SYMPATHETIC BLOCK

PROPOSED THERAPY IN TRAUMATIC SHOCK

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The purpose of this paper is to draw practical conclusions from recent experiments (Engel and Forrai, 1943; Engel, 1943) and to propose regional sympathetic block as a preventive and curative method in cases of extensive crush injuries. Though my proposal is based on animal experiments there is enough clinical evidence to support my views.

The result of the experimental work can briefly be summarized as follows: In "traumatic shock" the rate of filtration through the capillaries in tissue adjacent to trauma, and probably also in the traumatized tissue itself, is greatly increased in the first 1 to 5 hours following trauma, and reduced afterwards. The increased rate of filtration, suggesting increased capillary permeability, is thus restricted to the traumatized area and is not generalized over the whole body. It was further shown that by regional sympathectomy it is possible to reduce considerably the increased rate of filtration caused by trauma.

These results were obtained by a new method consisting of a constant intravenous infusion of a dye and a simultaneous perfusion of the two knee-joints with Ringer solution under various conditions. The dye appearing in the perfusate passes through the "articular barrier"—i.e., the capillary endothelium, the synovial membrane, and the thin layer of tissue or space between the two.

The practical significance of these experiments is that if it is true that capillary permeability is one of the most important initiating factors in the development of "shock," then every procedure that is capable of reducing capillary permeability is bound to have a beneficial effect.

Technique

The simplest and most practical way of paralysing sympathetic action and thus reducing capillary permeability in the traumatized area without damaging the patient is the paravertebral infiltration of the regional ganglia with novocain. This should be carried out as soon as possible—in any case, within the first 1 to 3 hours after trauma. We have seen that the period of increased capillary permeability lasts only 1 to 5 hours, to be followed by a period of decreased permeability. No effect can therefore be expected after 5 hours. These results were obtained in the cat; it is therefore possible that the time limits mentioned do not apply exactly to human beings.

An anaesthetic of short effect, preferably novocain, should be used, because the second period of decreased permeability does not develop in the sympathectomized limb, and it is very likely that a prolonged sympathetic block may have a similar effect to that of sympathectomy and fail its purpose. If the lower extremity is crushed the first to fourth lumbar and the first sacral ganglia, if the upper extremity is involved then the stellate and the first and second thoracic ganglia, have to be injected. About the technique of the sympathetic block nothing more need be said; it is standard method.

Observations in the Literature

I have no personal experience of sympathetic block as a prophylactic or curative method against shock. There are, however, numerous clinical observations which support the usefulness of this method.

Several authors (Philipowicz, 1942, and others) reported on the excellent results obtained by sympathectomy in cases of frostbite. The inflammatory exudate and odour diminish immediately after operation, the moist gangrene becomes dry, and the demarcation is accelerated.

It is also a well-known fact that abdominal operations performed under lumbar or splanchnic anaesthesia cause much less shock than those under general anaesthesia, in spite of the falling blood pressure, which would be expected to favour the development of shock. The fact that shock does not develop so readily under lumbar and splanchnic anaesthesia is possibly due to the reduced capillary permeability in the abdominal tissues, temporarily excluded from sympathetic innervation.

The idea that the sympathetic has an influence on the development of traumatic shock is not new. The literature on the subject has been reviewed up to 1941 by Harkins (1941). We shall therefore consider here only the most important papers which have some bearing on capillary permeability. It was Cannon (1923) who first suggested that the hyperactivity of the sympathetico-adrenal system, excited by sensory-nerve impulses, is an important factor in traumatic shock. This theory was based partly on clinical observation, partly on experiments showing that continuous sympathetic stimulation or hyperadrenalinaemia led to a decreased blood volume and low blood pressure. It was assumed that a prolonged arteriolar constriction resulted in a capillary anoxaemia with consequently increased permeability, fluid loss, and final shock.

The effect of adrenaline in various concentrations on blood pressure, blood volume, haemoglobin concentration, etc., was examined by several authors. Freeman and co-workers (1936, 1941) produced a prolonged hyperactivity of the sympathetic nervous system by injecting adrenaline in physiological doses or by spontaneous emotional activity of the pseudoeffective state. Both procedures resulted in a decreased circulating blood volume. This decrease did not occur when the vasoconstrictor action of the sympathetic was inhibited by ergotoxine, or when the emotional hyperactivity of the pseudoeffective state was produced in a totally sympathectomized cat. Freedman and Kabat (1940) could not confirm all the results and conclusions of Freeman; they did not, however, rule out the possibility that sympathetic hyperactivity may play a part in shock following haemorrhage and fluid loss.

The experiments of Slome and O'Shaughnessy (1938), as well as those of Lorber *et al.*, emphasize the importance of the nervous factor in the development of traumatic shock. Either they severed the limb from its nervous connexions (nerve section, spinal section; spinal-cord destruction, spinal anaesthesia) or they severed the limb from everything except its peripheral nerves. The significance of the nervous "factor" became evident from both these series of experiments, but neither has taken the fact into consideration that the three spinal nerves of the lower limb—the femoral, sciatic, and obturator nerves—carry the bulk of its sympathetic nerve supply. It is therefore not clear from these experiments how much of the recorded effect was due to sympathetic action or inaction.

