominate the animals are fully as susceptible as normal controls. We have never denied that polymorphonuclears have a distinct protective energy with some bacteria and in some locations.

[1] Gay, F. P., and Morrison, L. F., *J. Am. Med. Assn.*, 1923, lxxx, 1298.

[2] Gay, F. P., Clark, A. R., and Linton, R. W., *Archiv. Path.*, 1926, l, 857.

Opie[3] in a recent article has reported similar experiments in the peritoneum and shown that early stages of inflammation (polymorphonuclear) prevent temporarily the invasion of the blood stream by the streptococcus, but further shows in complete confirmation of our results that recovery from such infection depends on the establishment of a later stage of inflammatory exudation. He does, however, suggest that our conclusions as to the negative or actually injurious effect of an excess of polymorphonuclear cells are open to another interpretation. It might be, to paraphrase and extend his remarks, that the sterile irritant (aleuronat) so long as it remains injurious is evidenced by polymorphonuclears and the introduction of a second irritant, the streptococcus, at this period leads to a fatal result. We have often thought of still another objection to our own conclusions, namely, that the pleural exudate in 20 hours is excessive in amount (5-10 cc. on the average) and might actually furnish a favorable location for the streptococcus to multiply. This particularly in view of our later observations that actual destruction of the streptococcus in the protected animals takes place not in the exudate itself but in the granulation tissue of the parietal pleura.

These considerations by Opie and by ourselves made it desirable to assure ourselves whether the accumulated polymorphonuclear cells in the pleura were in themselves endowed with any protective value. We have tried to determine the possible protective value of polymorphonuclear cells *per se,* unaffected by excessive exudation, in the following ways:

1. Aspiration of a large part of the 20 hour exudate in an irritated pleura before injecting the streptococcus does not result in protection.

2. Aspiration of exudate plus intravenous injection of antistreptococcus rabbit serum does not protect. Such addition of immune serum still further increases the protection afforded by granulation tissue (Gay and Clark[4]).

3. Infection of the opposite pleural cavity, where no histological change has followed aleuronat irritation of the other cavity, and where protection has been shown to occur by "transpleural mobilization of clasmatocytes" (Gay, Linton and Clark[5]) results in uniform fatality in the case of animals prepared only 24 hours previously.

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[3] Opie, E., *J. Immunol.*, 1929, xvii, 329.

[4] Gay, F. P., and Clark, A. R., *J. Exp. Med.*, in press.

[5] Gay, F. P., Linton, R. W., and Clark, A. R., PROC. SOC. EXP. BIOL. AND MED., 1926, xxiv, 23.

These observations make it still more convincing that polymorphonuclear cells are inactive in our experiments in which mononuclears have a marked protective effect.

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Sympathetic Activity After Prolonged Administration of Thyroxin.

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Innumerable "acute" experiments have been reported in the literature on the effect of thyroid administration on the pressor response of laboratory animals to adrenaline. The results have been in the main ambiguous. In contrast to these experiments, in this work an effort was made to insure a hyperthyroid condition of the animal by the long-continued administration of thyroxin.

Large vigorous cats were selected. Some were first fed Squibb's thyroxin tablets daily and then received daily intravenous injections of synthetic thyroxin (Hoffmann-La Roche). Others received the intravenous injections alone from the beginning of the period of thyroxin treatment. The total amount of thyroxin given per cat varied from 40 to 80 mg. The length of period of thyroxin administration varied from 35 to 57 days. At the end of that time blood pressure tracings were made from the carotid artery in the usual manner before and after the intravenous injection of a constant dose of adrenaline chloride (0.3 cc. per kg. of a 1:100,000 solution in saline). Paraldehyde (1.5 cc. per kg.) given by tube on an empty stomach was used as an anesthetic, as suggested by the work of Luckhardt and Koppanyi.[1] Sodium citrate (10% solution) served as an anticoagulant. Since some animals proved resistant to the action of thyroxin, only those were taken for the measurement of the pressor effect of adrenaline that showed a decided loss of body weight, 450 to 1000 gm. (1/3 to 1/7 of the original body weight). Five animals were finally chosen. Thirteen normal cats, treated exactly as those in the thyroxin series except for the administration of thyroxin, served as controls. Averages of the results for both series were as follows:

[1] Luckhardt, A. B., and Koppanyi, T., *Am. J. Physiol.*, 1927, lxxxi, 436.