Medical Aspects of the Preoperative Management of Aneurysms: A Review

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SUMMARY: *The aim of medical management of a patient with a recently ruptured intracranial aneurysm is to preserve residual brain function and prevent systemic complications. Surgery should be performed as soon as the patient is in good neurological condition. Most fatalities result from the destructive effects of the initial hemorrhage, but delayed ischemic neurologic deficit can result from vasospasm and rebleeding. Systemic complications of the brain damaged state result in a smaller proportion of deaths. Common medical problems are reviewed as well as their medical management. In particular, some special problems related to subarachnoid hemorrhage such as cerebral edema and herniations, rebleeding, and vasospasm are also considered. Major recent advances have been the introduction of antifibrinolytic therapy, the realization of the importance of maintaining blood volume and pressure, as well as general advances in respiratory care.*

RÉSUMÉ: *Le but du traitement médical d'un patient souffrant d'un anévrysme intracrânien récemment rupturé est de protéger les fonctions cérébrales résiduelles et de prévenir les complications systémiques. La chirurgie devrait être entreprise dès que la condition neurologique du patient est bonne. La plupart des décès résultent des effets destructifs de l'hémorragie initiale, mais certains déficits neurologiques tardifs peuvent provenir de vasospasmes ou de nouveaux saignements. Les complications systémiques de l'état cérébral endommagé s'accompagnent d'une plus faible proportion de décès. Nous étudions les problèmes médicaux communs ainsi que leur traitement, en particulier certains problèmes reliés à l'hémorragie sousarachnoidienne, tels l'oedème cérébral, les hernies, les nouveaux saignements et le vasospasme. Les progrès majeurs récents furent l'introduction de la thérapie antifibrinolytique, la reconnaissance de l'importance à accorder au maintien du volume sanguin et de la tension, ainsi que les progrès généraux dans les soins respiratoires.*

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INTRODUCTION

Recent years have witnessed an increase in our knowledge of the natural history of the recently ruptured intracranial aneursym. There has also been a dramatic improvement in our ability to monitor the critical physiological parameters in the patients who are seriously ill. While the timing of surgical intervention remains controversial, it seems reasonable that it should be carried out as quickly as possible for the patient who is in good clinical condition. The principal threats to the patient appear to be the initial brain damage resulting from the first bleed and its direct sequelae, rebleeding, delayed ischemic neurologic deficit resulting from vasospasm, and medical-surgical complications. Significant progress has been made in reducing the incidence of rebleeding but our ability to ameliorate the results of the initial rupture remains limited. While numerous therapies have been proposed to prevent or treat vasospasm, they all have a somewhat tenuous basis with the possible exception of hypervolemia and hypertension. The surgical complications have been greatly reduced with the use of the microscope and microtechnique and medical complications have been minimized by adopting the techniques of critical care medicine and intensive care unit organization.

Natural History of Ruptured Aneurysm

A knowledge of the natural history of subarachnoid hemorrhage from aneurysm is an essential prerequisite to its intelligent management. There is only one major study of a defined population over a defined time interval without early surgery. This was carried out by Pakarinen in Helsinki, 1954-1961. For 389 cases of ruptured

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aneurysm the total mortality for the first 4 weeks was 51%. Seventy percent of the deaths in the first 4 weeks were due to the effects of the initial hemorrhage while 30% were felt to be due to the effects of a second hemorrhage (or in any event occurred following an initial improvement in clinical status). Fifteen percent of all deaths occurred following an initial improvement in clinical status. Fifteen percent of all deaths occurred prior to admission to hospital. For the group who died from the initial hemorrhage, 93% died in the first week, 5% in the second, 0% in the third, and 1% in the fourth (74% died in the first day!). For the group who died after initial improvement or who had no initial unconsciousness (the recurrent bleeds); 18% died in the first week, 23% in the second week, 20% in the third week, and 18% in the fourth week.

Advances in modern therapy, particularly in the field of respiratory support, have shifted the mortality curve to the right so that many patients survive somewhat longer but still eventually die. The critical fact to emerge from this study is that for most patients the die is cast at the time of the initial hemorrhage. Depending on the volume and rate of hemorrhage, the adequacy of compensatory pressure mechanisms, the cardio-respiratory fitness of the patient, and other factors, the patient's chances of survival are determined within the first few minutes.

In 98 consecutive cases of ruptured aneurysm admitted to the University of Alberta, the 33 who had an initial period of unconsciousness lasting hours had an 85% mortality. For the 65 who had no initial unconsciousness, or in whom it lasted only a few minutes, the mortality rate was 20% ($p<.005$).

