Abstracts

AB-3015-77
Carotid Artery Endoscopy (Autopsy) — Olinger CP (Stroke Research Laboratory, 4303 Medical Science Building, Cincinnati, Ohio 45267) — Surg Neurol 7: 7-13 (Jan) 1977*

Intra-arterial endoscopy of the common carotid artery and its bifurcation was accomplished in 100 random autopsy cases. Resulting still photographs and movies reveal fatty dots and streaks, elevated gelatinous plaques, mural thrombi, thrombosis, ulcerations and ulcers with or without organizing thrombi, boulder and stalagmite-shaped lesions, bridging and elevated plaques in the carotid artery. These findings suggest possible explanations for some surgical and angiographic complications, bruits, transient ischemic attacks or strokes following reportedly normal carotid angiograms. Endoscopy aids in understanding the theories of inception, progression and sequela of atherosclerosis, the roles of risk factors, trauma and hemodynamics.

AB-3016-77
Intracranial Hemorrhage Associated With Anticoagulation Therapy — Snyder M, Renaudin J (Wadsworth Veterans Administration Hospital, Los Angeles, California 90083) — Surg Neurol 7: 31-34 (Jan) 1977*

Nine patients with intracranial hemorrhage associated with long-term anticoagulation therapy were evaluated at the Wadsworth Veterans Administration Hospital over a 30-month period. The relationships of the hemorrhage to other variables, both in this series and in the review of the literature, are analyzed. The most common site of the intracranial hemorrhage was the subdural space. No direct correlation between the presence of arterial hypertension or prothrombin activity and intracranial hemorrhage existed. The outcome was favorable in six of the patients, four of whom had a subdural hematoma.

AB-3017-77
Giant Hemispheric Arteriovenous Fistula in an Infant — Antunes JL (Department of Neurological Surgery, Neurological Institute of New York, New York, New York 10032), DiGiacinto GV, Michelsen WJ — Surg Neurol 7: 45-48 (Jan) 1977*

The authors present a case of an 11-month-old boy with a giant hemispheric arteriovenous fistula, which was successfully excised. The pathogenesis of the hydrocephalus present in this case is discussed.

AB-3018-77
Dissecting Aneurysm of Intracranial Arteries — Johnson AC, Graves VB (Columbus Hospital, Great Falls, Montana), Pfaff JP Jr — Surg Neurol 7: 49-52 (Jan) 1977*

The authors present a case of the sudden onset of hemiplegia in an eight-year-old boy secondary to a dissecting aneurysm of the right internal carotid artery and middle cerebral artery.

AB-3019-77

The purpose of this study was to determine the difference in prognosis among patients with transient ischemic attacks (TIA) in the carotid arterial system and those with TIA in the vertebral-basilar arterial system. Nearly twice as many patients had TIA in the carotid system as had TIA in the vertebral-basilar system. The survival rates for patients with carotid TIA and those with vertebral-basilar TIA were similar. In both groups, the survival rate was lower than the expected survival rate, but only for the patients with carotid TIA was the difference significant. There was no significant difference in the probability of the occurrence of stroke between patients with carotid TIA and those with vertebral-basilar TIA. These data also indicate that the risk of stroke is much greater soon after the onset of TIA in either arterial system. Among all patients with TIA, the primary cause of death was cardiac disease. The causes of death had a similar distribution for patients with carotid TIA, for those with vertebral-basilar TIA, and for those with "mixed" or "unknown" types of TIA.

AB-3020-77
The Use of Computed Axial Tomography (CAT) for the Diagnosis and Management of Intracranial Angiomas — Kendall BE (Lysholm Radiological Department, The National Hospital, Queen Square, London WC1N 3BG, England), Claveria LE — Neuroradiology 12: 141-160 (Dec 15) 1976*

The manifestations of angiomas on computed axial tomography (CAT) are described. Diagnostic changes were present in 66% and a focal abnormality, which was frequently suggestive of the diagnosis, in a further 27.5% of a consecutive series of 41 patients. It failed to show some focal abnormality in only two cases in which intravenous contrast was not given and in one case with a dural angiomatous malformation. The value of CAT in elucidating the nature of the complications of arteriovenous malformations is emphasized.

AB-3021-77
Nervous System Effects of Cardiac Arrest in Monkeys. Preservation of Vision — Myers RE (Laboratory of Perinatal Physiology, National Institute of Neurological and Communicative Disorders and Stroke, Room 108, Auburn Building, Bethesda, Maryland 20014), Yamaguchi S — Arch Neurol 34: 65-74 (Feb) 1977*

Thirteen juvenile monkeys were taught two visual discrimination tasks. After 12 to 24 hours of food deprivation, ten underwent 14-minute episodes of cardiac arrest. Three
served as controls. Five of the ten arrested animals survived and were tested in the discrimination box. All continued to perform color and pattern discrimination tasks with one to eight days’ delay. All appeared neurologically intact, while brain pathologic examination after 11 to 64 days’ survival showed either intact brains or injury restricted to nuclear structures in the brain stem, cerebellar Purkinje cells, and hippocampus. Five animals died 4 to 36 hours after they were resuscitated. Two required prolonged cardiac massage and, despite return of adequate cardiovascular function, died early. A third animal dislodged its arterial catheter and exsanguinated. The remaining two animals, who received infusions of glucose just prior to arrest, developed widespread fasciculations and myoclonic seizures. They became decerebrate and opisthotonic and were killed after 10 and 36 hours. Their brains showed mild edema and widespread necrosis of cortex and basal ganglia. Thus, food-deprived monkeys tolerate 14 minutes of circulatory arrest well and show minimal neurologic and pathologic changes, while administration of glucose just before arrest markedly augments the severity of brain injury and alters its distribution.

**AB-3022-77**

**Pure Motor Hemiplegia Due to Cerebral Cortical Infarction** — Chokroverty S (PO Box 127, Hines, Illinois 60141), Rubino FA, Haller C — *Arch Neurol* 34: 93–95 (Feb) 1977*

Although pure motor hemiplegia has not been reported after cerebral cortical infarction, occasional exceptions may occur. We provide three such examples. Necropsy study confirmed the site of lesion in one patient, and laboratory results (EEG and computerized axial tomography) suggested cortical involvement in the other two patients.

**AB-3023-77**


The several neurologic manifestations of hereditary hemorrhagic telangiectasia (HHT) may be caused by complications of pulmonary arteriovenous fistulae or associated central nervous system vascular malformations. The presence of skin and mucosal telangiectases should alert the clinician to the possibility of the disorder and in turn of its potential for associated neurologic disease, including cerebral hemorrhage and abscesses. This report describes two cases and demonstrates that the clinical spectrum of HHT should be enlarged to include its admittedly rare, but serious, neurologic aspects.

**AB-3024-77**

**Anoxic-Ishemic Encephalopathy in the Human Neonatal Period. The Significance of Brain Stem Involvement** — Leech RW (Department of Neuroscience [Neuropathology], University of North Dakota School of Medicine, Fargo, North Dakota 58102), Alvord EC Jr — *Arch Neurol* 34: 109–113 (Feb) 1977*

Although the human brain stem is considered relatively invulnerable to ischemic anoxia, evaluation of 16 cases of a single acute asphyxial episode either at or following birth indicates that such involvement is a frequent and characteristic aspect of anoxic encephalopathy in the infant. Ischemic cell change, neuronal loss, and nuclear or reticular formation gliosis were present in the brain stem of all but one infant. At least two topographic patterns of anoxic encephalopathy exist: (1) a rostrocaudal pattern of decreasing vulnerability, with the cerebral cortex being most sensitive and the brain stem least sensitive, and (2) a pattern of brain stem and thalamic damage. Of the two, the latter pattern appears to follow most acute asphyxial episodes in the human neonate and infant.

**AB-3025-77**

**Moya-Moya Disease in Black Adults** — Makoyo PZ (Department of Surgery, Santa Clara Valley Medical Center, San Jose, California 95128), Rapoport AM, Fleming RJ — *Arch Neurol* 34: 130 (Feb) 1977*

We describe two instances of Moya-Moya disease in black adults. Little is known concerning the origin of this disease. Currently, there is no effective treatment.

