Systemic hypotension in neurosurgery

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The authors review the intraoperative use of elective hypotension to reduce the probability of hemorrhage, to increase pliability of the aneurysmal sac for ease of clip application, and to control hemorrhage. The optimum agent and techniques for lowering systemic blood pressure remain controversial, but trimethaphan, sodium nitroprusside, and halothane have been found most useful. When cerebral blood flow falls below the brain’s capacity to autoregulate, distinct time-related alterations occur biochemically and histologically. The profile of prolonged reduced adenosine triphosphate (ATP), low phosphocreatine, low glucose, and elevated lactate and lactate/pyruvate ratio is associated with swelling of perivascular astrocytes and “blebbing” of vascular endothelial cells with subsequent cerebral damage. To prevent permanent alteration it is desirable to observe time constraints and to employ other means of protection such as hypothermia, although the authors believe the latter unnecessary for short hypotensive periods. It has been proposed, but not substantiated, that anesthetics which depress rate of cerebral oxygen consumption but do not affect cerebral ATP level protect the brain from hypotension. Several investigations suggest that halothane, a vasodilator, satisfies the safety requirement. The most prominent contraindication to halothane, however, is elevation of intracranial pressure. At present hypotensive surgery for aneurysmorrhaphy is usually performed when intracranial pressure has returned to normal. Experimentally the electroencephalogram has been observed to show alterations prior to biochemical parameters for following brain vulnerability, so that it conceivably could be an effective monitoring technique during prolonged profound hypotension.

KEY WORDS • hypotension • blood pressure • cerebral ischemia • hypoxia

DELIBERATE induction of systemic hypotension is regarded as an intraoperative surgical adjunct with a limited and controversial application. Interest in hypotension persists since it allows conservation of blood volume and may provide a dry field for the operator. This technique has been used during prostatectomy, correction of coarcted aorta, radical mastectomy, hysterectomy, pelvic surgery, ophthalmic surgery, orthopedic surgery, head and neck surgery, and in neurosurgical procedures involving resection of vascular tumors (meningiomas and hemangioendotheliomas) or in the repair of vascular anomalies (aneurysms and arteriovenous malformations). Brief hypotension during aneurysmorrhaphy allows safer dissection.
with less danger of hemorrhage and greater pliability for application of an occlusive clip. This method is particularly valuable in neurosurgery, since even temporary ligation and vessel occlusion may irreversibly damage brain tissue. This paper reviews the usefulness of this technique in certain neurosurgical procedures.

**Technique**

There have been many approaches for reduction of systemic blood pressure. Cushing and Eisenhardt\(^4\) reported using arteriotomy to achieve a dry field following hemorrhage. This method was further developed by Gardner\(^4\) in 1946, but shock ensued resulting in vasoconstriction and inadequate tissue perfusion. Ganglionic blocking drugs were reported first by Enderby\(^8\) in 1950; they block sympathetic vasoconstriction, produce an increase in the peripheral vascular bed, and thus reduce blood pressure. Tissue perfusion generally persists. Drugs used include pentamethonium, hexamethonium, tetraethylammonium, phentacropinium, and trimethaphan (Arfonad). Such drugs are used extensively today, especially the latter, often in combination with other methods to achieve the desired blood pressure.

The hypotensive action of sodium nitroprusside (Nipride)\(^\dagger\,\ddagger\) was first investigated in 1929 by Johnson\(^5\) who studied the compound in several animal species. Sodium nitroprusside intravenous infusion results in hypotension occurring within seconds because of its direct vasodilating effect. It does not act at ganglionic sites and does not increase cardiac output and heart rate.\(^6\) The nitroprusside ion is converted to thiocyanate and is potentially toxic in long-term administration. Because of its evanescent duration of action, sodium nitroprusside must be given by continuous intravenous infusion.\(^5\) Deep halothane (Fluothane)\(\ddagger\) anesthesia\(^7\) also causes direct vasodilation with concomitant hypotension and is accompanied by decreased cardiac output. In addition, it potentiates effects of ganglionic blockers by suppressing vasomotor responses. Trimethaphan relaxes capacitance vessels and blocks sympathetic motor reflexes. It lowers arterial pressure both by arterial vasodilation, and by therefore decreasing cardiac output.\(^4\) Inactivation of pupillary reflexes with reference to trimethaphan by ganglioplegic drugs may cause confusion in the neurological evaluation of the patient. This has not been a postoperative problem in our experience.\(^8\) Trimethaphan, sodium nitroprusside, and halothane have been found most useful in neurosurgery. During aneurysmmorphy it is desirable to work on a pliable brain. Brain size may be decreased at surgery with osmotic diuretics, by drainage of CSF,\(^9\) and by administration of steroids preoperatively.