The overall results in any hospital will therefore depend most critically on the rapidity with which patients with ruptured aneurysms are admitted to that hospital. If 90% of the admissions take place within the first 24 hours following the hemorrhage, the mortality rate will inevitably be high. If, on the other hand, the average patient is admitted two or three weeks following the hemorrhage the patient will already have successfully run the gauntlet of the initial destructive effects of the hemorrhage, rebleeding and vasospasm and the mortality rate should therefore be minimal and almost solely a reflection of medical and surgical complications.

At the University of Alberta where immediate and rapid admission to the Neurosurgical Service is the rule and more than two-thirds of cases were admitted within 24 hours of their bleed, the death rate due to the initial bleed was 22%, from vasospasm 10%, rebleeding 8%, and operative causes 4%. Figure 1 is schematic represent-

TABLE I

Neurological Grading of Patients

Grade	Botterrell 1956	Nishioka 1966	Hunt 1967*	Alvord 1976
I	conscious ∓ meningismus	no symptoms	asymptomatic or minimal headache and nuchal rigidity	asymptomatic
II	drowsy with deficit	alert ∓ headache no deficit	moderate to severe headache nuchal rigidity no deficit other than cranial nerve	minor symptoms headache meningismus diplopia
III	drowsy with deficit probably a clot	lethargic without deficit or alert with deficit	drowsy confused mild focal deficit	poorly responsive but capable of protective response to pain
IV	major neurological deficit or deteriorating with clot or older people with pre-existing cerebro- vascular disease	severely obtunded hemiparesis dysphasia confused	stuporose moderate to severe hemiparesis possibly early decerebrate rigid- ity and vegetative disturbances	very ill but not likely to die within 24 hours
V	moribund failing vital centers extensor rigidity	moribund	deep coma decerebrate rigidity moribund appearance	unstable vital signs likely to die in less than 24 hours

*put patient in next higher grade if serious systemic disease such as hypertension, diabetes, arteriosclerosis, chronic pulmonary disease, and severe vasospasm.

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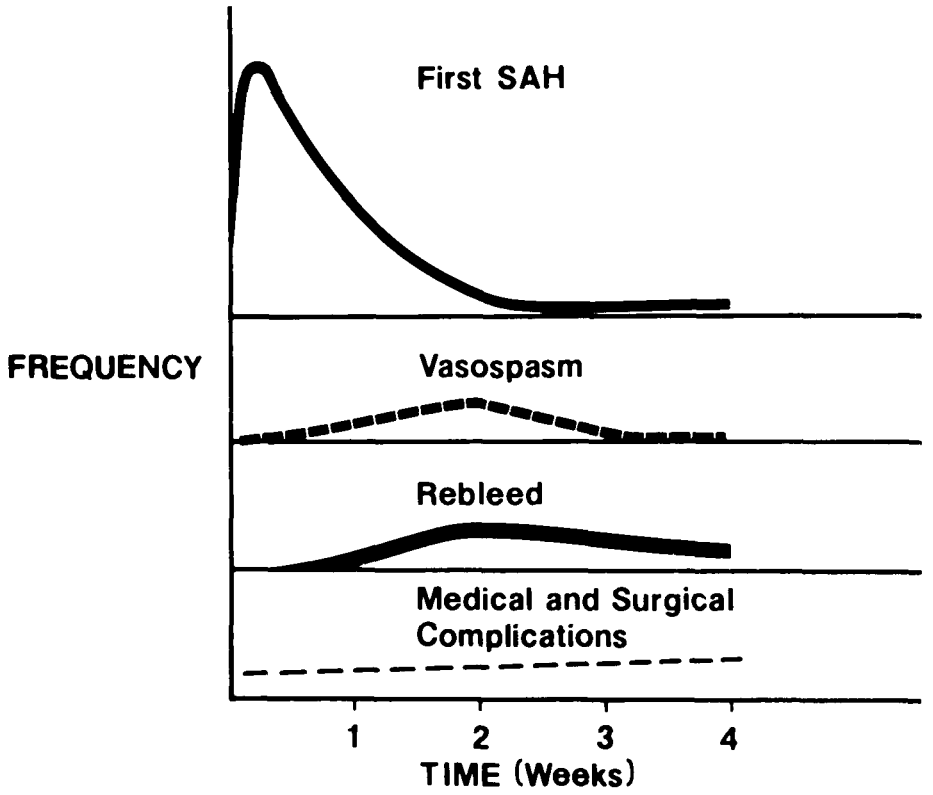

Figure 1 — Causes of death after aneurysm rupture

ation of the time course for mortality by etiological grouping.

