sp.gr. was identical in three individuals aged 2, 5, and 90 years—the youngest and oldest of the cases. Apart, possibly, from an undiscovered very high c.s.f. sugar content, no noteworthy change has been presented in the c.s.f. specific gravity from the pathological conditions which have been specified. From the spinal anaesthetist's point of view, and especially for those who use a timing technique, such a report may be reassuring.

Conclusions

The specific gravity of the cerebrospinal fluid at normal body temperature remains between 1.004 and 1.003, and is nearer 1.003.

The constancy was remarkable in a consecutive series of 314 cases of great variety.

The usual pathological states of the c.s.f. which may be met with can be ignored when spinal nerve-block is contemplated. Much more so is this the case in the healthy subject.

In an experience of over 1,500 spinal anaesthetics by my timing technique there has been no case of loss of control of the ascending hypobaric solution (nupercaine and spino-caine). It has therefore not been any surprise to find that the sp.gr. of the c.s.f. varies in but small degree.

I have to thank Dr. H. A. Fielden for the pathological work at the Torbay Hospital laboratory; and to my anaesthetists, Drs. R. A. Latley and M. Lambie, I am grateful for the collections of cerebrospinal fluid.

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TRAUMATIC ARTERIAL SPASM*

BY

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The interest created in recent years in this subject by a number of writers—notably, Griffiths (1940), Barnes and Trueta (1942), and Cohen (1940-1)—has prompted me to report this case of traumatic arterial spasm, associated with a closed fracture of the tibia and fibula in the middle third resulting in gangrene and amputation.

Case Report

A gunner aged 30 was struck on the antero-lateral aspect of the middle third of the right leg by a gun limber at 9 a.m. on Feb. 25, 1942. He was admitted to hospital approximately six hours after injury with the leg in a Thomas splint, with traction from his boot to the foot of the splint. X-ray plates revealed a fracture of both bones of the leg in the middle third with practically no displacement. He was taken to the operating-room, where inspection of the leg revealed some swelling at the site of fracture. The patient complained of severe pain at and below the site of fracture, and was given morphine gr. 1/4. He had been given morphine gr. 1/4 an hour and a half before admission.

Under pentothal and cyclopropane anaesthesia the Thomas splint and boot were removed. The circulation in the foot and toes appeared normal, judged by colour and warmth, but no attempt was made to feel pulsation in the dorsalis pedis or posterior tibial arteries. There was no wound or sign of injury in the whole limb aside from the swelling at the fracture site. A Steinmann pin was inserted through the os calcis and, with moderate traction of 15 lb. on a Braun splint with a well-padded wooden cross-bar against the junction of the middle and lower thirds of the thigh, a skin-tight plaster was applied from the toes to the tibial tubercle, incorporating the Steinmann pin. This took only about 15 minutes, and then the traction was released and the plaster carried up to the groin with the knee in flexion. The limb was then placed on a Braun splint with 7 lb. traction, the foot of the bed being elevated.

During the night the patient complained of severe pain in the leg, and was seen by the duty medical officer. There was no swelling of the toes and the circulation appeared normal. He required two injections of morphine gr. 1/4 during the night for severe spasmodic pain in the leg at and below the fracture site.

At 9 a.m. on Feb. 26 the cast was bivalved because of his complaints of pain, and the anterior half removed, leaving the posterior half as a back splint. The circulation in the toes appeared normal, but there was no pulsation in the dorsalis pedis artery. He had anaesthesia from the site of the fracture, distally involving the toes, and had no movement in the toes. At this time he claimed that the foot and toes had felt "dead" immediately after injury. No

sign of any pressure or tightness of the cast on the limb was observed.

At midnight on Feb. 26—39 hours after injury—the anterior tibial compartment seemed to be considerably swollen and tense, but the colour of the toes and the circulation in them appeared satisfactory. He was taken to the operating-room and the anterior tibial compartment was opened by a vertical incision. The muscles were somewhat greyish in colour and did not contract on stimulation. The anterior tibial artery was exposed, and was found to be contracted and pulseless. The artery was stripped of surrounding areolar tissue and freed through most of its course right up to the foramen in the interosseous membrane. Pulsation did not return after this procedure. There was no sign of injury to the artery, and no haemorrhage in the anterior compartment had occurred. The wound was left unsutured, with vaselined gauze dressing.

On Feb. 27 the degree of circulation to the toes had notably decreased; the toes were very cold and somewhat cyanotic, and the anaesthesia remained as previously described. He had a temperature of 101° F. and was still complaining of excruciating pain in the foot and leg.

He was taken to the operating-room again and excision of the dead muscles of the anterior tibial compartment was performed. The muscles were very grey and were obviously completely dead, with no response to stimulation. The only ligature applied was to the anterior tibial artery, which had a very slight ooze from it prior to application of the ligature. The wound was left open, with vaseline packing. At the time of operation the foot had a cadaveric appearance. A posterior incision was made at this operation to determine if the same condition had occurred in the posterior part of the leg. The gastrocnemius contracted readily on stimulation, so the wound was closed.

On Feb. 28, at noon, the toes were discoloured and definite gangrene was present in the foot. The temperature was 101° and the pulse 110, and the patient was still complaining of excruciating pain in the leg in spite of repeated doses of morphine. Amputation was done in the thigh, with a 12-in. stump. No tourniquet was used, and only four ligatures were necessary. No pulsation was present in the femoral artery just above the adductor tubercle, and the artery was very contracted. It was divided above this point, where pulsations could be felt. There was no sign of any injury in the tissues of the thigh or to the femoral artery itself.

Immediate dissection of the amputated leg revealed that the femoral, popliteal, posterior tibial, and peroneal arteries were contracted to the size of a fine string—about one-quarter their normal diameter. The vessels were carefully dissected and opened into their smaller branches; no sign of thrombosis or injury was present in the arteries or veins. There was a slight haemorrhagic exudate along the posterior tibial vessels and nerve. The muscles in the posterior compartment had degenerated. The superficial part of the gastrocnemius was only very slightly greyish in colour, whereas the deeper muscles were markedly grey and degenerated.

The stump healed by first intention and the patient made an uneventful recovery.

Comments

It would appear that this patient had persistent and extensive arterial spasm, affecting not only the main vessels but also the collaterals to such a degree that it resulted in massive death of muscle tissue first, followed by gangrene of the limb, necessitating amputation three days after the original injury. There was absolutely no sign of injury to the femoral, popliteal, tibial, or peroneal vessels. The sympathetic reflex vasospasm set up at the time of injury to the antero-lateral aspect of the leg in the middle third was not relieved by stripping of the anterior tibial artery.

No forcible distraction or manipulation was necessary, and Böhler's frame was not used. It is highly improbable that the method of maintaining position during application of the plaster could have aggravated the vasospasm.

On removal of the plaster there was no sign of pressure or tightness of the cast at any point.

Even after amputation, 75 hours from the time of injury, the vessels on dissection were still in a marked state of spasm.

The symptom of excessive pain on admission of a well-splinted fracture should make one suspicious of traumatic arterial spasm.

The aetiology and treatment of arterial spasm have been adequately dealt with in the references quoted, and a detailed discussion would be out of place in this case report.

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