In 1948, Griffiths and Gillies\(^9\) introduced high spinal anesthesia for sympathetic blockade, and epidural anesthesia was later similarly employed. These methods became archaic with the advent of ganglionic blockers. Both elevation of the head above the heart and application of negative pressure to the lower extremities have been used to decrease cerebral arterial pressure.\(^10\) Enderby\(^10\) has shown that with 30° to 35° head elevation cerebral systolic arterial pressure is 36 mm Hg when systolic pressure at the heart is 60 mm Hg. Increased airway pressure with resulting increased intrathoracic pressure and reduced venous return has been employed, but in general anesthesiologists prefer low intrathoracic pressure and have not used these techniques.\(^6\)

**Results**

**Morbidity and Mortality**

Reduction of blood pressure in itself obviously entails definite risks; therefore, it is instructive to survey past clinical experience. Statistics cited below are from hypotensive patients without regard for somatic system. Information specific to the nervous system is scant but will be indicated when possible. Eckenhoff, et al.,\(^2\) Shepperd and Grace,\(^3\) Rollason and Hough,\(^4\) and Moersch, et al.,\(^5\) have all reported no morbidity or mortality in excess of that observed for normotensive controls. Extrapolation of their salutary statistics to a neurosurgical population may not be

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\(^*\)Arfonad, a brand of trimethaphan camsylate, is manufactured by Roche Laboratories, Nutley, New Jersey 07110.

\(^\dagger\)Nipride is manufactured by Roche Laboratories, Nutley, New Jersey 07110.

\(^\ddagger\)Fluothane is manufactured by Ayerst Laboratories Inc., New York, New York 10017.
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valid since those data cited above were gathered from small series; furthermore, the surgery did not involve craniotomy, which might alter effects of hypotension on brain. Larger population studies have also revealed low morbidity and mortality attributable to hypotension. Enderby reported a mortality rate of 0.1% in 9107 cases but could attribute neither morbidity nor mortality directly to hypotension. Hampton and Little reported 27,930 cases with a mortality of 0.29% due to hypotension. We do recognize that it is sometimes difficult to attribute a complication to hypotension or to one of the many other variables of the intraoperative period. Larson gathered data from 1958 to 1963 and found in a series of 13,264 patients a 0.62% mortality directly attributable to hypotension. In a personal survey of the reported English experience between 1964 and 1973, he reviewed 16 papers that reflect nearly all data for elective hypotension reported during this period. The low number of papers probably reflects a decline in interest in hypotension in other surgical specialties and failure to report clinical experience. A total mortality of 0.33%, with a mortality due to hypotension of 0.22%, was collated in these papers. The latter figure includes three deaths, two due to cardiac arrest and one to renal failure. Severe morbidity included 17 cases of varied neurological deficits. Comparison with reports discussed earlier reveals little variance from the low mortality experienced in later years. In recent series and most commonly in earlier ones data reflect hypotension near 60 to 80 mm Hg systolic for about 60 minutes. One must be cautious in using this information as a psychological rule for neurosurgery, since patients subjected to hypotension have for the most part been in good condition prior to operation. In addition this information does not apply to patients who require hypotension to control bleeding from systemic disease or from malignant hypertension.

Intraoperative Results in Neurosurgical Patients

There is little published data regarding hypotension in neurosurgical patients. We have used this technique for over 5 years in aneurysm cases with excellent results. Allcock and Drake, Drake, and Sellery, et al., have had good-to-excellent results in 143 of 163 Grade I patients operated under light halothane anesthesia with spontaneous respiration and trimethaphan or nitroprusside hypotension to a mean arterial pressure (MAP) of 40 mm Hg up to 40 minutes without hypothermia. Spontaneous respiration in 140 patients with basilar aneurysm allowed some degree of clinical monitoring of brain-stem perfusion. Unassisted ventilation and normothermia used in all aneurysm cases since 1962 did not affect the incidence of postoperative vascular spasm.