Causes of Death

There are many possible causes of death following the rupture of an aneurysm. The final mechanism of demise by medullary failure can result from brain hernia due to space-occupying clot or edematous brain, cardio-respiratory failure, or metabolic dysfunction. Death can also result from the medical complications associated with the above.

Based on our autopsy series (Weir, 1978) it is likely that almost all patients dying acutely from the effects of the initial bleed have subarachnoid hemorrhage. One-half of our cases had intracerebral hemorrhage, almost one-third intraventricular hemorrhage, one-eighth mid-brain, and one-eighth subdural hemorrhages. Almost all the patients had cerebral edema. Half had systemic atherosclerosis and one-fifth significant renal disease. The most common systemic findings were in the lung. Initially pulmonary edema and congestion were extremely common but after a week or so pneumonia became relatively more common, as did pulmonary embolism.

Priorities in Medical Treatment

The priority in the preoperative medical management of ruptured aneurysms should be; firstly, the prevention of cerebral edema and herniation, secondly, the prevention of cerebral ischemia and infarction, thirdly, the prevention of rebleeding, and fourthly, the prevention of medical complications.

Patient Monitoring

A major step forward in our understanding of the natural history was the introduction of neurological grading systems. Some of the more widely employed ones are described in Table I. All of them have shortcomings although their widespread use has clearly established that the results of treatment, including surgery, are very importantly related to the grade of the patient at the time of treatment. The introduction in the last few years of the Glasgow Coma Scale (Jennett and Teasdale, 1977), particularly with reference to head injuries, has given an opportunity for the

TABLE 2

Glasgow Coma Scale

Eye Opening	
4	spontaneously
3	to speech
2	to pain
1	none
Best Verbal Response	
5	oriented
4	confused
3	inappropriate
2	incomprehensible
1	none
Best Motor Response	
6	obeys commands
5	localizes pain
4	withdraws
3	abnormal flexion
2	extension to pain
1	none

TABLE 3

Medical Complications in 100 Ruptured Aneurysms

Respiratory	54
Pneumonia	17
Pulmonary Edema	11
Hypoventilation	9
Atelectasis	6
Respiratory Arrest with Resuscitation	3
Pulmonary Embolism	1
Pneumothorax	1
Cardiovascular	23
Arrhythmia	14
Severe Hypotension	3
Thrombophlebitis	3
Myocardial Infarction	3
Genito-Urinary	26
Cystitis	16
Renal Failure	1
Vaginitis	9
Gastro-Intestinal	3
Hemorrhage	3
Preoperative Infection	3
Abscess Elbow	1
Abscess Scrotum	1
Parotitis	1

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TABLE 4

Orders on a Patient with Subarachnoid Hemorrhage from Ruptured Aneurysm and Significant Impairment of Consciousness

Nursing

Absolute bed rest
Restricted visitors
No smoking
Side rails, posev belt and mitts as necessary
Record vital signs q1/2 - 1 h as necessary
Intake and output hourly
Range of motion exercises to limbs q4h while awake
Log roll q2h if immobile
Anti-embolic stockings
Weigh daily
Naso-gastric tube to low suction
Rectal temperature probe and cooling blanket as necessary

Radiological Investigation

Cat scan on admission
Chest X-ray on admission
4-vessel angiography within day of admission
rCBF within 2 days of admission and on change of status

Laboratory Investigations

Hematology and biochemistry profile
Urinalysis
Serum and urine osmolality
Culture and sensitivity
— Tracheal aspirate
— Urine
— Blood
— Vaginal discharge
— Skin sores

Physiological Monitoring

Arterial BP
Intracranial pressure
Temperature
EKG
Central venous pressure and pulmonary artery wedge pressure if necessary
Arterial blood gases and ventilatory measurements

TABLE 5

Medication Used Following Sub-Arachnoid Hemorrhage in 100 Cases

Steroid	85	Histamine Blocker	15
Narcotic Analgesic	73	Muscle Relaxant	14
Tranquillizer	65	Anti-convulsant	9
Anti-Fibrinolytic	64	Pressor Drugs	9
Osmotic Diuretic	63	Aminophylline	7
Nitroprusside (O.R.)	52	Albumin	3
Anti-biotic	46	Hypotensive Drugs	3
Anti-pyretic	28	Thiopental Coma	3
Furosemide Diuretic	23	Digoxin	3
Antacid	21		

improvement in our assessment of patients with aneurysms (Table 2). Its wide-spread use in such patients should permit refinement in the assessment of results and factors influencing outcome. The nursing and resident staffs should carry out frequent neurologic assessment of the patients. This clinical monitoring is the essential data base upon which to make the critical decision regarding the timing of surgery and interim medical judgements.