**AB-3026-77**

**Superior Sagittal Sinus Thrombosis** — Gettellinger DM, Kokmen E (Department of Neurology, University of Michigan Medical Center, Ann Arbor, Michigan 48109) — *Arch Neurol* 34: 2–6 (Jan) 1977*

Seven patients had superior sagittal sinus thrombosis diagnosed by cerebral arteriography. We recommend that anticoagulants not be used in the therapy of this entity, but rather that one should rely on antiedematous agents and anticonvulsants.

**AB-3027-77**


In 84 patients the wrist pulses were lost after diagnostic left heart catheterization via the brachial artery. Sixty-six of these patients underwent surgical exploration of the brachial artery for restoration of pulsatile arterial flow to the hand. This aim was achieved in 64 instances. In all cases, the artery was found to be occluded by fresh thrombus. In 36 patients, balloon thrombectomy and accurate arterial closure were successful. In the remainder, in addition to thrombectomy, identification of an area of damage to the arterial wall required additional surgical measures, usually in the form of resection and anastomosis. The surgical approach to this problem and the importance of the recognition and treatment of arterial wall damage by the catheter are stressed in this paper.

**AB-3028-77**

**Stereotaxic Clipping of Arteriovenous Malformations** — Kandel El (Neurosurgical Clinic, Institute of Neurology, Moscow D-367, U.S.S.R.), Peresedov VV — *J Neurosurg* 46: 12–23 (Jan) 1977*
In carefully selected cases of arterial aneurysms and deep-seated arteriovenous malformations (AVM), when direct attack may be dangerous or impossible, the authors advocate stereotaxic aneurysmatic. A special device and technique for its application are described. The instrument is introduced through a trephine opening and clipping is monitored by angiography. Successful results have been obtained in 10 operations performed on eight patients, three of whom had arterial aneurysms (two internal carotid and one anterior cerebral-anterior communicating) and five with AVM’s.

AB-3029-77

Fate of Small Diameter Cervical Veins Grafted Into the Common Carotid Arteries of Growing Rabbits — Bannister CM (Department of Neurosurgery, North Manchester General Hospital, Crumpsall, Manchester M8 6RB, England), Mundy LA, Mundy JE — J Neurosurg 46: 72-77 (Jan) 1977*

Autogenous cervical veins were grafted into the common carotid arteries of rabbits during their active growing period. A patency rate of 58.8% was achieved but the thin-walled vein grafts underwent massive dilatation. This dilatation is likely to severely limit the usefulness of these veins as a source of bridging grafts in clinical practice, particularly for use within the intracranial cavity.

AB-3030-77

Cavernous Sinus Syndrome Produced by Communication Between the External Carotid Artery and Cavernous Sinus — Edwards MS, Connolly ES (Ochsner Clinic, New Orleans, Louisiana 70121) — J Neurosurg 46: 92-96 (Jan) 1977*

The authors present two cases of cavernous sinus syndrome with spontaneous onset secondary to arteriovenous malformations and review the cases reported previously. These malformations enlarge slowly and produce symptoms only in adult life. Diagnosis may be difficult when there is no associated bruit. Adequate evaluation necessitates selective angiography of both the internal and external carotid artery circulation and the vertebral circulation. Conservative treatment is recommended unless symptoms worsen or there is progressive loss of vision.

AB-3031-77

Trigeminal Artery and Microemboli to the Brain Stem. Report of Two Cases — Waller FT, Simons RL, Kerber C, Kiesel 10, Tanabe CT (Division of Neurosurgery, University Hospital, S-221 85 Lund, Sweden), Hagerdal M, Kaasik AE, Siesjo BK — Brain Res 119: 223-231 (Jan 1) 1977*

The authors report two cases of transient ischemic attacks (TIA’s) involving the brain stem. The TIA’s were due to microemboli that originated from a carotid bifurcation atherosclerotic plaque and travelled through a persistent trigeminal artery.

AB-3032-77

Transient Amaurosis Under Decreased Atmospheric Pressure With Sphenoidal Sinus Dysplasia. Case Report — Sugita K (Department of Neurosurgery, Nagoya University School of Medicine, Tsurumai 65, Showa ku, Nagoya, Japan 466), Hirota T, Iguchi I, Kageyama N, Ito A — J Neurosurg 46: 111-114 (Jan) 1977*

The authors report a unique case of transient amaurosis occurring every time the patient flew in a jet plane and frequently when he drove up a mountain. X-ray examination showed dysplasia of the sphenoidal sinus and optic canals. The pathogenesis and the treatment of the amaurosis is discussed.

AB-3033-77

Giant Serpentine Aneurysm. Report of Two Cases — Segal HD, McLaurin RL (Division of Neurosurgery, University of Cincinnati College of Medicine, Cincinnati, Ohio 45267) — J Neurosurg 46: 115-120 (Jan) 1977*

The authors describe two cases of giant middle cerebral artery aneurysms presenting as mass lesions. Angiograms in each case revealed a distinctive serpentine vascular channel surrounded by an avascular area causing a “mass effect.” Both lesions were resected in toto with excellent clinical results. Similar lesions in the literature are noted and the pathophysiology and origin of this group of aneurysms are discussed.

AB-3034-77

A Catecholamine-Mediated Increase in Cerebral Oxygen Uptake During Immobilisation Stress in Rats — Carlson C (Brain Research Laboratory, University Hospital, S-221 85 Lund, Sweden), Hägerdal M, Kaasik AE, Siesjö BK — Brain Res 119: 223-231 (Jan 1) 1977*

 Anxiety and grave apprehension have been supposed to increase cerebral metabolism, and it has earlier been suggested that intravenous infusion of adrenaline may increase cerebral blood flow (CBF) and cerebral oxygen consumption (CMRO₂). In an experimental model on rats, it could be shown that immobilisation stress increased CBF and CMRO₂ after 5 min (about 150% of control values) and 30 min (about 190% of control values). By previous adrenalectomy or by administration of a beta-receptor blocker (propranolol, 1.4 mg/kg) the changes in CBF and CMRO₂ could be prevented. It is concluded that the excessive increase in CBF and CMRO₂ was mediated via release of catecholamines from the adrenal glands.

AB-3035-77

A Theory of the Mechanism of Cerebral Vasospasm and Its Reversal, the Role of Calcium and Cyclic AMP — Peterson EW (703-1081 Carling Avenue, Ottawa, Ontario K1Y 4G2, Canada), Leblanc R — Can J Neurol Sci 3: 223-226 (Nov) 1976*

It is proposed that the basic mechanism of vasospasm which sometimes follows subarachnoid hemorrhage is dependent on increased free intracellular calcium ion produced by spasmogens from closely applied extravasated blood. Relaxation of this spasm occurs when the intracellular cyclic AMP levels are raised, resulting in sequestration of calcium ion by the vascular smooth muscle cell sarcoplastic reticulum.

*Authors' abstract
The Reactivity of Canine Cerebral Arteries to O2 and CO2 In Vitro — Steinbok P (Department of Surgery, University of British Columbia, Vancouver V5Z 1L5, British Columbia, Canada), Kendall MJ, Clarke RJ, Peerless SJ — Can J Neurol Sci 3: 255-262 (Nov) 1976*

The responses of canine middle cerebral arteries to changes in pCO2 and pO2 were tested in vitro. It was found that there was no response to changes in pCO2 from 38.1 mm Hg to 26.6 mm Hg, but there was some constriction of the vessels with lowering of the pCO2 below 26.6 mm Hg and there was minimal dilatation of the vessels when the pCO2 was increased from 38.1 mm Hg to 87.2 mm Hg. There was no response to changes in pO2 from more than 500 mm Hg to 59.6 mm Hg, but when pO2 was lowered below 50, local Po2, there was an increase, massive constriction of the arteries tested. It is postulated that this constriction is due to build-up of a substance (substances) during a period of hypoxia (pO2 < 50 mm Hg). The significance of the results obtained are discussed.