Discussion

Biochemistry and Physiology

It is instructive to review the biochemistry of central nervous system tissue during arrested circulation and cerebral hypoxia; profound hypotension may result in similar alterations, but little experimental information is available for hypotension. During ischemia there is a rapid drop in adenosine triphosphate (ATP), phosphocreatine (PC), glucose, glucose-6-phosphate, and α-ketoglutarate. There is no change in citrate or malate and there is an increase in adenosine monophosphate (AMP), adenosine diphosphate (ADP), lactate, pyruvate, and the lactate/pyruvate ratio. The decrease in PC precedes the fall in ATP and occurs simultaneously with tissue lactate rise. Total ischemia increases the glycolytic rate fourfold to sevenfold by facilitation of those enzyme steps which bring about phosphorylation of glucose and fructose-6-phosphate and the phosphorolysis of glycogen. The order of depletion of the reserve energy compounds was shown to be PC, glucose, ATP, and glycogen. The rate at which these compounds were depleted varied with age and was slowed by pentobarbital anesthesia. Ischemia caused by moderate hypotension produced slight to moderate increases in cerebral tissue lactate; small increases in cerebral 5’AMP and ADP also occurred, usually associated with elevated arterial pCO₂.

In contrast to ischemia, during extreme hypoxia without reduction of blood flow there is preservation of ATP, PC, ADP, AMP, glucose, glucose-6-phosphate, α-ketoglutarate, and citrate. Lactate, pyruvate, and the lactate/pyruvate ratio are elevated. In a study
by Duffy, et al., levels of glucose were little influenced and glycogen was decreased only by approximately one-half. Large changes however occurred in cerebral lactate in extreme hypoxia (4% to 7% oxygen), apparently due to an increase in glucose influx from the blood. After 30 minutes of hypoxia, fructose diphosphate was lower than in controls. This together with elevations of glucose-6-phosphate suggested that hexokinase as well as pyruvate and/or lactate dehydrogenase were facilitated. During severe hypoxia the cerebral metabolic rate is decreased nearly to the extent seen in deep anesthesia, apparently in an effort to conserve energy stores. In the latter situation adequate blood flow prevents depletion of glucose and its subsequent citric acid cycle intermediates.

Another important variable having a profound influence on tissue metabolism during hypotension and ischemia appears to be the tissue pCO2. Even severe (40%) hypercapnia at normotension caused no change in the concentrations of the cerebral adenine nucleotides. As arterial pCO2 rose at normotension, there was a progressive decrease in both pyruvate and lactate concentrations and a shift in the creatine phosphokinase equilibrium which favors a cerebral phosphocreatine breakdown. Elevated arterial pCO2 causes vasodilation and an increased cerebral blood flow and may have a beneficial effect on the brain during hypoxia by slowing the metabolic decline of the tissue. Hypercapnia in combination with hypotension leads to a breakdown of cerebral PC and ATP and elevated cerebral ADP and 5'AMP. It may be that hypotension is roughly equivalent to stagnant hypoxia complicated by hypercapnia.

Of great significance in the care of patients with reduced intraoperative blood pressure is the rate of high-energy ATP production from glucose and oxygen. Normal values for cerebral ATP are in the vicinity of 2.3 μmol/gm. In ischemic brain, ATP reserves rapidly deteriorate due to oxygen deficiency and cessation of aerobic metabolism. One may calculate rate of oxygen consumption by brain tissue, that is, cerebral metabolic rate for oxygen (CMRO2), by the formula

$$\text{CMRO}_2 = \text{CBF} \times (\text{O}_2 \text{ arterial} - \text{O}_2 \text{ venous}),$$

where CBF = cerebral blood flow and $\text{O}_2$ = oxygen concentration in cerebral vessels. Oxygen consumption by the adult human brain averages 3.0 to 3.5 cc/100 gm/min or for the whole brain, 40 to 50 cc/min. If one ignores oxygen concentration the problem is simplified to only requirements for CBF. Cerebral blood flow in normal brain averages 50 cc/100 gm/min with 80 cc/100 gm/min for gray matter and 20 cc/100 gm/min for white matter. Total flow depends directly on mean perfusion pressure, that is, the time-averaged difference between cerebral venous and arterial pressures, and is inversely related to cerebrovascular resistance. It is well documented that cerebral blood flow is capable of autoregulation, that is, maintenance of constant flow over a wide range of perfusion pressures by automatic changes in resistance factors. Since mean cerebral venous pressure is small compared to MAP it should be noted that CBF is constant from MAP 70 mm Hg to approximately 180 mm Hg. Below 60 to 80 mm Hg, CBF responds passively to MAP, since presumably the cerebrovascular system has reached its minimal resistance by maximal dilation. However, Fitch, et al., demonstrated that during hemorrhagic hypotension, autoregulation is maintained until a mean arterial blood pressure of approximately 65 mm Hg is reached, after which the cerebral blood flow is pressure-passive. With drug-induced hypotension, autoregulation persists to lower levels of mean arterial blood pressure (approximately 35 mm Hg) although there are slight variations between the different drugs studied.