Common Medical Complications

The medical complications encountered in our experience are documented in Table 3. It can be seen that respiratory complications are the most common grave and yet potentially treatable problems. In a large cooperative study assessing an antifibrolytic agent, the most serious medical complications encountered were uremia 3.5%, gastrointestinal hemorrhage 3%, convulsions 1.9%, pulmonary embolism .4%, and myocardial infarction .2%. More common but less serious medical complications were diarrhea 24.3%, restlessness 13.8%, genitourinary infection 10.1%, psychiatric disturbance 7.5%, nausea 6.7%, and vomiting 5.2%. Laboratory abnormalities were electrolyte imbalance 13.4%, anemia 8%, leukocytosis 7.5%, uric acid elevation 2.8%, and increased platelets .6% (Nibbelink et al, 1975).

A typical set of orders on a previously healthy adult patient with recent subarachnoid hemorrhage from ruptured aneurysm are listed in Table 4. It is impossible to make a totally inclusive listing and particular problems on individual patients will of course require specific management. Patients who are alert and cooperative will naturally require a less intensive level of nursing care. For a patient who is grade I it is probably wiser to err on the side of doing fewer vital signs or blood tests in order to keep the patient's anxiety level and blood pressure down. However, for the more seriously ill patient, where potent medications are going to be employed, a high level of observation and monitoring becomes mandatory.

Commonly Used Medications

In a survey of 100 consecutive cases,

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the medications received by our patients are documented in Table 5. Commonly employed medication orders are listed in Table 6. Many of these medications have complex side effects and the specific methods of administration and contraindications should be well known to those employing them (Goodman and Gillman, 1975). The dosages indicated would be for a 70 kg patient (Rotenberg and Hughes 1978). It should be stressed that the effect of some of these medications on the intracranial circulation both directly or indirectly is unknown, and that when several of these medications are employed simultaneously the possibility of drug interactions becomes significant. Most of these drugs have toxic side effects at certain dosage levels. Some of the major contraindications and side effects are listed in Table 7.

SPECIAL PROBLEMS

i) Cerebral Edema and Herniations

Cerebral edema and herniation can be treated with osmotic diuretics such as mannitol, hyperventilation, pentothal, ventricular drainage, hypothermia, steroids, and occasionally surgical decompression. Mannitol is usually effective (Langfitt, 1977). Thiopental can sometimes work in patients refractory to mannitol (Marshall et al, 1979).

ii) Rebleeding

The risk of rebleeding from aneurysms has been significantly reduced by the introduction of anti-fibrinolytic agents such as epsilon amino caproic acid (Amicar). The addition of hypotension either systemically by medication, or locally by partial occlusion of the carotid artery, has been claimed to further reduce the bleeding rate (Mullan et al, 1978) but unfortunately the consequences of this tend to aggravate the deleterious process of cerebral ischemia from other causes. Originally mortality and rebleed rates at 14 days post SAH were 20.9%, but with the use of antifibrinolytic agents this was brought down to a rebleed rate of 12.7% and a total

TABLE 6

Medication Orders (Assuming 70 kg. body weight)

Analgesics
- Meperidine HCl (Demerol) 50-100 mg/2-4 hrs im or po
- Codeine phosphate (Codeine) 30-60 mg/4-6 hrs im or po
- Acetylsalicylic Acid (Aspirin) 650 mg/4-6 hrs po

Sedative - Tranquilizer
- Phenobarbital (Luminal) 45 mg/6 hrs po
- Haloperidol (Haldol) 2.5-5 mg/4-6 hrs im

Anti-Convulsant
- Phenytoin (Dilantin) 100 mg/8 hrs po

Anti-fibrinolytic
- Epsilon amino caproic acid (Amicar) 32-48 gm/24 hrs iv

Steroid
- Dexamethasone Sodium Phosphate (Decadron) 8 mg/8 hrs iv

Antacid
- Magnesium Aluminum Hydroxide (Maalox) 10-20 ml/4 hrs
- Cimetidine (Tagamet) 300 mg/6 hrs po or iv

Diuretic, Anti-edema
- Furosemide (Lasix) 40 mg repeated in 1 hr prn iv
- Mannitol (Osmitrol) 200 gm/24 hrs iv

Stool Softener
- Dioctyl Sodium Sulfosuccinate (Colace) 50 - 480 mg/24 hrs po
- Psyllium (Metamucil) 4-10 gm/24 hrs po