Microflow was continuously recorded at four sites of the brain cortex (cat) during and after direct electrical stimulation of the brain. In some experiments local oxygen partial pressure (Po2) was additionally measured with a new combined element in the same capillary area where microflow was determined. This simultaneous measurement of both microflow and local Po2 in the tissue enabled us to analyze the kinetics of microflow and its dependence on local Po2 during activation. Microflow increased at all sites measured, in most cases within 1 — 2 s after the beginning of stimulation, reached the maximum of hyperemia after the end of stimulation and then gradually returned to the initial level within 30 s up to several minutes according to the intensity of the stimulation. The reaction pattern of microflow was uniform. As local Po2 normally did not decrease and did not even show an initial decrease after the onset of stimulation, the hyperemia could not be caused by local hypoxia. On the contrary, local Po2 always increased with the increase of microflow. This Po2 increase is necessary, because the tissue which consumes more oxygen needs higher Po2 gradients to transport the oxygen to the mitochondria.


Twenty-five patients have been operated on by means of extra-intracranial anastomosis (22 with STA-MCA anastomosis, 3 with occipital-MCA anastomosis).

Twenty-three patients underwent an angiographic study early after surgery (two weeks). The patency rate is 14 out of 23 (13 STA, 1 occipital anastomosis). In comparison to its pre-operative size, the afferent artery has enlarged in most cases, particularly in patients with complete obliteration, either of the carotid artery, or of the middle cerebral artery. In every case, only a limited part of the MCA territory is visualized through the anastomosis. In no case was the complete MCA field visualized; the frontal branches, particularly, are not supplied through a temporal anastomosis. In the case of occipital anastomosis, both upper and lower branches of MCA are supplied through the new channel.

Eleven patients underwent a second angiographic study, from one year through 28 months after the first one. In three patients with no patency on the first angiography the anastomosis remained non-patent. So, in this series, no anastomosis was seen to become patent secondarily.

In 8 patients with patency on the first control, the anastomosis remained patent on the second angiography. In patients with a pre-operative stenosis, no increasing of the size of the vessels could be noticed. The filling of the MCA branches is difficult to discuss, for in these cases, the angiographies were not performed selectively through the external carotid artery. In patients with a pre-operative thrombosis, an enlarging of the vessels was seen, as well as an extension of the intra-cranial filling through the anastomosis.

Clinical correlations are the following: most patients with TIA's had a stenosis. They presented no increasing of the size of the vessels. They were doing well after operation, as if a little more blood supply was sufficient to improve the general blood perfusion. Every patient with stroke had a pre-operative thrombosis and presented an enlarging of the vessels with a better filling on the second angiography, as if a great deal of additional blood supply was required; the clinical improvement is slow (3 out of 5) and remains often incomplete.

Abnormal CT-Scans in Migraine — Mathew NT (Department of Neurology, Baylor College of Medicine, Houston, Texas 77030), Meyer JS, Welch KMA, Neblett CR — Headache 16: 272-279 (Jan) 1977*

Twenty-nine patients with migraine were examined by CT-Scans. Ten of these had abnormalities on the scans which consisted of cerebral parenchymal low densitometric areas, ventricular enlargements and cortical atrophy. The cerebral parenchymal low densitometer zones tended to disappear on repeat scanning. Four cases in the series had hemiparetic migraine and all showed abnormalities on CT-scans. The incidence of complicated migraine was higher in patients who showed abnormalities on the scans than in those with normal scans. It is suggested that the parenchymal low density areas on CT-scans are the result of cerebral edema, but that repeated insults might lead to permanent changes in brain parenchyma which might be recognizable as cortical atrophy and ventricular enlargement.
AB-3040-77
Changes in Blood Clotting Systems During Migraine Attacks — Kalendovsky Z (Department of Neurology, University of Colorado Medical Center, Denver, Colorado 80220), Austin JH — Headache 16: 293-312 (Jan) 1976*

The activity of various blood clotting systems was measured during attacks of migraine complicated by prolonged focal cerebral dysfunction and contrasted with those observed when the same and other patients had uncomplicated migraine. Results differed in the two groups. In patients with complicated migraine, plasma coagulability rose during both complicated and uncomplicated attacks. During a complicated migraine attack, platelet aggregability to ADP also rose in all but one patient. Platelet aggregability fell in the same patients when they had uncomplicated migraine. Plasma coagulability consistently increased more when the headache was associated with focal cerebral dysfunction. In patients with uncomplicated migraine, plasma coagulability dropped during the later phase of their attacks. This finding may reflect the release of endogenous heparin from mast cells and basophilic leucocytes. Platelet aggregability to ADP decreased in some but rose in others. The results support the hypothesis that prolonged focal cerebral dysfunction may reflect thromboembolic phenomena. The influence of estrogens and genetic factors on clotting systems is reviewed.

AB-3041-77
"Migrainous" Neurologic Dysfunction in Patients With Prosthetic Cardiac Valves — Caplan LR (Neurology Department, Beth Israel Hospital, Boston, Massachusetts 02215), Weiner H, Weintraub RM, Austen WG — Headache 16: 218-221 (Nov) 1976*

Two patients developed frequent, multifocal, transient episodes of central nervous system dysfunction, frequently accompanied by headache, 14 and 42 months after insertion of prosthetic cardiac valves. The disorder satisfied the criteria for the diagnosis of classic migraine; it was self-limited in one patient and remitted after diphenylhydantoin in the other. Three other patients with "migrainous accompaniments" after cardiac surgery have been seen. "Symptomatic migraine" may occur in patients with prosthetic cardiac valves. The mechanism and etiology of this disorder is obscure.

AB-3042-77
Propranolol for Migraine Prophylaxis — Forssman B (Department of Neurology, University Hospital, Linköping, Sweden), Henriksson K-G, Johannsson V, Lindvall L, Lundin H — Headache 16: 238-245 (Nov) 1976*

The preventive effect of propranolol on migraine attacks was compared to placebo in a double-blind cross-over trial. Thirty-two patients with serious and prolonged migraine participated in the 12-week study. The effect of propranolol was significantly better than that of placebo. The number of migraine attacks during the propranolol period was reduced in 22 patients (69%), and in 11 of these (34%) a reduction of more than 50% was seen. The intensity of headache was significantly reduced during the propranolol period. The intake of analgesics and preparations containing ergotamine was significantly reduced during the propranolol period also. No serious side effects were noted.

AB-3043-77
Platelet Dysfunction in Migraine: Effect of Self-Medication With Aspirin — Deshmukh SV (Department of Neurology, Baylor College of Medicine, Houston, Texas 77030), Meyer JS, Mouche RJ — Thrombos Haemostas (Stuttg) 36: 319-324 (Nov 30) 1976*

Circulating microembolic index (CMI) was determined by drawing one blood sample into EDTA-formalin and the other into DTA alone in patients with migraine and compared with matched normal controls. Platelet aggregates, if any, are fixed in EDTA-formalin but disaggregated by DTA. Ratios of these two counts approximate "unity" in normals and are proportionately less than unity, depending on the number of platelet aggregates. 26 untreated migraineurs and 19 migraineurs with history of self-medication with aspirin taken within 72 hours of the test, were studied in headache-free intervals. Results were compared with those from 20 healthy, age and sex matched volunteers, without migraine, who were medication-free for at least one week. Mean CMI in untreated migraineurs (0.77 ± 0.03 SEM) was significantly lower than the mean in normal controls (0.94 ± 0.02, p. < 0.002). Migraineurs with self-administration of aspirin had mean CMI of 0.88 ± 0.02, differing significantly from untreated migraineurs (p < 0.01) but not from normal controls (0.1 < p < 0.2). Results suggest excessive platelet aggregation in migraineurs which tends to be corrected by treatment with platelet inhibitors such as aspirin.

AB-3044-77

Aneurysm of the internal carotid artery was repaired in three instances by a combination of gas endarterectomy and aneurysmorrhaphy. No shunt was used during cross clamping. The occlusion was uncomplicated. Both patients (in one the procedure was bilateral) have been well for two years. Because of its simplicity and effectiveness, the method is recommended.

