standards, such as will debar as few promising candidates as possible and yet limit future risks. It cannot be too strongly urged that decisions in scholastic cases are decisions for a lifetime, and not merely decisions for the three years or so of military service. It is therefore better in every way to stop a dubious case at the very beginning, when a diversion of interest is easy, rather than to risk the grievous hardship of a stoppage in the future, with the possible wrecking of an established career.

REFERENCE.

¹ The Consequences of Myopia as an Industrial Disease of the Eyes, *Trans. Ophthalm. Soc. U.K.*, 1922.

THE EFFECT OF ANAESTHETICS ON THE LUNGS.

(Preliminary Communication.)

BY

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In the issue of this JOURNAL for November 11th, 1922, was published the report of the discussion at the Annual Meeting of the British Medical Association on broncho-pulmonary complications following operations under anaesthesia, and it was a strange coincidence that the succeeding article should be on the effect of anaesthetic vapours on tissues, in which Flemming has collected a very interesting series of experiments which show their detrimental effects on tissues and living organisms in general. In the first article stress was placed on the possible irritant effect of the vapours on the lungs; in the second no special emphasis was laid on the effect on the lung tissue.

It does not seem to be appreciated that the lung tissue may, apart from mere irritation, suffer serious damage as a result of actual contact with the vapour, apart from concentration in the blood.

Until quite recently knowledge of the function of the lung tissue was comparatively small, apart from the fact that the lungs were adapted for the supply of oxygen to the blood. The function of the bronchial muscles has been largely ignored and the physiology of the pulmonary circulation has been avoided, largely because of the experimental difficulty involved in its study and the negative results obtained by several well-known investigators. From book to book it had become quoted that the vasomotor mechanism in the lungs was negligible. Why there should be such an amount of muscular tissue in the lungs remained unexplained.

Much controversy centres round the action of adrenaline on the pulmonary circulation, as it is accepted that generally this drug acts only on tissues supplied by the sympathetic nervous system. While many observers have obtained negative or indefinite results, others, including Plummer, Starling, and Sharpey Schafer, have brought forward good evidence that adrenaline constricts the pulmonary vessels as it does the systemic. This suggests that the vessels have a sympathetic control, although in the light of more recent knowledge referred to below, this has not been reinvestigated.

By the introduction in 1919 by Sharpey Schafer of a convenient method of recording the pressure in the pulmonary artery new light has been thrown on the pulmonary circulation, and amongst the findings has been the fact that the effect of drugs which act on the circulation was greatly modified by anaesthetics.

I first noted that the effect of drugs on the pulmonary circulation would be much more easy to demonstrate on decerebrate animals, or animals under light ether anaesthesia, than on those anaesthetized with chloroform. For example, it was shown that the action of amyl nitrite on the pulmonary circulation can be prevented by deep anaesthesia. An investigation is in progress* to find out to what extent the functional activity of the lung tissue may be impaired by anaesthesia. It has already been shown that the action of histamine, now acknowledged to be a pulmonary constrictor (Dale and Laidlaw, Morris), may be similarly abolished.

There is evidence, too, that the action of adrenaline on the pulmonary circulation may be prevented while the systemic effect may be unaffected. This has occurred where all possibility of backward pressure through an incompetent mitral valve and cardiac effects have been excluded.

When the literature of the subject is surveyed it is found that the most probable reason for the negative or variable

results of different investigators, largely in the days when chloroform was in more common use, was the effect of the anaesthetic. The effect of anaesthetics is not necessarily excluded in lung perfusion experiments, as the animal is under an anaesthetic immediately prior to death, and with the cessation of blood supply and anaesthetic the anaesthetic has all the better chance to produce its bad effects. In the positive experiments of Sharpey Schafer and of Starling, referred to above, volatile anaesthetics were not used except in the preliminary anaesthetization, but chloral and chloralose respectively.

This is not the first time that attention has been drawn to the action of anaesthetics on the lungs. In this JOURNAL, in 1880, McKendrick, Coats, and Newman described complete stoppage of the pulmonary circulation of the frog as the result of chloroform anaesthesia, with actual destruction of the lung epithelium.

When the evidence put forward by Flemming is considered, the importance of the local effects of anaesthetics on the lungs will be seen. He might also have added to his list the fact that a piece of muscle exposed to chloroform vapour is killed with great rapidity, a fact which is made use of in the thermopile experiments of Hartree and Hill on heat production.

My results so far agree with those of Flemming on isolated tissues. Recovery from the pulmonary effects occurs after ether, but after deep and prolonged anaesthesia with chloroform recovery is the exception. The actual death of tissue in the lungs as a result of the anaesthetic does not appear to be a remote possibility—indeed, may be easily considered the natural sequel. It is outside the scope of this paper to consider the evil results of such lethal action, but obviously the symptoms discussed at the Glasgow meeting might readily follow. That the absorption of toxic products from such dead tissue should produce delayed chloroform poisoning seems most reasonable.

