genetic nature which frequently occur with primary atypical pneumonia, but are not necessarily limited to this disease. Many patients with atypical pneumonia show an increase in virus neutralizing antibodies without development of cold agglutinins or the other reactions. Evidence for the specificity of the virus neutralization in atypical pneumonia has been presented in this and other publications.[1] Agglutination of the indifferent streptococcus seems to occur with greater frequency in primary atypical pneumonia than in virus pneumonias of the psittacosis group. This finding suggests a possible specific association, at least in certain cases, of the streptococcus and virus in the pathogenesis of the disease.[15]

Data presented elsewhere[1] indicate that the virus of atypical pneumonia isolated and propagated in chick embryos is distinct in respect to host range, pathogenicity, and other properties from viruses of the psittacosis-lymphogranuloma group. The results presented in this paper show that these agents are also antigenically distinct as judged by complement fixation and neutralization tests on human sera.

Summary. In 11 cases of primary atypical pneumonia with increases in neutralizing antibodies for the virus propagated in chick embryos no significant development of complement-fixing antibodies for agents of the psittacosis group were found. Ten of the patients had cold hemagglutinins and 7 showed significant titers of agglutinins for the indifferent streptococcus No. 344. In an additional 10 cases of virus pneumonia where the presence of an agent of the psittacosis group was demonstrated by virus isolation or complement fixation, no significant development of neutralizing antibodies for the virus of atypical pneumonia occurred. The serum of one of the latter 10 patients showed streptococcal agglutination at a dilution of 1:20 without change in titer.

Dr. Gordon Meiklejohn collected many of the specimens and compiled much of the clinical data. The assistance of Miss M. Dorthy Beck, Miss Marilla Corey, Miss V. Lee Hanford, Miss Marjorie Hunt, and Mr. William van Herick is gratefully acknowledged.

[15] Thomas, L., Mirick, G. S., Curnen, E. C., Ziegler, J. E., Jr., and Horsfall, F. L., Jr., *J. Clin. Invest.*, 1945, **24**, 227.

15145

Experimental Intestinal Myiasis in Man.*

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The possibility of a true intestinal myiasis has been much discussed.

It was demonstrated that, when swallowed, the maggots of many different species may cause violent abdominal distress, vomiting, diarrhea and even systemic reactions. It is not quite clear however if these disturbances are caused by the accidental presence of dipterous larvæ and will clear up after their rapid elimination, or if some species of maggots can adapt themselves to intestinal environments, survive for a certain time, even multiply and cause a true intestinal myiasis.

A few observations seem to indicate that such possibility exists. Herms and Gilbert[1] described a case in which a woman apparently suffered from intestinal myiasis that had extended for many years, Thebauld[2] recovered living dipterous larvæ from the stool of a child suffering from typhoidal affection. In

* This study was made possible through the facilities of the Governmental Medical Services of the Belgian Congo.

[1] Herms, W. B., and Gilbert, Q. O., *Ann. Int. Med.*, 1933, **6**, 941.

[2] Thebauld, V., *Arch. Parasit.*, 1901, **4**, 353.

many other cases different species of dipterous larvæ such as *Musca domestica, Fannia canicularis, Fannia scalaris,* Muscina, Sarcophaga, Calliphora, Eristalis, *Apiochaeta scalaris, Piophila casei,* etc., some of them dead, some alive, were recovered from stools or vomitus, but none of these observations seem to prove that they dealt with a true intestinal myiasis.

Description of the Experiment. Sixty human volunteers of Katanga, Belgian Congo, were fed living maggots of *Musca domestica,* Calliphora, and Sarcophaga. All larvæ were administered in large gelatin capsules on a fasting stomach, with 2 glasses of water. Each man received 20 larvæ of a single species. The reactions that followed can be summarized in 5 groups.

Group 1. In 18 cases vomiting occurred very shortly after administration of larvæ and the totality of maggots were recovered from the vomitus, some still in capsules, most of them free, with few exceptions all larvæ were alive. Vomiting subsided immediately after elimination of larvæ.

Group 2. In 12 cases gastro-intestinal disturbances occurred a few hours after ingestion. Late vomiting brought a few dead larvæ, while many dead maggots were recovered from diarrheal stools. The symptoms cleared 24 hours after administration.

Group 3. In 16 cases no vomiting occurred, but diarrhea following slight intestinal distress brought many larvæ, all dead, in the first 24 hours. All symptoms disappeared after 48 hours.

Group 4. In 4 cases, no vomiting, but severe nausea and abdominal cramps with diarrhea occurred in the first 24 hours. Many dead and occasional living maggots were recovered from the stools in the first 36 hours. All symptoms disappeared after 48 hours.

Group 5. In 10 cases no disturbances were caused by the larvæ. Many dead maggots were recovered from the stools during the 48 hours that followed the ingestion.

In all 5 groups the 3 dipterous species were represented, thus indicating that different reactions were dependent rather upon the individuals than upon the species of larvæ administered.

Of the 60 men, 4 remained under observation only 3 months, 22 for 6 months, and 34 could be observed for 12 months. Stools of all men were examined regularly once a month and on each occasion when gastrointestinal symptoms occurred. No dipterous larvæ were found in any of them. The occasional gastro-intestinal disturbances disappeared spontaneously in all cases, the percentage of which was not higher than in the local population not under experimentation.

