OCCLUSION OF THE INFERIOR VENA CAVA, AS A RESULT OF INTERNAL TRAUMA (DISSECTING VARIX ?)*

THE CASE OF THE LATE DR. W. RIVERS POLLOCK.

BY

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THE parts which form the subject of this communication were bequeathed to the Royal College of Surgeons, for dissection, by the late Dr. W. Rivers Pollock, at the time of his death (1909) Obstetric Physician to the Westminster Hospital; and this he did, by reason of the singular, if not unique, clinical history connected with his disease. I may first give the latter, then describe the specimen, and finally make a short comment on the case.

History.

In 1884, Pollock, when about 24 years of age, and in a condition of perfect health, won the 120 yards hurdle race in the Cambridge University sports—16½ seconds.

In the same year he ran successfully for Cambridge against Oxford, performing the same feat in 16 seconds. He held his breath during the whole time.

This record has since been equalled; and was broken in 1907 by K. Powell, of King's College, Cambridge.

Immediately after the race was over he lay on the grass, and within a few seconds complained of pain in the lumbar spinal region. He was put to bed, where he remained for six months.

Oedema of the legs, and to a lesser degree of the abdomen and scrotum, supervened at once, and persisted for the period mentioned. Whilst in bed, the superficial veins began within a few days to dilate, and this dilatation slowly progressed.

During the rest of life the distended veins were supported by the systematic use of carefully adjusted elastic pants reaching as high as the thorax.

Albuminuria appeared directly after the event, and persisted throughout life. Death occurred on October 5th, 1909—twenty-five years later.

Mr. Arthur Evans, who attended the patient during the last six years of his life, informs me that he was subject to attacks of phlebitis and thrombosis in the enlarged saphenous veins, these attacks being readily brought on—for example, by the pressure of a crease in the elastic pants.

On September 25th, 1909, the patient noticed some tenderness and discoloration behind the right internal malleolus; this extended to the dorsum of the foot; on the 29th the temperature was found to be 100° F., and he had a slight rigor. The next day his throat became sore, and on October 1st the signs of well-marked tonsillitis appeared, the lesion on the foot having vanished. The throat became more painful, and the temperature kept up. On October 4th, the right tonsil was freely incised under gas; not more than three or four drops of pus escaped. Death took place on the day following, and was attributed to tonsillitis and septicaemia.

Dr. J. N. Bernstein, who made the autopsy, tells me that, although carried out within six hours after death, there was such a degree of generalized emphysema as to render a satisfactory examination of the viscera impossible.

Description of the Specimen.

The preparation comprises the superior and inferior venae cavae, wanting their cardiac terminations. The right azygos vein is considerably dilated.

Except for its highest part, the whole of the portion of the inferior cava preserved is converted into a flat impervious ribbon, which is most contracted and thinnest for a distance of 6.5 cm. (2½ in.) opposite and below the renal veins. A portion has been cut away from the front of the vessel below the veins last named, showing that its lumen is completely occluded. The common iliac veins and the portions of the external and internal preserved are

likewise flattened and obliterated, though somewhat less reduced in size, owing to the presence of internal adherent coagulum. The tributaries and trunk of the left renal vein are pervious, though, as tested with the probe, the entrance of the latter into the cava is closed. The same is true of the trunk of the right renal. The right spermatic vein, as far as its entrance to the cava, is likewise pervious. From the left side of the lower part of the cava there projects the occluded end of one of the lumbar veins of the same side. The upper divided end of the inferior cava is pervious, though reduced in size.

It was found during the dissection that the hepatic veins were unoccluded.

REMARKS.

Occlusion of the inferior vena cava as a result of infective thrombosis is a well-known condition, and not a few examples of it have been met with, the thrombus extending into the main trunk from some of its tributaries, either from the femoral and iliac veins below—for example, in typhoid infections—or from the hepatic veins above. Occlusions arising from the direct invasion of the wall by new growth, or by the extension of malignant tumours, of the kidney along the renal vein, are likewise well recognized, though rare occurrences in pathological anatomy. Any suggestion of such an infective origin in the case under notice, however, is excluded by the history. It is incredible that an infective disease should have been present in an athlete in such perfect condition as to have accomplished the feats recorded. Contrast it, for example, with the following, which lately came under the care of Dr. C. R. Box at St. Thomas's Hospital.

