seem to be proof positive that the disturbance was not of an organic nature, at any rate not totally so. It is quite conceivable that pathological changes may be present in the region of the bundle and primary branches which would require very little additional change for the precipitation of block. Under such conditions, however, one should expect to find slight transition periods and increasing block. A mechanical factor could quite conceivably add the necessary additional sudden and temporary factor. That there is a mechanical factor playing a part is substantiated by the fact that the periods of normal conduction in these patients were induced by respiratory maneuvers. The Müller's or Valsalva experiments, either of which were accompanied by the sudden conspicuous changes in intraventricular conduction, were apparently concurrent with the cardiac dilatation.

The clinical value of the observations lies in the realization of the fact that the serious prognostic significance of permanently organically produced bundle branch block is not to be applied until the possibility of a temporary disturbance is ruled out. Furthermore, as generally recognized, patients with bundle branch block are known to die suddenly at the slightest provocation, especially during a minor therapeutic or surgical procedure. Any blood pressure may be enough to press the already over exerting heart to a point of causing ventricular fibrillation and exitus. During the periods of normal conduction these patients are relatively good surgical risks. In one of our patients the discovery of the transient nature of the condition led us to follow her carefully electrocardiographically and to subject her to 2 successful surgical operations, a Cesarean and a later sterilization, during periods in which she was not blocking, without precipitating any evidence of undue cardiac embarrassment.

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Subarachnoid Spinal Adhesions: An Attempt Experimentally to Produce and Prevent Their Recurrence.

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Subarachnoid spinal adhesions may at times produce clinical symptoms. Such a case has been observed by us in which following a laminectomy for paraplegia numerous adhesions of the spinal subarachnoid space were found. These adhesions were divided, but

as adhesions involving all serous membranes are apt to recur, the possibility of a recurrence of these subarachnoid adhesions was considered. As Herrmann and Musser,[1] and Ochsner and Herrmann[2] had successfully prevented the recurrence of adhesions in the pericardial cavity and Ochsner and Mason[3] in the peritoneal cavity experimentally by the use of digestants, it was thought possible that such procedures might be employed in the prevention of the recurrence of spinal subarachnoid adhesions.

Our purpose in this study was to produce subarachnoid adhesions by introducing an irritant into the spinal subarachnoid space in the lower thoracic region of dogs, later to divide these adhesions, and to attempt to prevent their reformation by introducing a digestion solution. We have been able to find no record of a previous attempt of this kind, although Dr. E. R. Schmidt, of the University of Wisconsin, has employed a digestant in the subarachnoid space in one case in order to attempt to prevent the reformation of adhesions.

The experimental animals employed were dogs. An attempt was made to inject the irritant into the spinal subarachnoid space by spinal puncture, but because of the difficulty encountered in performing a lumbar puncture, this method was abandoned. A formal laminectomy was employed in the remainder of the animals, and the irritant was injected into the subarachnoid space by means of a syringe and cannula. The animals were prepared in the usual manner; ether was used as the anesthetic agent. Following the injection of the irritant the wounds were closed in layers without drainage. Twenty-six dogs were operated upon; 6 died within 3 days and 7 of the remaining 20 died within 4 weeks. The 7 animals dying within 4 weeks all showed evidence of a paraplegia following operation and rapidly developed decubitus ulcers associated with marked emaciation. At autopsy in this group of animals an advanced myelitis at the site of operation was present in each case, and the spinal cord was so liquefied that no specimen could be obtained for microscopic study. No gross adhesions were demonstrable.

The 13 additional animals survived. Although 1 animal died within a week, it is reported in detail because in this particular animal a second operation was performed.

Protocol. Dog A1-3. August 7, 1928, 0.5 cc. aleuronat solution

[1] Herrmann, George, and Musser, J. H., PROC. SOC. EXP. BIOL. AND MED., 1928, XXV, 314.

[2] Ochsner, Alton, and Herrmann, George, *Arch. Surg.*, 1929, xviii, 365.

[3] Ochsner, Alton, and Mason, Frank, PROC. SOC. EXP. BIOL. AND MED., 1928, XXV, 524.

injected within the dura, the lower dorsal region, following a laminectomy performed under ether anesthesia.