Discussion. Causey[3] who fed larvæ of a number of species to dogs, never observed any of the larvæ survive in the stomach for longer that 3 hours, and Chandler[4] believes that in cases when larvæ survive the passage through the gastro-intestinal tract, it is associated with low HCl in the stomach or a particularly rapid passage into the intestines. Since in our experiment larvæ were administered on a fasting stomach to avoid high acidity, this condition together with accelerated peristalsis due to the presence of maggots, might have permitted the passage of a few living larvæ.

Komarek,[5] who conducted a series of experiments with dipterous larvæ, has shown that larvæ need free oxygen, but this seems not to be true in the case of gastrophilus, a parasite of the alimentary canal of horses, where larvæ live until mature and ready to pupate.

In the case of Herms and Gilbert, not only a survival of larvæ is noticed, but this case implies intestinal pedogenesis, since the duration of the recovery of maggots was lasting far beyond the normal time of larvæ evolution of Calliphora, Lucilia, and Sarcophaga, and it is difficult to understand such abnormal multiplication.

Conclusions. From 60 volunteers fed with living maggots of *Musca domestica,* Calliphora, and Sarcophaga, under conditions to avoid their destruction in the stomach, only 10 failed to have symptoms of gastro-intestinal disturbance.

In 50 cases men had nausea, vomiting, intestinal cramps and diarrhea together or as

3 Causey, O. R., *Am. J. Hyg.,* 1938, **28**, 481.

4 Chandler, A. C., *Introduction to Human Parasitology,* 1936, 6th ed.

5 Komarek, J., *Mém. du Musée Roy. d'Inst. Nat. de Belg.,* 1936, **3**, 23.

separate symptoms, but all symptoms disappeared within 48 hours following the elimination of the larvæ, of which only a few were found alive in the vomitus and stools.

These findings seem to indicate that though temporary gastro-intestinal distress may follow the ingestion of such dipterous larvæ as *Musca domestica,* Calliphora, and Sarcophaga, they do not produce a true intestinal myiasis in man.

15146 P

Impedance Changes in the Cerebrum Following Concussion.*

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Since cellular injury is associated with impairment of cellular surface membranes (Osterhout[1]), it seems reasonable to surmise that cerebral concussion produces similar changes in the cerebrum. The electrocorticogram does not permit definite conclusions (Walker, Kollros, and Case[2]) regarding the cellular surface films, since other factors may affect it. We approached this problem by measuring the A.C. impedance of the cerebral hemispheres in 14 cats *in vivo* and in 50 guinea pigs immediately post mortem, at a low frequency (l = 570 cycles per second) and a high frequency (h = 5100 cycles), and computing

$$\Delta = \frac{(\text{Conductivity}_h - \text{Conductivity}_l) \times 100}{\text{Conductivity}_l}.$$

Δ is a measure of the polarizability. In agreement with Gildemeister's[3] theory, we have given experimental evidence on artificial membranes and frog skin (Spiegel and Spiegel-Adolf[4]) that decrease of polarizability is associated with increase of electrolyte permeability. Acceleration concussion (Denny-Brown and Russell[5]) was produced by means of a pendulum, under nembutal anesthesia.

Results. In control experiments on cats Δ remained constant for at least 2 hours when the experimental conditions were identical except for omission of the blow. Blows inducing signs of concussion, typical changes of blood pressure, transitory apnea, loss of the corneal-, light-, pinna reflex, induced a mean decrease of Δ of 23.4 $\pm$ 1.6%. Usually it became most marked within ½-1 hour after the blow. Then a slow rise of Δ from the minimum began to develop. However, 4 hours after the blow a definite decrease of Δ was still noticeable. These changes of Δ did not appear if the pendulum struck a dead animal. It should be emphasized that the decrease of Δ developed independently of the fluctuations of conductivity, which were too irregular to permit definite conclusions.

Since in these experiments the skull had to be trephined for introduction of the electrodes before concussion, these measurements were repeated in guinea pigs concussed with the skull intact. Δ was measured only following the blow. After decapitation in various stages of concussion (see below), the head, with the cerebrum exposed, was placed in an incubator (38°C). In Group I (13 nonconcussed, but otherwise identically treated animals; 10% nembutal .2 cc per lb) the mean Δ was

* Aided by a grant from the John and Mary R. Markle Foundation.

1 Osterhout, W. J., *Injury, Recovery, and Death in Relation to Conductivity and Permeability,* J. B. Lippincott Co., Phila., 1922.

2 Walker, A. E., Kollros, J. J., and Case, T. J., *J. Neurosurg.,* 1944, **1**, 103.

3 Gildemeister, M., *Handb. d. norm. u. path. Physiol.,* 1928, **8**, II, 693.

4 Spiegel, E., and Spiegel-Adolf, M., *Am. J. Psychiat.,* 1936, **92**, 1145; *Am. J. Physiol.,* 1941, **133**, 459; Spiegel-Adolf, M., *J. Gen. Physiol.,* 1937, **20**, 695; Spiegel-Adolf, M., and Spiegel, E., *J. Colloid Science,* in press.

5 Denny-Brown, D., and Russell, W. R., *Brain,* 1941, **64**, 93.