A man, aged 21, ten weeks before his admission to the hospital, felt out of health, and had a rigor, which he attributed to a bicycle ride. During the next five weeks the rigors recurred on two or three occasions; finally he fainted whilst at work. Swelling of the abdomen followed, and 5 pints of serum were removed. When admitted, on September 6th, 1912, he was suffering from ascites and enlargement of the liver; there was no evidence of nephritis. Paracentesis on September 10th was rapidly followed by reaccumulation; the abdomen was opened, but no cirrhosis or tuberculous disease of the peritoneum was found. There was no appreciable oedema of the legs.

Death occurred, with dyspnoea and cyanosis, on September 23rd. He had no rigors whilst in the hospital, but there was slight fever of remittent type; the urine contained a trace of albumen. *Post mortem*, there was an adherent thrombus, with softened centre in the inferior cava behind the liver; the cava below was filled with more recent non-adherent clot, which did not entirely occlude it. The clot did not reach into the iliac veins, but superiorly it extended for a short way into the right auricle. Organizing thrombi were found on microscopic examination in the hepatic veins throughout the liver. The portal vein and its tributaries were normal.

The liver, spleen, and kidneys were congested; the lower lobes of both lungs were the seats of pale infarcts. In Dr. Pollock's case the occlusion of the cava arose under such exceptional circumstances as to indicate some exceptional factor in its etiology. And the hypothesis which I venture to put forward is that in consequence of the extreme distension of the inferior cava, occasioned by the holding of the breath throughout the race, a localized rupture of the intima, or of this and the media, took place, which was followed by the forcible extravasation of blood into the walls of the vein—whilst, that is, the exertion was still in progress; that the lesion, in short, in its initial stage, was the counterpart of a dissecting aneurysm of the aorta. Since by classical usage the word "aneurysm" is confined to arterial dilatation (Galen), whilst swelling of the veins has from the time of Celsus been known as varix, the term "dissecting varix" would be that best suited to meet the condition supposed.

The initial lesion would be like that of a dissecting aneurysm, but the two would soon lose their resemblance. In the case of the aorta, the internal pressure is maintained, and the dissection may proceed to great distances, the blood in most cases finding its way again into the arterial system through a consecutive and

* Read before the Pathological Section of the Royal Society of Medicine.

more distant rupture of the intima; and the intramural channel, if death does not ensue, becoming lined with organized coagulum, and converted into an adventitious collateral.

In the case of the vein, on the relief of the abnormal internal pressure, further extravasation into the wall would cease—the normal intracaval pressure would be too low to prolong it. But, as a result of the intramural extravasation, the calibre of the vein would be so reduced, if not actually closed, that secondary thrombosis would take place, which would eventually occlude the channel. All the nucleated cells of the body are capable of furnishing thrombokinase. And after the partial rupture supposed, the ferment would be furnished by the injured tissues as well as by the leucocytes. From the intramural clot the coagulum would extend, at the site of the rent, into the diminished lumen of the vein, and so bring about its complete occlusion.

The intramural coagulum would subsequently undergo a transformation similar to that occupying the lumen, and in process of time become indistinguishable from the proper coats of the vessel.

The transverse rent of the intima and media that is so readily produced in the aorta or other artery by the application of a tight ligature, or by compressing the vessel transversely in a Spencer Wells's forceps, is not producible by the same violence in the cava or other vein. This difference is to be explained mainly by the direction of the muscle fibre. In the case of the artery the ligature sinks without much resistance into the media by displacing its circular fibres, the externa remaining intact; the intima is ruptured last. This can be shown by tying an aorta upon a test tube.

If the force used is not extreme, it will be found on slitting up the artery and examining the cut edge with a hand lens, after making gentle longitudinal traction, that the externa and intima are intact, whilst the media is cleanly ruptured. In the larger veins, like the cavae and iliaes, the longitudinal disposition of the chief part of the musculature, whether on the inner or outer side of the circular fibres, together with the admixture of white fibrous tissue with the muscle, prevents the ligature from penetrating in the same way. The inner coats of a vein can be ruptured, however, by internal tension.