Another function shown to be abolished is that of the bronchioles, which limit the amount of air passing in to the alveoli. Brodie and Dixon first demonstrated that the vagus no longer caused bronchiole constrictions under deep anaesthesia. The muscles or their nerve supply were considered to be paralysed by direct absorption of the anaesthetic through the mucous membrane. I have demonstrated that the action of adrenaline on the bronchioles may similarly be prevented by deep anaesthesia.

Now the abolition of the bronchiole function must necessarily throw a greater strain on the alveoli and cause them to be exposed to particles which otherwise would not reach them. Even if actual death of tissue does not occur, the mere loss of this protective mechanism temporarily must expose the lung to possible damage.

The investigation is proceeding, but from the results already obtained it is seen that the local effects of the common anaesthetic cannot be ignored, not only from the purely experimental standpoint, but also from that of the clinician. Although anaesthetics may be given to thousands of cases yearly without effect, it must be urged that we are still without an ideal anaesthetic for general use.

From the evidence given it is seen that deep or prolonged anaesthesia with ether, and especially with chloroform, should be avoided whenever possible. It is suggested that narcotics should be more extensively used to supplement volatile anaesthetics.

INTESTINAL OBSTRUCTION FOLLOWING ACUTE APPENDICITIS AND PERITONITIS.

BY

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In the case here reported adhesions must have begun to form immediately after the first operation. The second operation was undertaken because of the increasing distension and in spite of the fact that there was no vomiting and that the patient's bowels had moved on the previous day. The part of the ileum opened on the fifteenth day was below the site of obstruction, which had been liberated at the second operation; the opening acted simply as a safety valve, and by relieving distension allowed the intestines to recover tone.

On February 6th, 1922, a man aged 20 was sent in to Stirling suffering from acute appendicitis. His illness had started suddenly thirty hours, approximately, before admission. When I first saw

* The research is being assisted by a grant from the British Medical Association.

him he had a temperature of 101° and a pulse of 112. He was very tender all over the lower half of the abdomen, and there was marked muscular rigidity. He gave no history of previous attacks, and in all other respects he was healthy and well developed. He was operated on a few hours after admission, and a gangrenous appendix with two concretions was removed. There was a considerable quantity of pus in the pouch of Douglas, and there was marked injection of all the peritoneal surfaces which came into view. A corrugated rubber drain was introduced into the pouch of Douglas. He was given normal saline and 6 per cent. solution of glucose alternately every two hours by the bowel, and morphine 1/2 grain on the first night. On the day following he was able to take liquids by the mouth and had no vomiting, so the saline and glucose enemata were stopped. His temperature came down to normal on the morning after the operation, and never rose again above 100°. His pulse came gradually down to 96 on the third day, and to 80 to 84 on the fifth day. The tenderness and rigidity also passed off in the first three days. In all respects he appeared to be making a good recovery, except that from the evening of the second day he began to complain of intermittent pain, of slight distension, and of a desire to have his bowels moved. Because of this constant demand on the part of the patient, on the evening of the third day he was given a small turpentine enema, which was returned coloured with a little constipated faecal matter. His distension continued slowly to increase, and his discomfort was more marked. He was given castor oil on the afternoon of the fourth day, and another turpentine enema. The bowels moved slightly that night, and they moved three times the next day, aided by 1/2 grain of calomel hourly up to 2 grains and another enema. Still the distension did not diminish and his discomfort was no less. He was given nupenthe mxxv on the night of the sixth and seventh days. He still had no vomiting. His tongue by this time was dry and coated. His pulse remained at about 88, and his temperature never rose above 99°.

On the seventh and eighth days he had several small motions, mostly liquid, but his distension increased and also his discomfort, and owing to the distension the incision for the removal of his appendix stretched and broke down. Still no vomiting.

On the ninth day I opened the abdomen in the middle line and found the small intestine very much distended above a dense adhesion which bound the small bowel firmly to the mesentery of the pelvic colon. There were two other adhesions between the small intestine and the floor of the pelvis. These adhesions were all separated and the raw areas were covered over. A small quantity of pus was found in the pouch of Douglas. The distended bowel was all replaced in the abdomen, and I put a rubber drain into the pouch of Douglas, which was removed the next day. He had some sickness after this operation, and some dilatation of the stomach, which improved rapidly with gastric lavage. He was given pituitrin, and enemata, and castor oil by the stomach tube thirty hours after the second operation. The next day he had a copious liquid evacuation. He went on fairly well for a day or two, but never was quite comfortable and his distension did not diminish. Then he got worse again, and on February 21st—that is, fifteen days after the first operation—I opened the ileum, which was presenting at the original appendix incision, and stitched a small rubber tube into it. This tube drained freely at once and continued to do so for several days. His distension came gradually down and his bowels moved freely two days after. He made a slow recovery, being troubled with diarrhoea for some days, but he was able to go home six weeks after the date of his admission with all his wounds healed. By the third week of April he was wonderfully well. There was, however, a hernia at the site of the appendix incision.