AB-3045-77
Regression of Myointimal Thickening Following Carotid Endothelial Injury and Development of Aortic Foam Cell Lesions in Long Term Hypercholesterolemic Rats — Clowes AW (Department of Pathology, Harvard Medical School, Boston, Massachusetts 02115), Breslow JL, Karnaevsky MJ — Lab Invest 36: 73-81 (Jan) 1977*

In an earlier report (Clowes AW, Ryan GB, Breslow JL, Karnaevsky MJ: Lab Invest 35:6, 1976) we demonstrated that cholesterol feeding of rats led to hypercholesterolemia but no increase in smooth muscle cell (SMC) proliferation in

*Authors' abstract
right carotid arteries subjected to a standard endothelial injury when compared with normolipemic control animals. We have now examined these plaques at 6 months and 1 year after injury. In control animals, the carotid intimal thickening regressed to a relatively small, acellular, fibrous scar; there was no evidence of renewed endothelial injury and secondary SMC proliferation. Regression of the intimal thickening in the injured carotids of cholesterol-fed animals proceeded exactly as in control animals except for the accumulation of lipid. Unlike control animals, cholesterol-fed rats developed aortic intimal lesions containing extracellular lipid crystals and lipid-laden macrophages derived from mononuclear phagocytes in the blood. In addition to the lack of continued intimal SMC proliferation in the injured carotid, in the face of severe hypercholesterolemia the intima of the aorta did not contain mature SMC, or SMC-derived collagen and elastin. There was also no evidence of increased permeability to Evans blue, injected intravascularly. These findings suggest that hypercholesterolemia in the rat does not produce chronic endothelial injury, development of proliferative fibrous plaques, or enhancement of established SMC lesions.

**AB-3046-77**

Seven patients with fibromuscular dysplasia of the internal carotid arteries have been operated upon at Walter Reed Army Medical Center. One lesion was treated by graduated dilatation with Bake’s dilators combined with resection, end-to-end anastomosis, and vein patching of a tortuous segment. All other lesions were treated by graduated dilatation with an arterial dilator-shunt. All of these patients are asymptomatic presently. One patient has been operated with an arterial dilator-shunt. All of these patients are asymptomatic presently. One patient has been operated with an arterial dilator-shunt.

**AB-3047-77**
Computed Tomography: Contrast Enhancement in Resolving Intracerebral Hemorrhage — Messina AV (Department of Radiology, Section of Neuroradiology, New York Hospital-Cornell University Medical Center, New York, New York 10021) — Am J Roentgenol 127: 1050–1052 (Dec) 1976

Two cases are described in which resolving intracerebral hematomata about six weeks old appeared on CT scans as low density or mixed density lesions. The injection of 120 ml of Conray 60 caused enhancement of the ring-like capsules of the organizing hematomata. This appearance is similar to that of tumors, abscesses, and infarctions. Perhaps delayed scanning or a prolonged infusion of contrast medium would have resulted in even more enhancement.

**AB-3048-77**

Canine jugular veins were placed in dimethyl sulfoxide (DMSO) and frozen in liquid nitrogen vapor for as long as 28 days, then thawed and implanted in dog carotid artery. Patency rate after a year (62.5–87.5%) was the same as that of grafts of fresh veins (75%). Muscular contraction in these DMSO-treated veins was preserved, as was the architecture of the endothelium, as revealed by scanning electron microscopy. Veins frozen without DMSO cryopreservation made unacceptable carotid grafts, having a patency rate of 0 to 12.5% at a year. The endothelium had sloughed or been disrupted, exposing thrombogenic collagen.

**AB-3049-77**

In 332 carotid endarterectomies, internal carotid back pressures were measured. The patients were hypocarbic (<35 torr), normocarbic (35 to 45 torr), and hypercarbic anesthesia (>45 torr). The inverse relationship between carotid back pressure and $P_{\text{aco}_2}$ was highly significant. Internal shunting was used selectively in those individuals having a back pressure less than 25 torr. Overall stroke rate was 2% and mortality was 1.2%. Stroke rate was higher in those having hypercarbic anesthesia, though the difference was not statistically significant. The authors suggest that hypercarbic anesthesia may predispose to stroke.

**AB-3050-77**

A synaptosomal-enriched fraction of normal and ischemic (middle cerebral artery occlusion) baboon cortex was used to measure neurotransmitter uptake by the membrane filtration technique. Neuronin S-6, glutamate decarboxylase (GAD), and gamma-aminobutyric acid (GABA) uptake were all decreased in the ischemic cortex. GABA catabolism was a more sensitive marker of ischemia than was GAD ac-
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AB-3051-77
Physiological Role of Cerebrovascular Sympathetic Nerves in the Autoregulation of Cerebral Blood Flow — Edvinsson L (Department of Histology, University Hospital, University of Lund, Lund, Sweden), Owman C, Siesjö B — Brain Res 117: 519–523 (Dec 3) 1976

Halothane-anesthetized rats were made acutely hypertensive with an angiotensin infusion. Operatively-placed platinum electrodes placed around the sympathetic trunk just below the superior cervical ganglion were used to stimulate the cranial sympathetics. Sympathetic stimulation during hypertension almost prevented the cerebral hyperemia that was seen in the unstimulated animals. This is evidence of an autoregulatory role of the cerebrovascular sympathetics.

AB-3052-77
Utilization of Barium-Impregnated Ferromagnetic Silicone in Vascular Occlusion — Snyder M, Rand RW Department of Surgery, Division of Neurosurgery, UCLA School of Medicine, Los Angeles, California 90024) — Bull Los Angeles Neurol Soc 40: 145–152 (Oct) 1976

Ferromagnetic silicone embolization controlled by an external superconducting magnet has been used to occlude aneurysms and the vascular beds of tumors. The authors have added powdered barium sulfate to the material and produced a radiopaque embolization compound that obviates the need for intraoperative arteriograms during embolization. The material is not histotoxic and occludes feeding arteries and the capillary bed. In dogs, experimental embolization of the kidney without magnetic control sometimes led to retrograde embolization and paralysis. A case is presented of a 55-year-old man with a hypernephroma who underwent embolization of the tumor with the material under magnetic control. The vascular bed of the tumor was completely occluded.

AB-3053-77

Although this study confirms the frequent association of headache and hypertension, it also underlines the lack of specificity and the lack of correlation of headache with blood pressure levels and other prognostic parameters. The present study does not allow any conclusions as to the frequency of headaches in hypertensive subjects but does allow conclusions as to the relative prevalence in certain subgroups. Analysis of the experiences at the Cardio-Vascular Clinic, Sydney Hospital, demonstrates no correlation between the prevalence of headaches and ocular fundus grading, renal function, cardiac involvement or the presence of cerebral complications. A positive correlation was demonstrated between headache as a significant symptom and females, age at presentation between 30 and 45 years, the presence of chronic pyelonephritis and/or analgesic nephropathy and a family history of hypertension. Some of these correlations may merely reflect findings in the total population.

The author concludes that headache appears to be a signal of a sociopsychological disorder rather than a truly hypertensive symptom. It is often precipitated or aggravated by the recognition of hypertension, either in the individual or the family. Symptomatic relief does not relate to blood pressure lowering but to reassurance, suggestion, and cessation of analgesic abuse.

AB-3054-77

Intramuscular dexamethasone combined with intravenous low-molecular-weight dextran (dextran 40) was compared with placebo in 40 patients with acute ischaemic cerebral infarction. A double-blind procedure was used. Dexamethasone was given for up to 14 days and dextran 40 for up to three days after the infarction. A weighted scoring system was used to evaluate neurological state and mobility. There were no differences in mortality or in improvement of the neurological or mobility scores between the two groups.