If a pair of lithotomy forceps is introduced into the common iliac vein, or the cava, and slowly opened out until a rending sensation is felt, it will be found on slitting up the vessel that the inner and middle coats present one or more longitudinal ruptures, the external remaining intact.

This is the lesion that I presume occurred in Pollock's case.

This view will commend itself the more if one endeavours to picture what the condition of the inferior cava would be in the particular circumstances under which the accident occurred.

In the first place it is an old observation (known as Müller's) that if a prolonged inspiratory effort is made with the mouth and nostrils closed, the negative pressure, or *vis a fronte*, so induced within the thorax, results in such a filling of the intrathoracic veins, systemic and pulmonary, and of the two auricles (the lungs being unable to expand and aid in filling the potential space) that the auricles become overdistended, and rendered incapable of efficient contraction; and this to so high a degree that the pulse becomes imperceptible.

The embarrassment of the auricles is due not to excessive internal pressure, but rather to the fact that their musculature is too weak to enable it to cope with the negative pressure acting without; for the ventricles may continue to beat, though, as they are receiving little or no blood, none reaches the systemic arterial system.

The connexion of Müller's name with this observation is, as Dr. Augustus Waller has shown, one of the incongruities encountered from time to time in the history of science. The observation was originally made by Frey, and in recounting Frey's experiment Müller appends a brief footnote, in which he suggests that the cessation of the pulse is due to kinking of the subclavian arteries. The name of Müller has, therefore, in some inexplicable way, become transferred from this, his suggestion, to the actual experiment.

Secondly, in the converse observation (Valsalva's), a prolonged, forcible, expiratory effort, with a closed airway, will likewise stop the pulse by reason of the general compression of the thoracic viscera.

Professor A. Keith¹ has pointed out that the venae cavae, with the innominate, iliac, hepatic, and renal veins, being closed off below by the valves in the common femorals, and above by those in the subclavian and jugulars, constitute a cistern or reservoir, which may be filled from its tributaries, but which can only empty itself through the right auricle.

In Pollock's case, so far as the *vis a tergo* is concerned, it is clear that the influx into the cavae, or the venous cistern, would reach its highest mark, seeing that the active muscular contractions of the limbs and trunk would drive the blood through the peripheral tributaries into the reservoir without its being able to return. Some reflux through the hepatic veins by way of the capillaries of the liver into the main, and valveless trunks of the portal system might occur, though this would be limited by the comparatively short space of time the effort lasted, and by the general compression of the abdominal viscera maintained by the contracted state of the parietes. But the state of things within the thorax is not so easily determinable.

The chest, it is true, was in the expanded position, but dynamically the condition was one of forced expiration, prevented by the closure of the airway; in addition to this there would be the less important positive pressure exerted by the elasticity of the expanded lung itself.

Under these circumstances, although little diminution in the volume of the lungs (only that due to compression of their contained air) could take place, the intrathoracic vessels and the heart would be amenable to the effects of pressure, since the exit of blood forwards by the aorta would be free, and the outlet backwards from the right auricle into the cavae would still remain open. The tricuspid valve, which is never fully competent in the normal condition (as tested after death), would allow, again, of some reflux into the right auricle, and so into the venous cistern.

The point not so easy to determine, then, is whether the expiratory effort with a full chest and closed airway would compress the auricles and intrathoracic veins so as to empty them, as in Valsalva's experiment; or whether these would remain overdistended and embarrassed, as in Müller's. The first view is probably the correct one.

In either case an intrathoracic stasis would result; and as the forcible expulsion of blood from the veins of the limbs and trunk into the cavae proceeded, the pressure within the cistern would continue to rise until it led to rending of the internal coat at some spot in its most capacious segment, namely, the inferior cava; and below the entrance of the hepatic veins.

The cava, in fact, behind the liver is supported by the sides of the deep sulcus, or even canal, in which it lies; the second arrangement is well shown in Braune's fifteenth plate.

This would tend to make the stretch below, the seat of highest distension; and this is where the obliteration has taken place. That the trauma arose below the entrance of the hepatic veins is shown not only by the patency of the latter, found after death, but by the total absence of ascites from the clinical symptoms.

