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.

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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

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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.

10.84 ± .16† (M_1‡). In Group II, 11 animals were subjected to severe blows (energy 21.3-25.7 foot-pounds, velocity of striking piece 23.8-26.2 feet per second) and decapitated 1-2 minutes after the blow during apnea and loss of the corneal-, pinna-, and righting reflexes; the mean Δ was 8.91 ± .27 (M_2). Groups III to V were subjected to 1-3 slighter blows (energy 3.2-17.0 foot-pounds, velocity 10.5-21.4 feet per second), which were however still sufficient to produce typical concussion. In Group III, 12 rats were decapitated 2-8 minutes after the blow; the mean Δ was 11.21 ± .23 (M_3); Group IV, 7 rats, decapitated 10-60 minutes after the blow, showed a mean Δ = 9.52 ± .18 (M_4); Group V, 7 rats, decapitated 2-5 hours after the blow had a mean Δ = 11.05 ± .26 (M_5). The t values§ for the differences M_1-M_2, and M_1-M_4 were 5.6 and 3.3 respectively, *i.e.*, above the values necessary to consider these differences significant, while M_3 and M_5 lay within the normal variability. Thus only severe blows produced a definite decrease of Δ within the first minutes following the blow. Following slight, but still concussive blows a decrease of Δ was not demonstrable within the first minutes (Group III), but only 10-60 minutes following the blow (Group IV), at a stage when the apnea and disturbance of reflexes had long subsided. Group V shows that Δ returned to normal several hours after the blow.

Analysis. These changes of Δ were also observed in cats kept under artificial respiration, so that cerebral anoxia due to apnea is, at most, only a contributory factor. Decrease of Δ developed with as well as without initial rise of femoral blood pressure. However, the subsequent fall of blood pressure below the initial level may be at least partly responsible for the impairment of the cells. Accumulation of lactic acid probably is of little importance, since artificial acidosis lowers the polarizability of the brain only slightly if at all (Spiegel and Spiegel-Adolf[6]). Similarly to some other postconcussional changes (chromatolysis, Windle, Groat, Fox[7]), appearance of nucleosides in the cerebrospinal fluid (Spiegel-Adolf, Henny, Wycis, and Spiegel[8]), increase of brain volume (White, Brooks, and Goldthwait[9]), the injury of the cellular surface membranes indicated by the decrease of polarizability becomes manifest or reaches its maximum at a stage in which the transient symptoms immediately following the concussion have already disappeared. The impairment of the cellular surface films by itself apparently does not suffice to explain the genesis of these symptoms. Rather it may be of importance in the development of symptoms appearing some time after the blow (confusion, headache, vertigo, etc.).

† Figure after ± equals probable error of the mean.

‡ This value is not comparable to that previously obtained by us (*J. Nerv. Ment. Dis.*, 1941, **93**, 750) due to differences of the electrodes.

§ t = difference between the means divided by the standard error of the difference.

[6] Spiegel, E., and Spiegel-Adolf, M., *J. Nerv. Ment. Dis.*, 1939, **90**, 188.

[7] Windle, W. F., Groat, R. A., and Fox, C. A., *Surg., Gynec., and Obstet.*, 1944, **79**, 51.

[8] Spiegel-Adolf, M., Henny, G. C., Wycis, H. T., and Spiegel, E., *Fed. Proc.*, 1945, **4**, 104.

[9] White, J. C., Brooks, J. R., Goldthwait, J. C., and Adams, R. D., *Ann. Surg.*, 1943, **118**, 619.