August 8—Animal is paraplegic.

From August 7 to August 14 the animal lost weight rapidly and became emaciated.

August 14—Operation under ether anesthesia. Previous laminectomy wound is opened under sterile precautions. The dura is opened, and numerous adhesions are found involving the subarachnoid space. These adhesions are divided and 0.5 cc. of a 1:1000 solution of papain is placed within the dura. The dura is, however, so friable that a satisfactory closure is not possible. The wound is closed. The animal died that night.

The remaining 12 animals survived for periods varying from 7 to 51 weeks, an average of 23.5 weeks. Six were paraplegic and showed evidence of myelitis at autopsy; 6 were not paraplegic and showed no evidence of myelitis at autopsy. Those which survived the longest were in general the ones that were not paraplegic, although 1 paraplegic animal survived for 28 weeks. The average survival for the paraplegic animals was 19 and for the non-paraplegic animals, 28 weeks. One of this latter group was killed in a fight when apparently in good condition. The amount of aleuronat solution varied from 0.1 cc. to 1 cc. Of the 6 paraplegic animals, 2 received 0.1 cc., 2 received 0.3 cc., 1 received 0.4 cc., and 1 received 1 cc. Of the 6 non-paraplegic 2 received 0.1 cc. of aleuronat, 3 received 0.3 cc. of aleuronat, and 1 received 0.5 cc. of aleuronat. None of the dogs that died early showed any adhesions except the one reported in detail, AL-3, and in this one there was a slight infection of the wound which may have been responsible for the adhesions. Of the 12 dogs which survived more than 4 weeks, 2 had very fine adhesions, whereas 9 showed no adhesions. In one animal, AL-4, at autopsy no adhesions in general were found, but along the line of incision the cord was firmly adherent to the overlying muscle. The dura in this particular animal could not be identified. This animal had been paraplegic.

Microscopic examination of the sections from this group of 12 animals did not show any evidence of adhesions. All animals showed some paresis following operation, but in those reported as non-paraplegic the paresis cleared up in varying degrees. In a few a moderate degree of paresis persisted, but in the majority there was apparently a complete recovery.

No valuable conclusions can be drawn from this experiment. It appears dogs do not tolerate foreign substances within the sub-

arachnoid space. Following the injection of an irritative substance into the subarachnoid space evidence of damage to the cord was seen in all animals and in only 6 was there recovery to such an extent that the use of the lower extremities returned. Only 4 animals developed adhesions and in 2 of these adhesions were scant and extremely fine. The failure to produce satisfactory adhesions prevented the performance of the second part of the experiment; *i. e.*, the prevention of the reformation of adhesions.

The occurrence of myelitis in all animals may have been due (1) to mechanical pressure and (2) to chemical irritation. We are inclined to accept the latter view (1) because of the extreme degree of cord destruction of those animals that died early and (2) because there was little clinical difference between the animals which received large and small amounts of aleuronat.

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Experimental Polyneuritis in Chickens Given Jamaica Ginger.

J. H. WATKINS. (Introduced by J. H. Musser.)

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Four apparently healthy chickens weighing approximately one kilo each were used in this experiment. Jamaica ginger obtained from a community where there were many patients with peripheral neuritis was used. Two of the chickens received daily doses of 2 cc. fluid extract of ginger and the other 2 received an equal amount of 83% ethyl alcohol. The chickens were kept in a cage out of doors and their diet consisted of cracked corn and oats. The chickens receiving the alcohol were accidentally killed 10 days after beginning the experiment.

Thirty-eight days after receiving the first dose of ginger and after each chicken had been given a total of 70 cc. there were no signs of muscular weakness. There had been some loss of weight during this time. On the thirty-ninth day the chickens exhibited slight motor weakness and loss of coordinating power in both extremities. Feeding ginger was discontinued at this time. During the next 2 days there was a progressive motor weakness and the difficulty in walking or standing was marked. Control of the feet was completely lost and the toes were turned under the feet when an attempt was made to walk. The legs rather than the feet were used