The safety of the auricles under overdistension in general may be, as Professor A. Keith² has suggested, due to the support the pericardium would furnish when its capacity of reserve was overdrawn.

The possibility of such an injury having arisen not from distension, but from mechanical strain acting *ab extra*, may be dismissed. The cava is nowhere tightly fixed to the spine, it is not included between the crura of the diaphragm; and as the diaphragm would be in some position of descent, no longitudinal stress arising from causes external to the vein can be assumed to have taken a part in producing the result.

Lastly, there arises a question whether in Pollock's case there was any predisposition to the occurrence of thrombosis—that is, an exalted coagulability which might occasion thrombosis under conditions that would not otherwise have availed.

This question is raised by the fact that one of the patient's brothers had previously died abroad with oedema of the lower part of the body, which had been attributed to the pressure of a supposed sarcoma upon the inferior vena cava, and that another brother had died from embolism of the pulmonary artery following thrombosis of the femoral vein.

That there may be a familial condition which is the converse of the haemophilic, one of exalted coagulability, is quite credible, but I cannot find that such has as yet been observed in healthy individuals—that is, a constant abnormal rapidity of coagulation in the shed blood as distinguished from the variations which occur under well-recognized conditions in the same individual.

REFERENCES.

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VACCINE-THERAPY IN CHRONIC BRONCHITIS.

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It seems probable that a large proportion of cases of bronchitis are caused by an invasion of the bronchial mucosa by pathogenic organisms.

Ritchie¹ made a number of *post-mortem* examinations of children who died of acute bronchitis, and showed that the bronchitis was caused by micro-organisms, chiefly the *Diplococcus pneumoniae*, streptococcus, *B. influenzae*, and *M. catarrhalis*. Berkart² believes that some forms of bronchial asthma are due to a specific inflammatory condition of the bronchial mucous membrane caused by microbic influences.

If the micro-organisms are responsible for causing attacks of bronchitis, we should expect that relief and cure would follow the injection of a suitable vaccine in suitable doses. Clinical experience has already borne this out. Latham³ recorded 5 cases which he successfully treated by vaccines. H. R. Smith⁴ recorded 3 cases treated by vaccines, with favourable results, the duration of these cases before treatment being sixteen years, eight years, and six years respectively. Shivdas⁵ has recorded 39 cases, of which 21 were completely satisfactory.

I have treated bronchitis—both acute and chronic—by this method, but I propose to record here only the first 8 chronic cases in the order in which I attempted to benefit them. I used only autogenous vaccines with one exception. Stock vaccines may be sometimes effective, but a stock vaccine of similar morphology to the causal organism does not necessarily correspond to it in every respect. So that it seems safer to use only autogenous vaccines.

The sputum was collected as far as possible aseptically—boiled water was used for brushing the teeth and rinsing the mouth; a little was also gargled and swallowed. The next phlegm was expectorated into a bottle that had been boiled. Smears of the sputum were made and stained for tubercle bacilli and other micro-organisms. A little of the sputum was then washed and shaken up with sterile saline and one or two loopfuls sown on one or more of the following media: Agar, Pfeiffer's agar, and nasagar. From the growth of fifteen to thirty six hours the vaccine was made up. The dilution with sterile saline was made with a view to avoid the necessity of subculturing, as it has been said that subculturing reduces the efficiency of the vaccine. By this means it is impossible to obtain pure cultures, but it is easy to judge what is the important micro-organism, and to regulate the doses accordingly. The frequent accompanying forms of *M. catarrhalis* can do no harm, and possibly are often requisite to make the treatment successful.

There can be no doubt that a number of Gram-negative organisms are classed together under the term *M. catarrhalis*, and it is difficult at present to know which to exclude and which to include in the vaccine.

CASE I.

Female, aged 75, had suffered with a cough for forty years, with occasional attacks of acute bronchitis. When she began this treatment in November, 1911, she had just recovered from an acute attack. The sputum was nummular; there were no tubercle bacilli, but smears showed that it was full of pneumo-

coccus. By the dilution method I obtained a practically pure growth of pneumococcus. The vaccine was given about once a week in increasing doses, starting at 25 million. After three months' treatment she had lost her cough, had improved in general health, and gained 3 lb. in weight. Treatment was continued for five months altogether. There has been no recurrence of the cough up to the present.