AB-3055-77
Measurement of CBF and Carotid Artery Pressure Compared With Cerebral Angiography in Assessing Collateral Blood Supply After Carotid Ligation — Jawad K, Miller JD (Division of Neurological Surgery, Virginia Commonwealth University, Medical College of Virginia, Richmond, Virginia 23298), Wyper DJ, Rowan JO — J Neurosurg 46: 185–196 (Feb) 1977*

Angiographic assessment of collateral circulation to the brain at the circle of Willis was compared with measurements of cerebral blood flow (CBF) and internal carotid artery pressure during temporary carotid clamping in the prediction of tolerance of unilateral carotid ligation as treatment for intracranial carotid aneurysms in 92 patients. From CBF studies it was predicted that a substantial number of patients (27%) would suffer severe cerebral ischemia if carotid ligation were carried out. No single angiographic feature provided this predictive information. Bilateral fetal type of posterior communicating arteries were associated with significantly lower carotid artery back pressure on temporary carotid occlusion, and unilateral absence of posterior communicating arteries was related to lower CBF, but neither feature was associated with a significant reduction in the rate of successful carotid ligation. We believe that preliminary percutaneous digital carotid compression with electroencephalographic monitoring, followed by intraoperative measurement of the change in regional CBF and internal carotid artery pressure during temporary carotid clamping provides a safe method of selecting patients for carotid ligation. Carotid angiography with or without contralateral carotid compression is of little value in this respect.

*Authors' abstract
AB-3056-77

Treatement of Neurogenic Orthostatic Hypotension With a Monoamine Oxidase Inhibitor and Tyramine — Nanda RN (University Department of Neurology, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF, Scotland), Johnson RH, Keogh HJ — *Acta Neurol Belg* 76: 295-300, 1976*

The clinical response of treatment with a chemical preparation of tyramine and tranylcypromine, a monoamine oxidase inhibitor, is described in six patients with neurogenic orthostatic hypotension.

Previous therapy with fluorocortisone, ephedrine, elastic garments, postural training and, in one patient, an anti-G suit was unsuccessful. Oral and intravenous tyramine produced no pressor response. However, after treatment with tranylcypromine five of the patients when supine showed a marked rise of blood pressure to *intravenous* tyramine which was sustained for over two hours when they stood up. Tyramine given *orally* with tranylcypromine produced a moderate rise of blood pressure in the supine position which was sustained for over 3-4 hours in the erect position enabling patients to walk about without symptoms of orthostatic hypotension. Measurement of circulating adrenaline and noradrenaline during therapy suggested that the pressor response was due to release of noradrenaline. Three patients have had marked improvement for four, fifteen and twenty-four months respectively. In a further patient, therapy has been successful in treating his orthostatic hypotension although his mobility has been restricted due to cerebellar ataxia. One patient developed a confusional state during treatment and the therapy was stopped. The only patient in whom the drugs produced no pressor response had orthostatic hypotension with evidence of adrenergic innervation of blood vessels, but failure of noradrenaline release. It is suggested that the pressor response to a monoamine oxidase inhibitor and tyramine should be examined in patients with neurogenic orthostatic hypotension in whom conventional therapy is unsatisfactory and those who respond should receive a trial of this treatment.

AB-3057-77

Platelet Aggregation and Fibrinolytic Activity in Transient Cerebral Ischemia — Andersen LA, Gormsen J (Coagulation Laboratory, Sundby Hospital, DK-2300 Copenhagen S, Denmark) — *Acta Neurol Scand* 55: 76-82 (Jan) 1977*

The present study was performed in 34 patients with transient cerebral ischemia, TCI. Twenty-four of the patients were examined angiographically. Atherosclerotic abnormalities were demonstrated in 13 and a total occlusion of the interior carotid artery was found in one patient. The angiograms were normal in 10 patients. One patient suffered from hyperlipoproteinaemia, type IV, and one from diabetes mellitus. The platelet aggregation in *vitro* was increased significantly, as more patients than normal controls showed secondary aggregation with low ADP-concentrations: ≤ 1 μmol (p < 0.01). The fibrinolytic capacity was significantly reduced (p < 0.01) but not particularly in the patients with increased tendency for platelet aggregation. No correlation found between changes in platelet aggregation, the fibrinolytic activity and the angiographic findings. The results described may favor the concept that a prophylactic use of drugs exerting an antiaggregation effect on platelets might be useful in patients suffering from TCI.

AB-3058-77

Regression and Progression of Early Femoral Atherosclerosis in Treated Hyperlipoproteinemiac Patients — Barndt R Jr (Cardiology Section, SCOR-Atherosclerosis Program, University of Southern California, Los Angeles, California 90033), Blankenhorn DH, Crawford DW, Brooks SH — *Ann Intern Med* 86: 139-146 (Feb) 1977*

Femoral angiograms were done to evaluate change in early atherosclerosis in 12 patients with type IV hyperlipoproteinaemia and 13 with type II hyperlipoproteinaemia. The patients' average age was 48 years; only one had claudication. Elevated blood lipids and blood pressure were treated with drugs and diet. Repeat angiograms after an interval of 13 months showed regression of atherosclerosis in nine patients, no change in three, and progression in 13. Comparison of preangiogram levels with average levels between angiograms showed significant reduction in serum cholesterol, triglyceride, and blood pressure in the group with lesion improvement but not in the group with lesion progression. Sporadic examples of human atherosclerosis regression are known, but most other studies in man indicate only atherosclerosis progression. Our different result appears due to our selection of patients and radiographic method. We have studied patients with earlier atherosclerosis than previous workers, using a radiographic procedure more sensitive to small changes in lesions.

AB-3059-77

Computer Densitometry for Angiographic Assessment of Arterial Cholesterol Content and Gross Pathology in Human Atherosclerosis — Crawford DW (University of Southern California School of Medicine, Los Angeles, California 90033), Brooks SH, Selzer RH, Barndt R Jr, Beckenbach ES, Blankenhorn DH — *J Lab Clin Med* 89: 378-392 (Feb) 1977*

Sequential change studies in human atherosclerosis are desirable in disease regression trials but are now limited by dependence on the occurrence of epidemiologic end-points. Prior radiographic studies have pertained to advanced obstructive atherosclerosis. This is a study of measures applied by computer-generated densitometry of angiograms to assess early to advanced nonobstructive atherosclerosis. Measures are based on pathologic and angiographic appearance of all stages of atherosclerosis and include image edge roughness, local width, and local contrast density changes. Femoral angiograms were made in 21 cadavers under simulated clinical conditions, with a pressurized radiopaque casting material. Full-size color photographs were made of 10 cm. segments of opened artery, with matching cast and arterial specimens analyzed for cholesterol content. Four graders, on two occasions, sequenced the photographs in increasing order of disease on the basis of the International Atherosclerosis Grading scheme. The correlation
Dural Arteriovenous Malformation Treated by Artificial Embolization With Liquid Silicone — Manaka S (Department of Neurosurgery, University of Tokyo Hospital, Bunkyo-ku, Tokyo, Japan), Izawa M, Nawata H — Surg Neurol 7: 63–65 (Feb) 1977*

The successful treatment of a dural arteriovenous malformation in the region of the transverse-sigmoid sinus by artificial embolization with liquid silicone is reported. The clinical and radioanatomical characteristics of the lesion are described. The technical feasibility of embolization is discussed and compared with other surgical treatments.

Effect of Sodium Nitroprusside on Cerebral Blood Flow in Conscious Human Beings — Brown FD (Division of Neurosurgery, University of Virginia, Richmond, Virginia 23298) — Am J Roentgenol 128: 225–230 (Feb) 1977*

Hemispheric cerebral blood flow was measured in 14 patients prior to and after the injection of sodium nitroprusside. The intracarotid method of Xenon-133 was utilized, and flow was calculated by the flow initial technique. With a small reduction in blood pressure during the administration of sodium nitroprusside, the cerebral blood flow fell significantly by 15.9 ± 5.6%.

Micro-Balloon Catheter for Superselective Angiography and Therapeutic Occlusion — Pevsner PH (Department of Radiology, Medical College of Virginia, Richmond, Virginia 23298) — Am J Roentgenol 128: 225–230 (Feb) 1977*

A miniature (0.5 mm) flow-directed balloon catheter with two different silastic balloon tips has been developed. One tip is used for perfusion; the other can be inflated and released to produce vascular occlusion. Examples of the developmental work in dogs and clinical use in patients are presented.