A number of factors influence total cerebrovascular resistance, including vessel caliber, blood viscosity, temperature, cerebrospinal fluid (CSF) pressure, and possibly the state of mental activity. To what extent these variables are interdependent and whether a unified mechanism may explain autoregulation is unclear. Classically three theories have been invoked to explain autoregulation. The metabolic theory ascribes CBF alteration to changes in extracellular fluid (ECF) and tissue pO2, pCO2, and pH. Hypoxia below an arterial pO2 of 50 mm Hg at a constant pCO2 causes large increases in CBF. At normal oxygen tensions pCO2 effects predominate over those due to pO2. Cerebral blood flow increases in sigmoid fashion from arterial pCO2 10 to 90 mm Hg.
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in normotensive animals. This effect becomes less pronounced at lower MAP's and disappears near 60 mm Hg. MacMillan and Siesjö have extended this concept and have shown in normotensive, hypoxic (pO₂ = 28 mm Hg) rats that doubling pCO₂ has a beneficial effect in lowering cerebral lactate accumulation. However, when pO₂ was normal but rats were hypotensive below 60 mm Hg, an increase in pCO₂ from 40 to 65 mm Hg was detrimental, causing a lower intracellular pH, an elevation of tissue lactate, and a sharp decrement in energy state of the tissue. This detrimental effect of CO₂ in hypotensive animals was attributed to acidosis. The acidosis caused inhomogeneity of CBF (capillary areas dropping out of the circulation) as demonstrated by a greater reduction in the energy state at a higher cerebral venous pO₂. This information and studies of lactate levels during hypoxia suggest that at least one other mechanism is responsible for CBF change. Severinghaus, et al. have proposed that the tone of each individual arteriole is controlled by its own extracellular fluid pH, and that in addition CO₂ acts as a shuttle between cerebral tissue and cerebral arterioles. Hypothetically, fresh blood carries CO₂ away and provides oxygen for metabolism of lactic acid produced by the brain. Thus the hydrogen ion associated with lactic acid is consumed and the arteriole contracts. Betz and Skinhej have considered this hypothesis and have enumerated data which cannot be correlated.

A neurogenic theory for control of CBF is based largely on the anatomic distribution of nerve fibers from the cervical sympathetic chain and from the parasympathetic components derived from the greater superficial petrosal and facial nerves. Stimulation of sympathetic nerves causes mild vasoconstriction, but section of either sympathetic or parasympathetic nerves does not appreciably affect CBF. Millar recently collected evidence for cervical control of CBF in baboons over a wide range of arterial pO₂ and pCO₂. Dahl has demonstrated in rhesus monkeys that innervation of extracerebral blood vessels is restricted to the adventitial layer. Once these vessels penetrate the pial membrane and become by definition intracerebral, they lose the adventitial layer and are devoid of nerve supply. This indicates that although extracerebral vessels may be responsive to nervous control intracerebral vessels probably are not.

The myogenic theory of autoregulation implies that smooth muscle in vessels dilates independently of other mechanisms with decrement in blood pressure. Symon, et al. have shown that the autoregulatory response is extremely rapid (within 15 seconds) to a pulse of increased blood pressure. They suggest that this rapidity of response makes metabolic regulation unlikely.

Haggendahl, et al., studied effects of increased viscosity on CBF. Cerebral blood flow was constant with a hematocrit from 30% to 60%. Below this range CBF increased, probably due to low pO₂, and above this range CBF did not respond as efficiently to pCO₂. It has been shown in cases of hemorrhagic shock that capillary flow is enhanced by Dextran-40 due to reduction of viscosity. In addition Heiss, et al., demonstrated a significant increase in CBF in stroke patients with infusion of Dextran-40.