Lena Stern (1942) recently reported good results by stimulating the sympathetic centres and lowering the parasympathetic tone. She injected potassium phosphate direct into the cerebrospinal canal in all those cases in which a fall of blood pressure, a lowering of cardiac activity and respiration, etc., were manifest. Stern saw all these signs disappear, in men and in experimental animals, by her method. Her treatment is based on the view that "the sympathetic and parasympathetic nervous centres play the most important part in the production of shock." She warns against the use of potassium phosphate "in cases in which there is increased excitability of the sympathetic nervous centres, such as sometimes occurs in the first stage of shock"; potassium phosphate should be given only in the second stage.

It is not possible fully to co-ordinate the experimental and clinical results of Stern with my own; the following suggestions seem, however, to be justified. The first period of sympathetic stimulation of Stern is very probably identical with my first period of increased permeability, and her second period of sympathetic depression identical with my second period of decreased permeability. Should this assumption be correct, our two ways of therapeutic approach would prove to be complementary: the sympathetic block would be indicated during the first period, so as to prevent plasmorrhoea through the capillaries and thus the development of the second phase. Should the second phase develop in spite of the sympathetic block, or should the patient come under treatment at this late stage, an intrathecal injection of potassium phosphate may be indicated, provided that further observations should confirm Stern's report.

Another explanation for the apparent discrepancy between Stern's results and mine might be the interesting fact described in earlier papers of Stern, that there is an "antagonism between the central and peripheral parts of the central nervous system as regards their reaction to the same substance." It is therefore possible that a central, intraventricular stimulation of the

sympathetic is equivalent in its physiological effect to a peripheral inhibition. Further experiments will be required to solve this question.

Conclusion

This short review of the literature and my own experiments should suffice to indicate the significance of the sympathetic nervous system in shock. It is the purpose of this article to lay down principles which, if put into practice, may prove to be of value. It is not advocated that other methods which have proved, so far, to be indispensable (blood transfusion, etc.) should be neglected. The combination of both should be tried in suitable cases. They are certainly harmless, and may be of great advantage to the patient. Only clinical experience will enable us to accept or reject these recommendations.

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Medical Memoranda

Suppurative Pylephlebitis

This case is reported for its clinical rather than its pathological interest.

CASE HISTORY

A woman aged 60 was admitted after the gradual onset of vague lumbar pain and nausea. She had no urinary or bowel symptoms, no shoulder pain or vomiting. She was obese and had always had a diffuse brown pigmentation, especially on the arms and legs. Her temperature was 99° and pulse 70. Her abdomen was soft, nowhere tender, and her liver was only just palpable. Within a few hours she lost both pain and nausea, but the next morning had a rigor. From then on she had a total of 31 rigors at intervals of 1 to 12 days. The temperature with a rigor varied from 100.4° to 105°; between rigors it swung between 97° and 101°. There was no rhythm or regularity either in the frequency of the rigors or in the daily temperature variation.

Apart from the rigors she had no other symptoms or signs. In fact, at the seventh week, after her longest interval free from rigors, she seemed so well that she was sent home; but she soon had another rigor and came back. After ten weeks she had a slight pain low in the left posterior axillary line, and we heard a pleural rub. This was replaced by a patch of dullness to percussion; but paracentesis yielded nothing and we thought that the dullness gradually disappeared. There followed another long period in which she felt perfectly well and ate heartily; this was the perplexing part of the picture. Then one evening in her eighteenth week she had a sudden attack of violent coughing, produced some foul green-brown muco-pus, became rapidly very ill, and died.

Investigations.—Blood:—1st week: Hb, 83%; leucocytes, 17,000—polys 83%, lymphs 13%, monos 3%; plasma chloride, 608 mg. per 100 c.cm.; culture sterile; agglutination negative for typhoid, Salmonella, dysentery groups, and abortus. 2nd week: Culture—no pathogen. 4th week: Hb, 85%; leucocytes, 12,000—polys 74%, lymphs 22%, monos 3%; culture sterile; agglutination negative for typhoid group and abortus; staphylococcal antileucocidin index, 0.16 K (upper limit of normal). 6th week: Leucocytes, 18,000. 9th week: Leucocytes, 54,000—polys 93%, lymphs 4%, monos 3%; culture sterile; W.R. negative; Kahn negative; urea, 70 mg./100 c.cm. Urine:—1st week: N.A.D.; diastatic index 5. 2nd week: N.A.D. Faeces:—1st week: Culture—no pathogen. 9th week: Culture—no pathogen. Sputum:—3rd week: Atypical haemolytic streptococci. 18th week: Offensive green-brown fluid; mixed bacteria, mostly *Staph. aureus*. X-ray:—Gall-bladder: No opaque calculi. Graham's test suggestive of pathological change. Chest: 2nd week—N.A.D.; 6th week—N.A.D.; 11th week—left basal pleural effusion; left diaphragmatic outline not seen; right diaphragm normal.

