pulseless disease, first described by Takayashu in Japan. Ask-Upmark [1] described 45 cases outside of Japan. This syndrome is characterized by absence of pulses in the upper extremities and in the carotids. This is the opposite of the manifestations that occur in atheromatous occlusion of the aorta at its bifurcation where pulsations cannot be felt in the lower extremities. Severe degrees of Takayashu's disease, as in the case reported, may involve all extremities.

Other conditions where peripheral pulsations may be absent include syphilitic involvement of the aorta; Raynaud's disease (where arteriolar spasm can cause color changes of blanching, redness or cyanosis); Buerger's disease, manifested by local phlebitis and involvement of larger arteries); and pronounced arteriosclerotic disease.

In the anesthetic management of a patient with pulseless disease both clinical judgment and use of special instrument interpretations are important. Clinical impressions of appearance of the patient, peripheral capillary refill time, temperature of the extremities, application of a precordial stethoscope and electrocardiograms, as well as accurate estimation of blood loss were used to great advantage in this case. Of interest in the course of management was the apparent release of a certain amount of vasospasm occasioned by general anesthesia so that an arterial blood pressure of 110 70 resulted after induction of general anesthesia. Vasospasm apparently returned when a certain amount of blood loss occurred suddenly.

Various important monitoring devices can be used in patients with pulseless disease. These include digital plethysmographs which record pulsation amplitudes in a finger or toe.[2] Electroencephalography would be valuable to indicate that circulation—at least of the central nervous system—remained adequate during the period of anesthesia. Other useful measurements would be monitoring of venous oxygen tension, venous pressure, and measurement of urinary output to determine renal circulation. The electrocardiogram is not an indication of effective cardiac output, although ST changes occur with hypoxia secondary to insufficient coronary perfusion. Direct arterial pressure readings by means of a needle or catheter inserted intra-arterially should *not* be used in a patient with severe vascular disease associated with vasospasm.

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## Control of Abnormal Hypertensive Responses by Halothane

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In Hongkong between the years of 1957–1960 a series of 100 cases of Pott's disease in children were surgically treated by the anterior fusion method described by Hodgson and Stock in 1956 [1] and others in 1960.[2]

The anesthetic technique employed in this series consisted of thiopental-nitrous oxide, curare and controlled respiration by means of a Rees' modified T-piece arrangement.[3]

Five cases showed an abnormal hypertensive response to anesthesia and surgery. Four were cases with dorsal involvement, two with paraplegia. These cases were approached and fused through the left chest. One case had a

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lumbar lesion with no obvious cord involvement and was fused via a left twelfth rib incision.

In one case the hypertension was sustained and gave rise to increased bleeding. Further increments of thiopental, curare and increased gas flow with hyperventilation had only a slight effect in lowering the blood pressure. It was decided to introduce 1 per cent halothane vapor by means of a Fluotec into the circuit rather than use a ganglionic blocking agent. This seemed to have the desired effect of lowering the blood pressure to a reasonable level. Halothane in similar concentration was employed in three of the remaining cases. Possible causes of hypertension in these cases: (1) Too light a plane of anesthesia and inadequate curarization. (2) Inadequate ventilation—hypoxia and hypercarbia. (3) Abnormal autonomic response: Possible increased sympathetic tone in some of these children. This may be hypthalamic in origin, which following cortical depression by premedicant drugs and thiopental, may manifest itself as a hypertensive episode, triggered by some stimulus and showing varying degrees of over-activity. This is hypothetical and there is no evidence to show any existing sympathetic over-activity. In paraplegics, altered spinal reflex activity similar to that seen in cases following spinal cord injuries may occur.[4, 5, 6] This syndrome can apparently take the form of hypertension, etc., and can be triggered by a full bladder, cutaneous, visceral or proprioceptive stimuli.

Unfortunately, studies could not be carried out in the conscious children with paraplegia to illicit these responses. Similarly, facilities were not available to estimate catecholamine excretion. This syndrome has been demonstrated during anesthesia and operation in paraplegics,[7] and ganglionic blockade was suggested as a means of counteraction. Indeed, if all five cases described had paraplegia, an easier explanation of this hypertensive response could be offered. In all four cases introduction of 1 per cent halothane vapor seemed to have the desired effect of lowering the blood pressure to a reasonable level with controlled respiration and curare.[8]

The mechanism by which hypotension occurs with halothane is still not clear.[9, 10, 11] Children undergoing other types of operation and anesthetized by the thiopental-nitrous oxide-curare-controlled respiration do not normally show an abnormal hypertensive response if the technique is properly executed,[12] though this may occur in unsuspected pheochromocytoma.[13]

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