Anesthetic Protection

It has been suggested that certain anesthetics provide protection from anoxia. One mechanism suggested is that anesthetics, notably barbiturates, increase cerebral ATP levels. More recently this view has been challenged. Minard and Davis demonstrated no elevation of ATP in the rat on administration of chlorpromazine and phenobarbital anesthesia. Michenfelder, et al. agreed that no alteration of ATP occurs over a wide range of normal anesthetic conditions. However, anesthetics do have a profound effect on CMRO₂, and it has been reasoned, therefore, that anesthetics which depress CMRO₂ without affecting cerebral ATP levels should provide a degree of protection from anoxia. Halothane and barbiturates decrease CMRO₂ while nitrous oxide increases it. Michenfelder and Theye suggest that no protection from ATP decay occurs during anoxia produced by decapitation of dogs anesthetized with halothane, thiopental, or nitrous oxide halothane, although wide variations in CMRO₂ were noted. Hypothermia with its concomitant low CMRO₂ in the same range did prolong the half-life of ATP. Such lack of protection by those anesthetic agents may be linked to the demonstration in vitro that anesthetics alter oxygen re-
quirements only in functioning (stimulated) brain tissue. In Michenfelder and Theye's experiments the electroencephalogram became silent 15 seconds after decapitation in all animals. This was also true in cerebral circulatory arrest as demonstrated by Yashon, et al.

Halothane has been shown to have apparent advantage over several other methods of induction of hypotension. Ransohoff, et al., showed in cats that during trimethaphan hypotension retinal vessels showed marked constriction, while during halothane hypotension the vessels remained the same caliber as normotensive controls. Magness, et al., demonstrated that during trimethaphan-induced hypotension the EEG became isoelectric at 30 mm Hg, but during hemorrhage to the same MAP the EEG was preserved. Kaasik, et al., and Nilsson and Siesjö measured cerebral ATP exhaustion and cerebral lactate accumulation in rats held at 30 mm Hg MAP by bleeding, and by 2% halothane hypotension augmented by increased airway pressure. During halothane hypotension there was little ATP decline from normal, but there was an elevation of lactate from a normal 1.28 mm/kg to 5.34 mm/kg after 30 minutes. With bleeding, ATP declined from 2.72 mm/kg to 1.98 mm/kg, and lactate rose from 1.46 mm/kg to 11.13 mm/kg after 10 minutes. Yashon, et al., measured canine cerebral lactate accumulation for 1 hour at an MAP of 30 mm Hg induced by hemorrhage, trimethaphan infusion, ATP infusion, and deep halothane anesthesia. Lactate elevation during halothane anesthesia was one-third to one-half that of the other methods. It is well documented that halothane is a cerebral vasodilator, but at 30 mm Hg the cerebral vasculature should be maximally dilated. It is possible therefore that halothane protects intracellular metabolism intrinsically. It is also notable that in a recent series of 200 cases of intracranial aneurysm, elective halothane hypotension with 100% oxygen was used. Yashon, et al., noted during halothane hypotension with 100% oxygen that cerebral lactate elevation is as great as with the other hypotensive techniques, suggesting that the halothane protective effect is not present. In Nilsson and Siesjö's series of rats an increase from 30% to 100% O₂ during 2% halothane hypotension with increased airway pressure did not significantly alter cerebral ATP and lactate. These authors also showed that hyperventilation with a pCO₂ of 20 mm Hg during 2% halothane hypotension to 30 mm Hg resulted in no change in cerebral ATP level, but cerebral lactate rose to 9.4 mm/kg. During 7% CO₂ and 2% halothane inhalation cerebral ATP was unchanged and cerebral lactate rose slightly to 2.89 mm/kg. Thus hyperventilation during clinical hypotension should be avoided if lactate accumulation can be assumed to represent cerebral ischemia.

A major disadvantage of halothane anesthesia is a concomitant increase in intracranial pressure. Gordon demonstrated that rise to be marked and accentuated by preexisting elevated intracranial pressure (as with a tumor). It has been postulated that this rise is secondary to increased CBF, and that with a preexisting elevated pressure, normal compensatory mechanisms such as redistribution of CSF, venous blood, and to some extent brain have already been utilized. Since pressure gradients are known to exist in brains with expansile lesions, brain herniation may occur. It has been suggested that this may be the mechanism by which some patients with mass lesions undergo sudden deterioration during general anesthesia.