Anti-hypertensive
- Hydrochlorothiazide (Hydrodiuril) 50-100 mg/12-24 hrs po
- Propanolol (Inderal) 20 mg/24 hrs, increase by 20mg/24 hrs for 1 week
- Methyldopa (Aldomet) 250 mg/8-12 hrs po
- Diazoxide (Hyperstat) 300 mg/4-12 hrs iv, start with 100 mg bolus
- Sodum Nitroprusside (Nipride) 500 mg/1000 ml 5% D/W ∞ response

Hypertensive agents
- Dopamine HCl (Intropin) 200 mg/500ml ∞ response
  adjust rate for CVP 10-15 cm H_2o or PAWP 14-18 mmHg

Volume Expander
- Human Plasma Protein Expander (Plasmanate) 83% Albumin in .9% NaCl 1000-1500 ml/24 hrs

Intra-cranial hypotensive agent
- Thiopental (Pentothal) 3-5 mg/kg initially then ∞ to response ICP

Bronchodilator
- Salbutamol (Ventolin) 1.25-2.5 mg in 5 ml .9% NaCl through IPPV at 10-20 cm H_2O for 5-20 min.

Cardiotonic
- Digoxin (Lanoxin) .25-.5 mg/4-6 hrs slowly iv as loading dose

Antibiotic and Antifungal agents
- As dictated by results of sensitivity testing

Muscle Relaxant
- Pancuronium Bromide (Pavulon) 3.8-5.6 mg iv

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mortality rate of 11.4% at 14 days in 469 patients (Nibbelink et al, 1975). In 50 patients treated with Amicar and hypotension, spasm and edema accounted for 20% of the deaths, rebleeding 10%, and postoperative deaths 6% (Ransohoff et al, 1972). In 103 patients reported with recent subarachnoid hemorrhage from supratentorial non-giant aneurysms by Mullan et al, 1978, the initial hemorrhage was the cause of death in 6%, secondary deterioration in 9%, and rebleeding in 6%. We have had very few deaths from rebleeding while the patient was actually receiving adequate (32-48 gm/day I.V.) amounts of Amicar and feel it should be continued up to the clipping of the aneurysm.

The institution of hypotensive treatment as prophylaxis against rebleeding is a controversial area in the medical management of patients with ruptured aneurysms. The hypertension frequently seen in the first two or three days following aneurysmal rupture in obtunded patients is frequently a compensatory mechanism resulting from the intracranial hypertensive state. In 109 cases receiving hypotensive therapy in the cooperative study (Sahs et al, 1969), it was not convincingly demonstrated that mortality could be reduced by greater reductions in systolic blood pressure. Mullan introduced the idea of subtotal reversible carotid occlusion using partial carotid clamping as prophylaxis against rebleeding. Mullan's initial paper on the subject warned that the hypotensive regime was dangerous in the severely obtunded patient (Mullan, 1975). In a subsequent report on 39 patients in whom subtotal carotid clamping was employed, he noted that 5 could not tolerate this procedure and that there were ten patients with complications (26%) (Mullan et al, 1978). Definitive recommendations on the efficacy of this treatment must await prospective studies, but we do not currently employ it. We think that the risk of rebleeding is less than the risk of ischemic neurologic deficit between

TABLE 7

Major "Contraindications" and (Side-Effects)

Analgesics
- Meperidine HCl "recent MAO inhibitors, supraventricular tachycardia" (potentiates other CNS depressants, addicting, increases airway resistance, urinary retention, constipation, nausea, vomiting, etc.)
- Codeine Phosphate "Hypersensitivity, respiratory depression" (suppresses cough reflex, increases airway secretion viscosity, etc.)
- Acetylsalicylic acid "hypersensitivity, ulcer, bleeding tendency"

Sedative - Tranquilizers
- Phenobarbital (addiction, CNS depression, drug interactions)
- Haloperidol "Parkinson's disease, depression, coma" (Interacts barbiturates, narcotics, anti-coagulants, tachycardia, etc.)

Anticonvulsant
- Phenytoin "lymph node hyperplasia, exfoliative dermatitis, sinus bradycardia, S-A block, second or third degree A-V block, Stokes-Adams syndrome" (drug interaction, nystagmus, ataxia, confusion, constipation)

Anti-fibrinolytic
- Epsilon amino caproic acid "acute intra-vascular clotting process, cardiac, hepatic, renal disease" (too rapid infusion gives hypotension, bradycardia, arrhythmia, diarrhea)

Steroid
- Dexamethasone "systemic fungal infection" (hyperacidity, gastric ulcer, hypertension, salt and water retention, may mask signs of infection or increase susceptibility to infection)