Twenty years ago we were taught to give aperients and enemata to similar cases on the second day after operation. Now the practice is to withhold all stimulants to peristalsis either by the mouth or rectally till "convalescence is well established."¹ Mr. H. W. L. Molesworth,² in an excellent article on the after-treatment of acute abdominal disease, recommends giving no purgative till after the patient's bowels move, and he advocates a fairly liberal use of morphine. During the last two years or so it has been my practice not to give a purgative till the fifth or sixth day after operation. I give morphine readily for the first twenty-four hours; after that I try to avoid giving it.

We have had several cases of intestinal obstruction recently following pelvic peritonitis, and these cases have made me wonder if it is wise to withhold purgatives so long in all such cases. Is it not possible that by keeping the bowel at rest for six or seven days, with a fairly free use of morphine, we encourage the formation of adhesions and give them time to contract and so pave the way to intestinal obstruction?

REFERENCES.

¹ Keen's *Surgery*, vol. viii, p. 229. ² BRITISH MEDICAL JOURNAL, February 11th, 1922, p. 218.

DR. ARNOZAN, professor of clinical medicine in the University of Bordeaux, has retired from his chair after having spent fifty years in the practice and teaching of medicine.

The fifth Congress of the French Society of Orthopaedics will be held in Paris on October 12th. The subjects set down for discussion are: pes cavus; bone cysts, excluding hydatid cysts; congenital elevation of the scapula.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

THE CORRECTION OF MALPOSITIONS: THE RATIONALE OF THE PADDED BINDER.

In using the method of the padded bandage to produce a favourable version or rotation the aim is to apply localized pressures in the same direction as we should choose for pressure applied manually. The difference is that by the pads the pressure is exerted with less intensity but over a longer time than by the hand. The mechanics of the problem is very simple. The binder applied in a circle exerts nearly equal inward pressure at each point of its course, but when pads are applied under it the inward pressures through the pads are greater. Being applied at opposite points of the child's body these pressures give the mechanical couple which is necessary if we are to produce a rotation. To obtain the greatest number of successes it is important to individualize the cases by selecting the most effective points for the placing of the pads and the best form of each pad for the application of pressure to the underlying part of the child's body, but the essential in every case is to secure two pressures which are in opposite directions at points as remote as possible from the line which is the axis of the rotation we desire.

Dundee.

R. C. BUIST, M.D.

HYSTERICAL ELBOW.

The case of hysterical simulation of fracture associated with hyperextension of the elbow-joint, recorded by Drs. H. C. Woodhouse and F. Carlton Jones (December 16th, 1922, p. 1171), induces me to publish the following similar case:

On May 6th, 1921, an intelligent girl, aged 14, applied to me for treatment at Bootle Borough Hospital. The history was that she had had a slight fall on some stairs and had struck the back of her right elbow, which at once became stiff and could not be flexed.

The elbow was found to be hyperextended to about 15 degrees, and the arm was rotated outwards, the forearm fully supinated, and the wrist and fingers extended. The patient complained of pain in the elbow-joint, but no sign of local trauma was found. When attempts were made to flex the forearm the triceps was felt to contract and the amount of contraction was found to vary with the force applied. The left elbow could readily be extended to a similar degree as the right, but no other joint possessed an abnormal range of movement. The patient volunteered the information that this was her first day in domestic service.

A diagnosis of hysterical elbow was made, and I proceeded to take a photograph of the condition. While adjusting the camera tripod I noticed that the gross hyperextension gradually disappeared, and that the forearm became pronated. Pretending that I was about to move her chair into a better light I seized the girl's arm, and—without difficulty and against no resistance—fully flexed the elbow.

On June 11th, 1921, the patient returned with a history that she had gone back to domestic service and had struck her arm again, when the condition had recurred. On this occasion there was less hyperextension, and the wrist and fingers were in no fixed position, but again the arm was rotated outwards and the forearm supinated. I suggested to the patient that the spasm would pass off when the pain from the blow had subsided. I persuaded the mother that the condition was hysterical, and she promised to show no sympathy: at the same time she was advised to find more congenial work for the girl. The patient did not again apply for treatment while I was at the hospital.

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THE ETIOLOGY OF OPTIC ATROPHY.

WITH reference to the report of the discussion on the etiology of optic atrophy at the recent meeting of the Section of Ophthalmology of the British Medical Association (JOURNAL, December 16th, 1922), and more especially with reference to Dr. C. O. Hawthorne's remarks on the neuritis of acquired syphilis, which he said was a very exceptional event in the early stage of the disease, the following case may be of interest.

A woman, aged 25, was seen in the Finger Klinik here a few days ago, when she showed a typical syphilitic leucoderma of a week's duration. Inquiry elicited the information that infection had taken place about four months previously. The cerebro-spinal fluid gave a positive Wassermann reaction. Three days later the same patient was encountered in the ophthalmological department, and then showed a typical acute double optic neuritis.

On the authority of Professor Kyrle, of the Finger Klinik, it can be stated that most cases of syphilitic leucoderma show