Shapiro, et al., have shown, however, that various mechanical factors such as intubation and application of head clamps will also cause an acute increase of intracranial pressure in patients with space-occupying lesions. They found that thiopental could rapidly abort both mechanical and halothane-produced elevations of intracranial pressure.

Intracranial pressure becomes acutely elevated following aneurysmal hemorrhage, and gradually recedes providing there is no recurrent hemorrhage. In addition, operative mortality was seen to be significantly reduced when lumbar CSF pressure fell to 200 mm H₂O. Therefore, in humans halothane should be used for hypotension only when intracranial pressure is not increased or when it has returned to nearly normal.

Thus, although the ideal technique for reduction of blood pressure has not been established, halothane seems to offer several advantages, especially when compared to trimethaphan. The threat of increasing intracranial pressure cannot be ignored and it should be emphasized that human cerebro-
vascular dynamics may not completely correspond to animal models described above. Finally, although halothane may be preferred on theoretical grounds when hypotension is required for neurovascular surgery, it may not be the anesthetic of choice for other neurosurgical procedures.

**Pathological Alterations**

Histologically, complete ischemia produces swelling of perivascular astrocytes and intravascular "blebbing" of endothelial cells. These changes account for areas of brain which remain ischemic following restoration of blood flow ("no-reflow phenomenon"). The volume of tissue thus affected increases with duration of vascular obstruction. It has recently been shown that this represents swelling of mitochondria, the cellular organelle containing enzymes responsible for the citric acid cycle and energy production. In patients coming to autopsy following hypotension, findings consistent with localized or diffuse cerebral infarction have been described.

**Hypothermia and Hypotension**

Hypothermia has been used concomitantly with elective hypotension since oxygen consumption of the brain is reduced 6% to 7% per degree fall in temperature from 37°C to 25°C. In addition, CBF falls 6% to 7% per degree centigrade fall in temperature. Since this could be due to autoregulation, Forrester, et al., measured CBF during hypothermia at constant PCO2 levels, and CBF still decreased. It has therefore been proposed that these effects may be mediated by direct vasoconstriction or by an increase in blood viscosity. It is customary to separate hypothermic temperatures into moderate (26°C to 32°C) and profound (below 20°C), and often moderate hypothermia is induced to accompany hypotension for cerebrovascular surgery. Deep hypothermia is employed uncommonly but it has been used successfully when the surgical problem requires circulatory arrest. Arrest at normothermia is tolerated only 3 to 4 minutes and this time is doubled during moderate hypothermia. Kramer, et al., have shown the half-life for ATP exhaustion to be 3.8 minutes at normothermia and 13.3 minutes at cortical temperatures of 13°C. Clinical hypothermia to 15°C can provide cerebral protection for 30 to 40 minutes of arrested circulation. Moderate hypothermia is achieved by surface cooling in combination with peripheral vasodilators such as chlorpromazine and halothane for promotion of heat exchange. Deep hypothermia requires bloodstream cooling with cardiopulmonary bypass drawing either from the right atrium or from the inferior vena cava by way of femoral veins. Complications of deep hypothermia below 28°C include cardiac arrhythmias and abnormal coagulability not reversible by protamine sulfate. We have not used deep hypothermia in our patients but do use moderate lowering of temperature since it entails little additional risk. Drake suggested that hypothermia is not required during aneurysm surgery.

**Monitoring Techniques**

One might predict that hypotension is safe based on published data mentioned earlier. However, a means to monitor cerebral function to avoid morbidity seems desirable. Outstanding parameters that have been used include CSF pO2, CSF lactate, and internal jugular pO2. As has been pointed out earlier, Drake and coworkers used unassisted respiration as an indicator of brain-stem perfusion. It has been documented in the biochemical literature that brain function fails during anoxia as demonstrated by change or disappearance of EEG before significant decrease in ATP or major change in PC. Current hypotheses suggest that brain depression in severe hypoxia represents the exaggerated response of a protective mechanism; that is, when "power failure" is imminent, neuronal activity is curtailed by a specific mechanism to conserve dissipated energy stores. If energy expenditure were to continue unabated, the point of irreversible damage would be reached quickly. Instead, much of the nervous system is put to rest; if coma supervenes, this is favorable because it reduces oxygen expenditure on a priority basis. It becomes evident that at present the most sensitive index to hypoxia is the EEG. We have not found EEG monitoring to be necessary because of the short duration of hypotension we employ.
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