CASE II.

Female, aged 65, had had a bad attack of influenza seventeen years earlier. Ever since that attack she had had a chronic cough, bringing up about 2 oz. of phlegm daily. The vaccine of streptococcus and *Micrococcus catarrhalis* was given in doses of 1 to 20 million, beginning treatment on February 12th, 1912. She was at that time unable to lie down flat at night, but after four injections she was able to do so. The cough and sputum ceased at the end of five weeks, and her appetite and general condition improved. However, in July she began again to bring up a little sputum, but after three injections of a vaccine similar to the previous one the cough completely disappeared, and she has remained free from it since.

CASE III.

Female, aged 35, had had bronchial "asthma" for fifteen months. In March, 1911, she was an in-patient at the Radcliffe Infirmary for two weeks, where she was treated for "bronchial asthma." She sometimes improved a little, but nothing seemed to give her permanent relief. She was able to get about, but was very short of breath and frequently had dyspnoeal attacks, particularly at night. She used to wake regularly at 2 a.m. and had very little sleep. She was cook in a family, and her mistress valued her and was anxious to keep her on if she could be cured. So I suggested we might try a vaccine. I gave the autogenous vaccine of streptococcus at intervals of about a week in increasing doses, beginning with an injection of 15 million on February 27th, 1912. She was soon able to lie down at night and to sleep through until the morning. Treatment was continued for four months. The amount of sputum at the beginning was 8 oz. daily; at the end of treatment it was scarcely half a drachm. She was better in every way; she could go upstairs without shortness of breath. She gained 9 lb. in weight. The cough and dyspnoeal attacks have entirely ceased and there has been no relapse.

CASE IV.

Female, aged 65. Had suffered for eighteen months from bronchitis and dyspnoea. When I saw her first the cough was almost incessant, the breathing was laboured, and she could not lie down in bed. Her nights were very much disturbed. The daily amount of sputum was 1½ oz. The autogenous vaccine of pneumococcus and *Micrococcus catarrhalis* was given in increasing doses at intervals of about a week, beginning on March 26th, 1912, with an injection of 25 million. She soon began to improve, and within four weeks she was able to get downstairs, and was sleeping through the night instead of waking with the cough between 1 and 3 a.m. The expectoration was easier, and the daily amount of phlegm steadily diminished till at the end of nine weeks there was none at all. Treatment lasted three months. Sixteen injections were given altogether.

In this case there was recurrence in November, and she is at present having treatment with a fresh vaccine.

CASE V.

Female, aged 32; five months before I saw her she had an "influenza" attack, with a "cold on the chest," and loss of voice. From that time she had been suffering from repeated colds and coughs. I examined smears of the sputum, and found the predominating organism was *Micrococcus catarrhalis*. In May, 1912, I gave her two injections of a stock vaccine with an interval of five days. The cough was considerably improved, but as she could not stay longer in Oxford the treatment was not continued. I heard recently that her cough is still troublesome.

CASE VI.

Female, aged 42, had been subject for two winters to recurring attacks of bronchitis. She was generally "wheezy" on waking in the morning, and always got very short of breath on exertion. Treatment was commenced on May 9th, 1912, with the vaccine of pneumococcus and *Micrococcus catarrhalis*. She improved rapidly, and after three weeks' treatment she walked about five miles one day; she had not walked so far for several years.

At the end of two months she had completely lost her morning wheeziness and cough.

There was some recurrence in November, but it cleared up again after a few injections of the same vaccine.

CASE VII.

Female, aged 45, had a "bronchial cold" for three months, which did not clear up with ordinary drug treatment.

The autogenous pneumococcus vaccine was given first on June 19th, 1912—dose 42 million. After this dose she was conscious of more stuffiness on her chest, and there was also more cough, though the phlegm was looser.

The second injection of 84 million was given nine days later; there followed the usual negative phase with more cough and phlegm, but this was soon followed by improvement. She felt much more energetic, and there was hardly any cough.

Ten days later the third injection of 84 million was given. After this the cough completely cleared up.