Post-mortem Examination.—Pus in lower half of left pleural cavity, loculated by light adhesions, communicating at one place, through lung, with bronchi. Multiple small abscesses in liver; pus in terminal section of portal vein. Microscopy showed suppurative pylephlebitis, with pus in portal veins, in many systems replacing wall and tissue round veins. All other organs, including appendix, healthy, except the gall-bladder, which was fibrosed and contained stones. We could not say how infection reached the left pleura—probably directly through lymphatic channels; but it is curious that the right side was unaffected. No local track was detectable. It seemed unlikely to be blood-borne, for it was the sole distant focus.

COMMENT

A case of irregular pyrexia with frequent rigors and a mild leucocytosis throughout. The pigmentation reminded us of

Addison's disease, but this was quickly dismissed. Typhoid, abortus, and glandular fevers were not seriously considered because of the leucocytic activity; the first two were, in any case, eliminated by agglutination. A kidney abscess was eliminated, so far as was possible, by investigations. Eventually there remained the possibility of a chronic septicæmia. Three blood cultures were sterile; this, however, did not rule out a portal pyæmia. Mr. Henry Thompson was consulted, and the possibility of this and of a Charcot's biliary fever was discussed. Then, when pus burst into the left bronchi, we thought we might have missed a latent pulmonary or sub-phrenic abscess. Yet we were not greatly surprised at necropsy to find pylephlebitis: this insidious disease, whose nature remains cryptic for so long, could at the best have only been guessed at, and was.

Unusual features of this case were the large number of rigors; the fact that, throughout, these were the sole clinical manifestation (the only localizing signs were those of the pleural complication); the uncommon primary focus; and the unique termination.

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Broad Ligament Cyst in a Strangulated Hernia

Past records have shown that an inguinal hernia may contain a variety of different organs, both normal and pathological, but I think that this case is unusual enough to merit publication.

CASE RECORD

The patient, a widow aged 43, was first seen on May 5, 1944. She said that she had had a hernia for twelve months, but this had always been reducible and symptomless. At 5 p.m. on that day, while chopping wood, she had a sudden severe abdominal pain and repeated vomiting, and the hernia became irreducible. On admission her temperature was 101.2° and pulse 112, and she had a right irreducible inguinal hernia, but a cough impulse was present. A cystic abdominal tumour was noticed, arising from the pelvis and extending about two inches above the umbilicus. There were no signs of pregnancy, and her periods were normal. The passage of a catheter did not reduce the swelling. The patient stated that this swelling appeared to fluctuate in size from time to time.

At 10 p.m. operation was performed, and a sac of an inguinal hernia was dissected out. On opening the sac a fibrous mass the size of a pigeon's egg was delivered, attached to a cystic swelling which was not bowel or bladder. This in turn was opened, and a large quantity of clear yellow fluid escaped: five pints of fluid were aspirated, and the abdominal swelling dramatically diminished. The cyst was closed by a purse-string suture, and the fibrous mass, presumed to be necrotic ovary, was sent for section. (On microscopy, this later proved to consist of lymphoid tissue, with numerous foreign-body giant cells and granulation tissue, but no evidence of ovarian tissue.) The patient's condition did not justify laparotomy at this stage, so the hernia was repaired in the usual way.

On May 8 the temperature rose to 103° and thereafter was swinging; there was also some inflammation of the wound. Sulphathiazole (23 g.) was given. On May 14 she developed diarrhoea, which was presumed to be due to infection of the cyst.

On May 16 laparotomy was performed through a right paramedian incision. Intestines were packed off, and a large cyst was partly delivered into the wound, a purse-string suture was applied, and a French's needle was inserted. Four pints of thick pus were aspirated. The hole in the cyst was then closed. The cyst was then dissected out with difficulty owing to dense adhesions, and was found to be retroperitoneal, and to extend from the loin to the pelvis. The upper pole was in relation to the kidney, but not adherent to it. The lower pole was within the right broad ligament. The uterus and both Fallopian tubes and ovaries were normal. The cyst was removed and a drainage-tube was inserted through a stab wound in the loin, and another through the lower end of the incision draining the pouch of Douglas. During the operation one pint of blood was given intravenously, and, on account of the possibility of general peritonitis and paralytic ileus, intravenous glucose-saline was given post-operatively for 48 hours, and fluids by mouth restricted to sips. Sulphathiazole (27 g.) was given prophylactically. The tube in the loin was removed after 48 hours, and that draining the pouch of Douglas on the fourth day. Slight pyrexia persisted for 18 days, but the patient made an uninterrupted recovery.

COMMENT

It was concluded that this was a cyst of the broad ligament, a lobe of which had become strangulated in the inguinal hernia. Section of the cyst wall showed it to be composed of acute inflammatory tissue, lymphoid and fibrous tissue; the epithelial lining had been destroyed by the acute inflammatory changes.

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