Antacid
- Magnesium Aluminum Hydroxide "severe debilitation, renal failure, alkalosis, hypermagnesemia" (diarrhea)
- Cimetidine "impaired renal function" (muscle pain, dizziness, rash, gynecomastia, tachycardia with overdose)

Diuretic, Anti-edema
- Furosemide "hypersensitivity, complete renal shut-down, concurrent use of curare, chloral hydrate" (can produce profound water and electrolyte depletion, reduction of blood volume and pressure, may precipitate diabetes mellitus, may potentiate effect of other hypotensive drugs)

Anti-hypertensive
- Hydrochlorothiazide "azotemia and oliguria" (hypersensitivity, interaction with lithium, other anti-hypertensives, narcotics, barbiturates, hypokalemia, hypochloremic alkalosis, hyponatremia)
- Propanolol (Inderal) "asthma, allergic rhinitis, sinus brachycardia, heart block, right ventricular failure secondary to pulmonary hypertension, congestive heart failure" (abrupt discontinuance may exacerbate angina pectoris, it may induce severe hypotension during anesthesia, may get bradycardia and reduced cardiac output after beta-adrenergic blockade)
- Methyldopa "active liver disease, hypersensitivity" (may potentiate other anti-hypertensive drugs and anesthetics, hemolytic anemia, granulocytopenia, various CNS, cardiovascular and GI symptoms)
- Diazoxide "aortic coarctation or A-V shunt" (hypersensitivity, can cause severe hypotension, hyperglycemia, salt and water retention, hyperuricemia, azotemia, etc.)
- Sodium Nitroprusside "compensatory hypertension, severe renal disease, hypothyroidism" (severe hypotension, rare fatalities from large doses in patients resistant to its hypotensive effects, metabolic acidosis)

Diuretic, Anti-edema
- Mannitol "severely impaired renal function, severe congestive heart failure, if initial moderate dose aggravates pulmonary edema, active intracranial bleeding" (circulatory overload, dehydration, hypovolemia, hyponatremia, hypo or hypertension, thrombophlebitis, convulsions, tachycardia)

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Hypertensive agent
Dopamine HCl "pheochromocytoma, tachy-arrhythmias, use with extreme caution with cyclopropane and halogenated hydrocarbon anesthetics" (may aggravate primary pulmonary hypertension, can cause limb ischemia particularly in Raynaud's disease, diabetes, atherosclerosis)

Volume Expander
Human plasma protein or serum albumin (observe for cirulatory overload and pulmonary edema)

Intra-cranial hypotensive agent
Thiopental "hypersensitivity, status asthmaticus, porphyria, myxedema, anemia, myasthenia, severe hepatic or renal disease" (may induce laryngospasm, cardiac and respiratory depression, can cause shivering, bronchospasm and hypotension)

Broncholilator
Salbutamol "hypersensitivity, simultaneous use of other β-adrenergic drugs" (headache, nausea, arrhythmia, hypotension, paradoxical increase in airway resistance)

Cardiotonic
Digoxin "incomplete A-V block, ventricular tachycardia" (low margin of safety, suspect toxicity if HR $<$ 60/min, can induce any type of arrhythmia, anorexia, nausea, jaw pain, visual changes)

Muscle relaxant
Pancuronium Bromide (absolute requirement for mechanical ventilation)

NOTE: this is not an inclusive list, almost all drugs can cause hypersensitivity or cross-reactions or are toxic at high dosages.

TABLE 8

Pharmacological Treatment of Vasospasm

1) β-adrenergic stimulant *Sundt, 1975*
Isoproterenol (Isuprel) .8 - 1.2 mg/150 ml 5% GW at 15-20 microdrops/min, decreased if premature ventricular contractions or heart rate $>$ 120/min
Lidocaine .2 gm/500 ml 5% G and .25% saline with 20 mEq at 20 microdrops/min

2) Serotonin depleting drugs and absorption blocker *Heros et al, 1976*
Reserpine (Serpasil) .8 mg/24 hrs
Kanamycin 3 gm/24 hrs

3) Simultaneous universal smooth muscle relaxant and systemic smooth muscle constrictor Allen, 1976
Nitroprusside (Nipride) 3.5 - 5.5 μgm/kg/min
Phenyleprine .3 μgm/kg/min

4) Simultaneous adenyl cyclase stimulant and phosphodiesterase inhibitor *Flamm & Ransohoff, 1976*
Isoproterenol (Isuprel) 125 μgm/hr less if HR $>$ 140 Aminophylline 125 mg/hr

TABLE 9

Fluids - Alimentation

1. Dextrose 5% to 10% in 0.25% to 0.5% NaCl IV 30 to 50 ml/kg/24 hr (100 ml/kg/24 hr in infants) + KCl prn

2. 2000 - 4000 cal/24 hr/70 kg + amino acids & vitamins

3. Reduce fluids if hyponatremia develops

days three and fourteen following a subarachnoid hemorrhage if the patient is on adequate anti-fibrinolytic medication. Inducing hypotension by medication or carotid partial clamping probably reduces the rebleeding but can simultaneously aggravate any ischemic neurologic deficit. Unfortunataely, it appears that the risk of one is approximately equal to the risk of the other so the physician is truly on the horns of a therapeutic dilemma.