Response of the Cerebral Circulation in Baboons to Changing Perfusion Pressure After Indomethacin — Pickard JD (Medical Research Council Cerebral Circulation Group, University of Glasgow, Glasgow, Scotland), MacDonell LA, MacKenzie ET, Harper AM — Circulation Research 40: 198–203 (Feb) 1977*

An earlier study has demonstrated that indomethacin, a prostaglandin synthesis inhibitor, blocks the cerebrovascular response to hypercapnia. This response is believed to be mediated by a lowering of pH in the cerebral interstitial fluid. Should autoregulation of cerebral blood flow (CBF) to changing perfusion pressure also be mediated by a changing interstitial pH (the "metabolic" theory), then indomethacin should impair autoregulation. This hypothesis was tested in anesthetized baboons. CBF was measured by the intracarotid 133Xe clearance technique; the preparation and the indomethacin protocol were identical to those of our previous investigation. Arterial pressure was increased by the intravenous infusion of angiotensin and decreased by controlled hemorrhage. Indomethacin was given by continuous infusion into the internal carotid artery. Although it reduced resting CBF, the cerebrovascular response to changing perfusion pressure was unchanged. Because indomethacin affects the response to changing CO2 but not to changing perfusion pressure, the mechanisms for these two reactions presumably are different and it is improbable that changing interstitial pH is responsible for autoregulation in the cerebral circulation.

The Prognosis of Ischemic Stroke in Young Adults — Hindfelt B (Department of Neurology, University Hospital, S-221 85 Lund, Sweden), Nilsson O — Acta Neurol Scand 55: 123–130 (Feb) 1977*

The report provides prognostic information on 60 patients (aged 16 to 40 years) with ischemic stroke. Immediate mortality from stroke is low and long-term mortality is due to other causes than cerebrovascular disease. The recovery from neurological deficits is good except for patients with occlusions of the internal carotid artery or the proximal part of the middle cerebral artery. Reinfarction is rare (about 0.5 per cent annually) and other late neurological complications do not seriously affect long-term prognosis. More than 80 per cent of the patients will be able to resume work on a full or part-time basis.

Brain Infarction in Young Adults — With Particular Reference to Pathogenesis — Hindfelt B (Department of Neurology, University Hospital, S-221 85 Lund, Sweden), Nilsson O — Acta Neurol Scand 55: 145–157 (Feb) 1977*

Sixty-four young adults (aged 16 to 40 years) with ischemic stroke were analyzed in retrospect with regard to possible pathogenetic mechanisms. In older patients various predisposing factors emerge (arterial hypertension, hyperlipidemia etc.) which are rare among younger age groups. In patients lacking predisposing causes the stroke incidence exhibits a seasonal variation. It is suggested that infection may be important for the development of ischemic stroke.

After the transition state of decerebrate coma, at least four different kinds of the so-called apallic status can be identified:

1. **Complete apallic syndrome:** coma vigil, alertness without any awareness, mass movements only, impairment of sleep rhythm, absence of any emotional responses, postural abnormalities, some primitive motor responses, tetraplegia, and alteration of muscle tone.

2. **Incomplete apallic syndrome:** some of the features of the complete apallic syndrome are lacking, and the patient shows emotional reactions with appropriate grimacing and some appropriate motor responses.

3. **False apallic syndrome:** most signs of complete apallic syndrome are present, but the patient is in touch with the environment. This condition is somewhat similar to the so-called lock-in syndrome.

4. **Functional apallic syndrome:** full clinical picture of the complete apallic syndrome but full recovery within a few days.

Long-term results in 62 patients, aged between 4 and 62, affected by a post-traumatic complete apallic syndrome are reported. Thirty-two patients were operated upon and 30 were not operated upon. Out of these cases, 38 died after weeks or months; 3 patients entered a chronic apallic status; 2 patients are improving; 10 recovered with severe neurological or psychic sequels or both; 4 recovered with minimal sequels, and 5 without sequels; no patients in these two last groups were aged more than 20.

**AB-3067-77**

Prevention of Postischemic Impairment of Microvascular Perfusion — Hallenbeck JM (Neurophysiology Division, Department of Hyperbaric Medicine and Physiology, Naval Medical Research Institute, Bethesda, Maryland 20014) — *Neurology (Minneap)* 27: 3–10 (Jan) 1977*

Two groups of conditioned male mongrel dogs, designated group 1 and group 2, were subjected to 35 minutes of cerebrospinal fluid compression ischemia followed by 30 minutes of recirculation. Animals in both groups were heparinized, but in addition, the blood of group 2 animals was circulated through three glass-wool filters over the course of 1 hour prior to induction of ischemia. Focal zones of impaired perfusion were prevented and general reflow was significantly enhanced in group 2 animals, whose blood had been filtered through glass-wool. Animals in group 1 had focal zones of impaired perfusion and relatively poor general postischemic reflow. A hypothesis concerning blood-damaged tissue interaction in zones of acute tissue damage is introduced.

**AB-3068-77**

Carotidynia — Raskin NH (Department of Neurology, University of California, San Francisco, California 94143), Prusiner S — *Neurology (Minneap)* 27: 43–46 (Jan) 1977*

Nosologic uncertainty about carotidynia has arisen, in part, because the syndrome was initially classified as an atypical facial neuralgia. More recently, carotidynia has been characterized as a recurring vascular neck pain, often accompanied by carotid tenderness and soft tissue swelling and sometimes by vascular headaches. We now report that drugs useful in the prophylaxis of migraine appear to be effective in carotidynia. Eight women (ages 39 to 77) with unilateral, episodic neck pain of 1 to 19 years' duration have been observed for periods ranging from 7 months to 6 years. All experienced marked relief in the intensity and frequency of their pain syndromes after the administration of methysergide, ergonovine maleate, propranolol, or nortriptyline. No patient had evidence of arthritis. The responsiveness of both migraine headaches and carotidynia to similar drugs suggests a common pathophysiologic mechanism.

**AB-3069-77**

Effects of Pentoxifylline on Cerebral Ultrastructure of Normal and Ischemic Gerbils — Hartmann JF (Department of Neurological Sciences, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois 60612), Becker RA, Cohen MM — *Neurology (Minneap)* 27: 77–84 (Jan) 1977*

Normal gerbils and those made ischemic for 15 to 60 minutes by bilateral common carotid artery occlusion were studied ultrastructurally after administration of the vasodilator drug pentoxifylline. In both groups, hypertrophy of neuronal mitochondria was found in the hippocampus and cerebral cortex. Planimetry of electron micrographs revealed a statistically significant increase in average mitochondrial size of drug-treated animals compared with untreated ischemic gerbils and normal controls; the treated ischemic group showed the greatest increase. Incubated cortical slices from normal gerbils that were given the drug 5 hours before they were killed showed a significant increase in oxygen consumption compared with controls.

**AB-3070-77**

Possibilities for a Cholinergic Action on Smooth Musculature and on Sympathetic Axons in Brain Vessels Mediated by Muscarinic and Nicotinic Receptors — Edvinsson L (Department of Histology, University of Lund, S-223 62 Lund, Sweden), Falck B, Owman C — *J Pharmacol Exp Ther* 200: 117–126 (Jan) 1977

Cat pial arteries suspended in a water bath were found to relax in the presence of low-dose (up to 10^{-9} M) acetylcholine and to contract after high doses. Both these responses were competitively inhibited by atropine, showing that muscarinic cholinergic receptors are involved and not sympathetic nerves.

