Preparation for Anaesthesia - Hypofunction or Aberration in Function of the Sympathetic Nervous System (Dysautonomia)

Disorders of the sympathetic nervous system include Shy-Drager syndrome, Riley-Day syndrome, Lesch-Nyhan syndrome, Gill familia dysautonomia, diabetic dysautonomia, and the dysautonomia of spinal cord transection.

Although individuals can function well without an adrenal medulla, a deficient peripheral sympathetic nervous system occurring late in life poses major problems for many facets of life [153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163] ; nevertheless, a perioperative sympathectomy or its equivalent has been recommended by some. [164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177, 178] A primary function of the sympathetic nervous system appears to be the regulation of blood pressure and of intravascular fluid volume during changing of body position. Common features of all the syndromes of hypofunctioning of the sympathetic nervous system are orthostatic hypotension and decreased beat-to-beat variability in heart rate. These conditions can be caused by deficient intravascular volume, deficient baroreceptor function (as also occurs in carotid artery disease [179]), abnormalities in CNS function (as in Wernicke or Shy-Drager syndrome), deficient neuronal stores of norepinephrine (as in idiopathic orthostatic hypotension [154] and diabetes [19]), or deficient release of norepinephrine (as in traumatic spinal cord injury [153]). Patients with these conditions may have an increased number of available adrenergic receptors (a compensatory response) and an exaggerated response to sympathomimetic drugs. [180] In addition to other abnormalities, such as retention of urine or feces and deficient heat exchange, hypofunctioning of the sympathetic nervous system is often accompanied by renal amyloidosis. Thus, electrolyte and intravascular fluid volume status should be evaluated preoperatively. Because many of these patients have cardiac abnormalities, intravascular fluid volume might be assessed preoperatively using a Swan-Ganz catheter or transesophageal echocardiograph rather than measurement of central venous pressure(Chs. 30 and 31).

Because functioning of the sympathetic nervous system is not predictable in these patients, I usually employ slow, gentle induction of anesthesia and treat sympathetic excess or deficiency by infusing, with careful titration, drugs that directly constrict (phenylephrine) or dilate (nitroprusside) blood vessels or that stimulate (isoproterenol) or depress (esmolol) heart rate. I prefer these drugs to agonists or antagonists, which may indirectly release catecholamines. A 20 percent perioperative mortality rate for 2,600 patients with spinal cord transection has been reported, [161] indicating that such patients are difficult to manage and deserve particularly close attention.

After reviewing 300 patients with spinal cord injuries, Kendrick et al [181] concluded that autonomic hyperreflexia syndrome does not develop if the lesion is below spinal dermatome T7. If the lesion is above that level (the splanchnic outflow), 60 to 70 percent of patients experience extreme vascular instability. The trigger to this instability, or *mass reflex* involving noradrenergic and motor hypertonus, [155] can be a cutaneous, proprioceptive, or visceral stimulus (a full bladder is a common initiator). The sensation enters the spinal cord and causes a spinal reflex, which in normal persons is inhibited from above. Sudden increases in blood pressure are sensed in the pressure receptors of the aorta and carotid sinus. The resulting vagal hyperactivity produces bradycardia, ventricular ectopia, or various degrees of heart block. Reflex vasodilation may occur above the level of the lesion, resulting in flushing of the head and neck.

Depending on the length of time since spinal cord transection, other abnormalities may occur. Acutely (i.e., <3 wk from the time of spinal injury), retention of urine and feces is common and, by elevating the diaphragm, may impair respiration. Disimpaction of the intestine alleviates this respiratory problem. Hyperesthesia is present above the lesion;

reflexes and flaccid paralysis are present below the lesion. The intermediate period (3 d–6 mo) is marked by a hyperkalemic response to depolarizing drugs. [160] The chronic phase is characterized by a return of muscle tone, positive Babinski sign, and frequently, the occurrence of hyperreflexia syndromes (e.g., mass reflex; see previous).

Thus, in addition to meticulous attention to perioperative intravascular volume and electrolyte status, the anesthesiologist should know—by history-taking, physical examination, and laboratory data—the status of the patient's myocardial conduction (as revealed by ECG), the status of renal functioning (by noting the ratio of creatinine to blood urea nitrogen), and the condition of respiratory muscle (by determining the ratio of forced expiratory volume in 1 s to forced vital capacity [i.e., FEV_1 /FVC]) (Ch. 24). The anesthesiologist may also obtain a chest radiograph, if atelectasis or pneumonia is suspected on the basis of history-taking or physical examination. Temperature control, the presence of bone fractures or decubitus ulcers, and normal functioning of urination and defecation systems must be assessed. Confirmation of the latter prevents postoperative pneumonia or atelectasis caused by high positioning of the diaphragm.