A similar uncertainty surrounds the employment of lumbar puncture. Although others (Howe, 1977) recommend it in some circumstances, we currently are reticent to use this in patients in whom the diagnosis can be definitively and safely made by other means and in whom intracranial hypertension can be otherwise treated. In 23 patients out of 100, who were eventually transferred to us and who had lumbar puncture carried out, severe clinical deterioration occurred within 8 hours in 7 patients. While the association may be coincidental, prudence demands that this procedure be employed only where the possibility of meningitis is a serious consideration of differential diagnosis. The patient's intracranial pressure can be more safely monitored by intraventricular catheters or extradural electronic monitors. It is obvious that rebleeding from a recently ruptured aneurysm depends on the integrity of the clot in the aneurysmal wall and that reducing the intracranial extramural pressure while maintaining or raising the intramural arterial pressure is courting disaster. In addition, we know from autopsy studies and CAT scans that many patients have mass lesions and are poised on the brink of tentorial or foramen magnum herniations.

iii) Vasospasm

There is now little doubt that severe degrees of reduction in vessel caliber as seen angiographically are associated with a reduction in cerebral blood flow and the delayed onset of ischemic neurologic deficits, tending to occur between the third and fourteenth days

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following the hemorrhage. By delaying surgery for two weeks, this complication (as a postoperative complication) can generally be avoided (Flamm, 1977). The patient may be just as likely to develop it waiting for surgery and if the aneurysm is not clipped treatment will naturally be less aggressive and probably less effective.

As yet there have been no published controlled, prospective studies of drug regimens aimed at abolishing vasospasm and preventing or reversing the associated neurological deficit. We are therefore dependent on anecdotal evidence with respect to the efficacy of putative therapeutic agents. Some of the more widely known ones are listed in Table 8.

It seems that the data at present favor the use of a physiologic rather than a pharmacologic approach to the problem (Kosnik and Hunt, 1976; Gianotta et al, 1977; Brown et al, 1978, Pritz et al, 1978). Measures aimed at increasing blood volume and pressure as well as cardiac output should help to counteract the ischemic influences of the constricted larger arteries. In the presence of an unclipped aneurysm, hypervolemia and reduction of intracranial hypertension would appear to be safer than pharmacologically induced hypertension. However, if the patient is in danger of dying or is developing a massive neurological deficit, the judicious and controlled use of hypertension might be justified.

iv) Blood volume

The average hemoglobin in 100 of our patients fell from 14.5 ± .3 on admission to 12.6 ± at the lowest subsequent measurement. Serum albumin averaged on admission 3.9 ± .1 and also fell to 3.5 ± .1 subsequently. Careful studies by Maroon and Nelson, 1979 have established that there is a decrease in the red blood cell mass and total blood volume in patients following subarachnoid hemorrhage. They attribute this to bedrest, supine diuresis, negative nitrogen balance, decreased erythropoiesis, and iatrogenic blood loss. Great care must be taken in ensuring that the patient does not lose circulating red cell mass or blood volume as this can greatly aggravate other problems such as vasospasm or ischemia on another basis.

v) Fluids and Electrolytes

When a patient's level of consciousness is impaired or there exists some inability to drink fluids normally, caution must be exercised in maintaining alimentation. There is a great potential for iatrogenic misadventure. Basic requirements are indicated in Table 9 (Well and Henning, 1978). The patient should be kept in normal balance. The most common fluid and electrolyte problem is usually dehydration and inadequate replacement of electrolyte losses. Depending on the site of the aneurysm and the location of brain damage, however, specific problems can be encountered. The inappropriate secretion of antidiuretic hormone is one cause for reversible neurologic deterioration following aneurysmal rupture (Wise, 1978). This can be diagnosed when the serum sodium falls below 135 mEq/1, the serum osmolality is less than 280 mOsm/1, urinary sodium rises above 25 mEq/1, and the urine osmolality exceeds plasma osmolality. When these findings exist in the absence of significant renal or adrenal disease, peripheral edema or dehydration, the diagnosis is established (Fox et al, 1971). The other unique disorder which is rare following subarachnoid hemorrhage, but which must be diagnosed if it occurs, is diabetes insipidus, a water (not solute) polyuria. Urine specific gravity is between 1.001 - 1.005, urine osmolality falls to between 15-50 mOsm/kg, serum sodium rises above 145 mEq/1, and the patient is thirsty (Shucart and Jackson, 1976). Therapy of inappropriate secretion of ADH is water restriction, for diabetes insipidus it is replacement of fluid and electrolytes lost and the employment of antidiuretics. We use aqueous pitressin in the acute phase and DDAVP subsequently.