In another experiment, pial arteries were exposed to Hnorepinephrine, which is taken up by adrenergic nerves and is released during nerve stimulation, as if it were endogenous norepinephrine. Radiation in the efflux was measured before and during electrical field stimulation. Radiation, and therefore norepinephrine release, was increased by electrical field stimulation. This increase was abolished by bretylium or guanethidine and enhanced by hexamethonium. Stimulation of the perivascular sympathetic nerves increased the radioactivity, but this was reduced by nicotine or acetylcholine. This suggests the presence of nicotinic cholinergic receptors, possibly on the adrenergic nerve terminals, providing an inhibitory axo-axonal interaction.
Clinical and Angiographic Features of Carotid Transient Ischemic Attacks — Pessin MS, Duncan GW, Mohr JP (Behavior Laboratory, Massachusetts General Hospital, Boston, Massachusetts 02114), Foskaner DC — N Engl J Med 296: 358-362 (Feb 17) 1977

Ninety-five consecutive patients with carotid system transient ischemic attacks who had carotid angiography were studied. Fifty-two of them had transient hemisphere symptoms, 33 had had amaurosis fugax, and ten had had attacks of both kinds. Forty-nine patients had tight carotid stenosis (residual lumen less than 2 mm) or occlusion. Thirteen patients had intracranial branch occlusions, four of whom had tight carotid stenosis as well. The rest had open carotid arteries. Amaurosis fugax appeared with equal frequency in the tight and open groups.

Patients with long-duration transient hemispheral attacks (>60 minutes) tended to have open carotids, while those with short-duration attacks were about equally divided. Of patients who experienced non-simultaneous amaurosis fugax and hemispheral attacks, 80% had tight stenosis. Several patients with open carotids had long-duration hemispheral attacks with intracranial branch occlusion, suggesting embolic occlusion, probably from a cardiac source. Some patients with normal carotid arteries may have suffered lacunar strokes.

A Comparison of Intimal Proliferation in Experimental Subarachnoid Hemorrhage and Atherosclerosis — Alkne JF (University of California Medical Center, San Diego, California 92103), Branson PJ — Angiology 27: 712-720 (Dec) 1976

Fourteen adult Macaques underwent experimental subarachnoid hemorrhage. The histology of their cerebral vessels was studied at intervals of four to 70 days after the hemorrhage. The changes progressed from subendothelial edema, medial necrosis, and fragmentation of the elastica, to intimal proliferation. The proliferation began in the first week. After 70 days, some vessels were nearly normal and some were nearly occluded.

It is felt that this same sequence from edema to intimal proliferation takes place in atherogenesis and may be a nonspecific vascular response to injury.

Optimal Exposure of the Internal Carotid Artery for Endarterectomy — DePalma RG (University Hospitals of Cleveland, Cleveland, Ohio) — Surg Gynecol Obstet 144: 249-250 (Feb) 1977

Conventional arteriotomy at the carotid bifurcation may give inadequate surgical exposure if the atheromatous plaque extends cephalad into the internal carotid artery. The author describes a method which provides better exposure. The incision begins behind the ear lobe and follows the neck skin lines to the midline. After obtaining exposure of the common carotid artery and its branches, the hypoglossal nerve is carefully retracted, the stylohyoid and digastric muscles are divided, and so is the ansa hypoglossi. Tran sillumination of the internal carotid can be used to identify the cephalad extent of the atheroma.


Takayasu’s arteritis is an inflammatory disease of the aorta and its major branches, occurring predominantly in females (8.5:1), and beginning between the ages of ten and 20. In half the cases, the onset coincides with a transient syndrome of fever, anorexia, weight loss, joint pains, hypertension, and an elevated sedimentation rate. The chronic phase is one of gradual deterioration, sometimes with brief exacerbations. Neurologic symptoms are due to hypertension and to brain and cord ischemia. Clinically, peripheral pulses are decreased (96%), bruits are present, and the ocular fundi may show a capillary flush, wreath-like peripapillary arteriovenous anastomoses. Blindness from cataracts may occur. Hypertension from renal arterial involvement is common. Four types are recognized, determined by the areas of arteritic involvement. In the commonest type (type III, 65%), the abdominal aorta and supraaortic trunks are involved. Tuberculosis may play a role in the etiology. Hypertension and heart failure need treatment. The role of steroids in management of this disease is unclear.


Three types of angiodysplasia are described: the arteriovenous fistula (dilatation of the artery and vein with rapid circulation), capillary angiodysplasia (abnormally rich arterio-capillary density with slow circulation and slightly dilated artery and vein), and venous angiodysplasia (stag-
nent phlebectasias without dilatation of artery and vein). Twenty-seven cases have been safely embolized using superselective catheter arteriography. Twenty-one had satisfactory results. Seven patients underwent more than one embolization.

AB-3077-77
Ischemic and Post-Ischemic Effects on the 2-deoxy-D-[H]Glucose Uptake in Cerebral Capillaries — Spatz M (Laboratory of Neuropathology and Neuroanatomical Sciences, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health, Bethesda, Maryland 20014), Mrsulja BB, Micic D, Mrsulja BJ, Klatzo I — Brain Res 120: 141-145 (Jan 14) 1977

Ischemic and non-ischemic capillaries were isolated from the brains of Mongolian gerbils. Capillary uptake of 2-deoxy-D-[H]glucose was decreased in capillaries from brains in which the carotid occlusion had lasted for six minutes, but not when it had lasted one to three minutes. After the release of the occlusion, 2-deoxy-D-[H]glucose uptake increased over control values, and the level uptake depended on both the duration and the severity of the ischemia. The uptake may be energy-dependent, at least during ischemia. The post-ischemic augmentation of uptake could be due also to leakage from damaged capillaries.

AB-3077-77
Effect of Repeated Cerebral Ischemia on Metabolites and Metabolic Rate in Gerbil Cortex — Mrsulja BB (Institute of Biochemistry, School of Medicine, Belgrade, Yugoslavia), Lust WD, Mrsulja BJ, Passonneau JV — Brain Res 119: 480-486 (Jan 7) 1977

In the Mongolian gerbil, which often has anomalies of the circle of Willis, occlusion of one common carotid artery often results in hemispheric ischemia, with neurologic symptoms and cortical depletion of glucose, glycogen, ATP, P-creatine, and putative neurotransmitters. Carotid occlusion was maintained for one hour, then released for one hour, and then repeated for either five or 60 minutes. During this second ischemic insult, metabolite reduction was considerably more reduced than by a single ischemic insult. After longer periods of recovery (five or 20 hours; one or two weeks), however, the brain was more tolerant to ischemia, and a second period of occlusion had little effect on metabolite and neurotransmitter determinations, and the clinical neurologic manifestations were much less marked.

AB-3077-77
Role of Pressure Gradients and Bulk Flow in Dynamics of Vasogenic Brain Edema — Reulen HJ (Neurosurgical Clinic, University of Mainz, Langenbeckstrasse 1, 65 Mainz, West Germany), Graham R, Spatz M, Klatzo I — J Neurosurg 46: 24-35 (Jan) 1977*

The authors present the results of an investigation of the vasogenic type of brain edema using cold injury in cats as a model. Their findings indicate that bulk flow and not diffusion should be considered the main mechanism for the spread of edema through the white matter. This conclusion is based on: 1) comparison of the distances actually traveled by various substances during edema spread with those calculated theoretically for migration of the substances by diffusion; 2) coincidence in the speed of movement by two substances (sucrose and albumin) with widely different diffusion coefficients; 3) measurement of interstitial fluid pressure (IFP) at various distances from the lesion showing the presence of increased IFP in the lesion area and decreasing pressures along the edema pathway toward the normal tissue; and 4) the fact that spreading of edema can be significantly impeded by inducing before the cold lesion an intracellular type of brain edema that reduces the size of the extracellular space (ECS) and increases the resistance to flow of edema fluid. The pressure-volume curve of the brain ECS, as derived from determinations of IFP and tissue water content, indicates that an initial steep slope in IFP probably represents the high resistance to fluid mobility through the small diameter extracellular channels and the counteracting resistance of the intermingled structures of brain parenchyma to be separated. Once the IFP exceeds these opposing forces, the ECS dilates, fluid mobility increases, and the edema front advances.

