femoral vein just as it appeared on the medial side of the femoral artery. Using this surface marking, the surgeon intending to resect the terminal part of the long saphenous vein would not come upon that structure in this case. Instead, on tracing the superficial circumflex iliac and superficial epigastric veins to their termination, he would pass imperceptibly beneath the deep fascia, cribriform in this region, into the plane of the femoral vessels. If the vessel which then presented itself was the femoral vein, this might be ligated in error. On the other hand, if the dissection were carried low enough to encounter the long saphenous vein, the close relationship of this vessel to the femoral artery might result in the inclusion of both artery and vein in the ligature. That the femoral artery has been injured in the course of Trendelenburg's operation, occasionally with subsequent loss of the limb or death, is beyond doubt.

Luke and Miller (1948) cited three certain cases and one probable case of ligation of the femoral artery. McPheeters (1945) described two cases of ligation of the femoral artery, and in one of these the femoral vein was also tied. At least three cases have come to my notice in this country in which either injury or ligation of the femoral artery occurred during this operation. For obvious reasons it is difficult to get details of the operative findings, but it is possible that the anomaly described above may have been present in some of these cases.

A somewhat similar course of the long saphenous vein was described and illustrated by von Lanz and Wachsmuth (1938). It was also depicted in Fig. 5 of an article by Anson and McVay (1938), so that it cannot be exceptionally rare. No mention was made of its significance in either of these texts.

Whatever the explanation of the surgical errors associated with Trendelenburg's operation it is obvious that the surgeon should be aware of the deceptive variation described in this report. The technical difficulties which it presents are slight. The real danger lies in not knowing that such a condition may exist.

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REFERENCES

- Anson, B. J., and McVay, C. B. (1938). *Anat. Rec.*, **72**, 399.
Von Lanz, T., and Wachsmuth, W. (1938). *Praktische Anatomie*. Springer, Berlin.
Luke, J. C., and Miller, G. G. (1948). *Ann. Surg.*, **127**, 426.
McPheeters, H. O. (1945). *Surg. Gynec. Obstet.*, **81**, 355.

Two Cases of "Novalgin" Agranulocytosis

Numerous reports of the occurrence of agranulocytosis following the administration of "novalgin" and other drugs containing amidopyrine have been published (Kracke, 1931; Plum, 1934; Stenbeck, 1935; Benjamin and Biederman, 1936; Moloney and Vidoli, 1943; Kneidler, 1946). Now that these drugs have been put on the Fourth Schedule of the Poisons List, and the danger of their use is more widely appreciated, cases such as those recorded below should become rare.

CASE 1

A female domestic worker aged 70 was admitted as a suspected case of diphtheria in July, 1950. For three weeks previously she had been taking novalgin tablets (which she obtained direct from the chemist) for rheumatic pains, the exact dose being unknown. Two days before admission she had chilly sensations with malaise, and the next day a sore throat and difficulty in swallowing.

On admission she was found to be well nourished but not fat, and was obviously very ill, with stertorous breathing and a hoarse nasal-sounding voice. Both tonsils, uvula, and soft palate were covered with patchy exudate, greenish yellow in colour, and on the left tonsil it was becoming black, indicating early gangrene; fever was pronounced and an icteric tinge was visible on the conjunctivae. The tonsillar lymph nodes were moderately enlarged. The blood pressure was 135/70. Blood count: Hb, 80%; red cells, 3,800,000;

white cells, 750 (lymphocytes 100%, no granulocytes). From the throat swab *Streptococcus viridans* only was grown. Penicillin, 500,000 units, and pentnucleotide, 10 ml., were given intramuscularly immediately on admission, but the patient died three hours afterwards, before arrangements for a transfusion of fresh compatible blood could be made. Necropsy revealed a superficial pharyngeal necrosis extending from tonsils to epiglottis and haemorrhagic congestion and oedema of the lungs with subpleural and subpericardial haemorrhages.

CASE 2

A female domestic worker aged 68 was admitted to hospital with tonsillitis and pneumonia in May, 1952. In the previous month she saw her doctor on account of painful stiff joints, when novalgin was prescribed in doses of 2 tablets, each containing 5 gr. (0.32 g.), three times a day; over a period of three weeks she had taken a total of 90 tablets, when she became febrile and weak with sore throat, dysphagia, and hoarseness. Novalgin was discontinued at once, and two days later she was given sulphamerazine, a total of 30 g. in five days, without apparent response, and was admitted to hospital.

She was obese, pale, and obviously ill, with hurried noisy breathing; her temperature was 101° F. (38.3° C.), pulse 120, and respirations 30. Exudate covered most of the tonsillar surfaces and spread to the uvula and soft palate with underlying ulceration. The associated lymph nodes were enlarged and tender, and moist sounds were heard at both lung bases without evidence of heart failure. Blood count: Hb, 78%; white cells, 2,000 (neutrophils 3%, lymphocytes 90%, monocytes 7%). Some of the lymphocytes were atypical early forms. A heavy growth of *Str. viridans* was obtained from the throat swabs, but haemolytic forms were not present. Crystalline penicillin, 1,000,000 units daily, was given for seven days, with amelioration of the symptoms and rapid disappearance of the exudate. Immediately thereafter she received 9.5 g. of chloramphenicol over four days on account of persisting bronchitis, to which she was known to be prone. Two days after penicillin was started the white cells numbered 3,100 (neutrophils 10%, lymphocytes 78%, monocytes 12%), and two days later the count was almost normal, subsequent recovery being uneventful.

COMMENT

Despite the similarity of the two patients in respect of age, occupation, and physical state, and probably also of the amount and duration of ingestion of novalgin, the clinical differences in the character of the disease—in the one brief and rapidly fatal, and in the other, over a week and mild with prompt resolution—are impressive, suggesting that idiosyncrasy may be the most important aetiological factor. There was no history of previous ingestion of the drug, although this could not be eliminated completely, especially in Case 1.

The absence of recognized respiratory pathogens, such as haemolytic streptococci, suggests that the faucial angina is a degenerative or allergic rather than an inflammatory condition, and therefore unlikely to respond to penicillin. Alternatively, *Str. viridans* may, under particular conditions, become pathogenic to the respiratory mucosa, as has been long suspected in association with virus invasions. Aureomycin therefore seems to be the antibiotic of choice, as chloramphenicol has been shown capable of causing both agranulocytosis and aplastic anaemia. Sensitivity tests are probably essential in severe cases to find the correct antibiotic.

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REFERENCES

- Benjamin, J. E., and Biederman, J. B. (1936). *J. Amer. med. Ass.*, **107**, 493.
Kneidler, W. H. (1946). *British Medical Journal*, **1**, 876.
Kracke, R. R. (1931). *Amer. J. clin. Path.*, **1**, 385.
Moloney, W., and Vidoli, M. (1943). *Ibid.*, **13**, 317.
Plum, P. (1934). *Ugeskr. Læg.*, **96**, 916.
Stenbeck, P. (1935). *Nord. med. T.*, **9**, 81.