vi) Respiratory Failure, Neurogenic Pulmonary edema, Pulmonary Emboli

As indicated previously, the lungs are the site of much of the life threatening pathology which follows aneurysmal rupture. Intensive chest physiotherapy is essential in the obtunded patient. We now prefer intubation with a soft cuffed tube to tracheotomy if it is uncertain that respiratory support will be necessary for longer than a week or so. Clinical and laboratory indications for intubation in our hospitals are indicated in Table 10 (King, 1977). There have been several excellent recent reviews of the respiratory aspects of critical care

TABLE 10

Clinical Indications for Intubation

1. Ineffective cough or swallowing
2. Ventilatory incoordination or shallowness
3. Exhaustion due to excessive inspiratory and/or expiratory effort
4. Secretions of such consistency or quantity that patient cannot remove them
5. Iatrogenic muscular paralysis

Laboratory Indications for Intubation

1. Spontaneous tidal volume $V_T < 7$ ml/kg
2. Forced vital capacity $FV_c < 12$ ml/kg
3. Maximum negative pressure against a closed system Pinsp $<$ 20 cm H_2O
4. Alveolar to Arterial Oxygen Difference $A.aDO_2 > 300$ mm Hg
5. Ratio of Dead-space to Spontaneous Tidal Volume $V_d//v_t > .55$
6. If $[H^+]$ and $PaCO_2$ are increasing

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and the reader is referred to them (Skillman, 1975; Berk et al, 1976; Frost, 1977; Weil and Henning, 1978).

Neurogenic pulmonary edema is a catastrophic complication of subarachnoid hemorrhage of fatal proportions (Weir, 1978). Some patients can, however, recover. Its occurrence demands immediate intubation, ventilatory support, and frequent tracheal toilet. The standard drugs utilized in treating pulmonary edema such as morphine and digoxin should be considered for use. Furosemide in mega-dosage (i.e., ±1 gm) has proved life-saving in some cases (Schutz, 1978).

Pulmonary emboli are common following prolonged bed rest in patients after subarachnoid hemorrhage, particularly if there is a hemiplegia. The incidence is probably increased if steroids are in use. Mini-dose heparin is probably not indicated unless the aneurysm has been clipped. The introduction of an automated intermittent leg compression apparatus might prove useful in the prophylaxis of this complication (Mullan, 1978).

vii) Infection

It has not been our experience that prophylactic antibiotics have been useful in seriously ill patients with ruptured aneurysms; if anything the reverse was true. Controlled studies have not shown a benefit from the use of antibiotics in unconsciousness (Berk et al, 1976). Evidence for systemic infection demands prompt examination and appropriate cultures. Treatment should be specific for the offending organism. Meticulous technique on the part of those caring for him is the patient's best defence.

Patients on respirators are particularly susceptible to *enterobacter, klebsiella, serratia, pseudomonas, proteus, e. coli*, most of which are sensitive to gentamycin and carbenicillin or cephalothin. Bladder catheters are associated with urinary infections from the same organisms. Intravascular catheters are often associated with infections from *pseudomonas, enterobacter, serratia, candida, staph. epidermis*, and *enterococci*. Specific antibiotics should be selected (Berk at al, 1976). Penicillin G and cloxacillin may be drugs of choice for the staphylococcal infections. Amphotericin is the drug of choice for systemic candidiasis.

CONCLUSION

The prevention of medical complications requires the close cooperation between the neurosurgeon and his colleagues in critical care medicine. Good nursing and respiratory care are the bulwarks of such therapy. Surgery should be carried out on the patient as soon as he is in good neurological condition. Successful medical treatment will permit this surgery for definitive obliteration when the patient is in the best possible condition and it will preserve brain structure and function in the interim. Many of the systemic complications of coma, intracranial hypertension, and focal brain lesions can be treated or ameliorated. This successful treatment requires the combined skills of different specialties working in concert and supported by a dedicated nursing and technical staff.

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