AB-3080-77
Influence of Systemic and Cerebral Vascular Factors on the Cerebrospinal Fluid Pulse Waves — Hamer J (Department of Neurosurgery, University of Heidelberg, D-6900 Heidelberg, West Germany), Alberti E, Hoyer S, Wiedemann K — J Neurosurg 46: 36-45 (Jan) 1977*

In anesthetized, artificially ventilated dogs, the intracranial cerebrospinal fluid (CSF) pulse waves were studied simultaneously with the central aortic pressure, central venous pressure (CVP), and the sagittal sinus pressure under physiological conditions and in normovolemic arterial hypotension and hypertension, in acute cardiac insufficiency of the right atrium, in raised intracranial pressure (ICP), and in arterial hypoxemia. The physiological CSF pulsations are shown to be mainly arterial in origin. In the diastolic phase, the descending part of the pulse curve can be modified by venous superpositions coinciding with the right atrial “A” wave. With increase of ICP the configuration of the CSF pulsations changes; the venous superpositions disappear and the waves become more and more arterial in shape. Furthermore, the pulse amplitude increases considerably. The same change can be observed when cerebral vessels are dilated by arterial hypoxemia. During cardiac insufficiency and consecutive increase of CVP, the CSF pulse curve is venous in shape and the right atrial “A” wave predominates. In arterial hypotension, CSF pressure decreased. Conversely, in angiotensin-induced systemic arterial hypertension, CSF pressure and its pulse amplitude increased. It is concluded that both systemic arterial blood pressure and cerebrovascular reactivity are major determinants for the shape and the pressure amplitude of the intracranial CSF pulse waves. In the presence of cerebral vasodilatation, systemic arterial blood pressure may be an important factor in raising ICP and altering the brain tissue compliance, because cerebral vascular damping of the arterial pulse is diminished and the arterial pressure head may be directly transmitted to the cerebral capillary bed.

*Authors' abstract
Cerebral Blood Flow Changes in Cluster Headache — Norris JW (MacLachlan Stroke Unit, Sunnybrook Hospital, Toronto, Ontario M4N 3M5, Canada), Hachinski VC, Cooper PW — Acta Neurol Scand 54: 371–374 (Oct) 1976*

Serial cerebral blood flow studies performed by the intra-carotid xenon method were fortuitously determined during the course of a cluster headache in a 32 year old man. The initial study was performed about 10 min after the headache began and showed values at the upper limit of normal. Twenty min after the headache started a second procedure showed that the autoregulatory response on hyperventilation was normal. Ergotamine tartrate was given intra-muscularly 23 min after the headache began and there was partial relief. A third cerebral blood flow estimation showed abnormally high values. The probable reasons for this are discussed.

Relationship of Cerebral Oxygen Uptake to EEG Frequency in Isolated Canine Brain — Fitzpatrick JH Jr (Department of Neurosurgery, University of Wisconsin, Madison, Wisconsin 53706), Gilboe DD, Drewes LR, Betz AL — Am J Physiol 231: 1840–1846 (Dec) 1976*

Cerebral oxygen uptake was correlated with electroencephalographic (EEG) frequency and amplitude in 87 isolated canine brains. Group I (71 brains) was perfused with diluted blood and Group II (16 brains) was perfused with whole blood equilibrated with oxygen at various partial pressures. The EEG's were classified as follows: A, highest frequency ≥ 17 Hz, α (8–13 Hz) amplitude < 50μV, δ (≤ 3.5 Hz), amplitude < 100μV; B, highest frequency ≥ 17 Hz, α amplitude ≥ 50 μV, and/or δ amplitude ≥ 100 μV; C, highest frequency 8–16 Hz, α amplitude ≥ 25 μV, and/or δ amplitude ≥ 100 μV, D, highest frequency 0.5–16 Hz, α, if present, amplitude < 25 μV, and/or δ amplitude < 100 μV, E, highest frequency 0–16 Hz, α, if present, amplitude < 10 μV, and/or δ amplitude < 15 μV. The Group I oxygen uptakes in ml/100 g of brain per min ± SE for the five EEG classifications were: A, 4.39 ± 0.06, B, 4.13 ± 0.08, C, 3.76 ± 0.09, D, 3.40 ± 0.12, and E, 2.55 ± 0.60, whereas the corresponding Group II values were A, 4.64 ± 0.22, B, 4.28 ± 0.15, C, 3.82 ± 0.24, D, 3.39 ± 0.40, and E, 1.38 ± 0.42. As the EEG deteriorates, cerebral oxygen uptake tends to decrease in a significant and parallel manner in both the diluted and whole blood groups.


Cerebral embolism can manifest itself in certain cases as pure psychosis. In the absence of neurological symptoms it might be mistaken for schizophrenia or manic-depressive psychosis.

Cardiac disease and cardiac surgery involve a high risk of embolism. Microembolism plays a special role with extracorporeal circulation. There is a significant increase of postoperative psychosis in cases with E.C.C. in comparison to closed heart surgery. Immediately postoperatively there occurs what has been described as the "catastrophic reaction" or "immobilization syndrome". This reaction is in fact an akinetic, parkinsonian-like state for which there is good evidence that it is due to transient microembolism of the basal ganglia ("striatum apoplexy"). After its disappearance around the 3rd-5th day "cardiac psychoses" (cardiac delirium) may manifest themselves.

Patients who develop these "late" psychoses have a significantly higher correlation with endogenous psychoses in their family histories. On the psychopathological level — in the absence of disturbances of consciousness and orientation — it is not possible to differentiate between "exogenous" and "endogenous" psychosis. A special type of psychopathological reaction is dependent, as in neurological disease, on the severity of brain damage, its localization and on hereditary factors.

Changes in Carotid Flow Velocity During Catheterization of the Aortic Arch and Common Carotid Artery — Thijsen HOM (Department of Neuroradiology, St. Radboud Hospital, University of Nijmegen, Nijmegen, The Netherlands), Colon E, Merx H — Neuroradiology 12: 171–175 (Dec 15) 1976*

Quantitative percutaneous flow velocity measurements are possible in carotid arteries by means of a directional Doppler flow velocity device and registration on a polygraph. Carotid flow velocity changes were recorded during catheterization of the ascending aorta and common carotid artery and after infusion of 20 cc contrast medium in the ascending aorta. The results indicate an increase in the flow velocity in the right common carotid artery after infusion of the contrast medium in the ascending aorta, which is thought to be attributed to a diffuse vasodilatation, at least in the cerebral circulation. The catheterization of the ascending aorta and common carotid artery seems to interfere with the common carotid flow velocity.

Effects of Bupranolol, a New β-Blocker, on Platelet Functions of Rabbit and Human In Vitro — Umetsu T (Research Laboratory, Kaken Chemical Co., Ltd., Honkomagome 2-28-8, Bunkyo-ku, Tokyo, Japan), Sanai K, Kato T — Thrombos Haemostas (Stuttg) 36: 376–387 (Nov 30) 1976*

The effects of bupranolol, a new β-blocker on platelet functions were investigated in vitro in rabbits and humans as compared with propranolol, a well-known β-blocker. At first, the effect of adrenaline on ADP-induced rabbit platelet aggregation was studied because adrenaline alone induces little or no aggregation of rabbit platelets. Enhancement of ADP-induced rabbit platelet aggregation by adrenaline was confirmed, as previously reported by Sinakos and Caen (1967). In addition the degree of the enhancement was
proved to be markedly affected by the concentration of ADP and to increase with decreasing concentration of ADP, although the maximum aggregation (percent) was decreased.

Bupranolol and propranolol inhibited the (adrenaline-ADP)-induced aggregation of rabbit platelets, bupranolol being approximately 2.4-3.2 times as effective as propranolol. Bupranolol stimulated the disaggregation of platelet aggregates induced by a combination of adrenaline and ADP, but propranolol did not. Platelet adhesion in rabbit was also inhibited by the β-blockers and bupranolol was more active than propranolol. With human platelets, aggregation induced by adrenaline was inhibited by bupranolol about 2.8-3.3 times as effectively as propranolol.

From these findings, we would suggest that bupranolol might be useful for prevention or treatment of thrombosis.

**AB-3086-77**

**Inhibitory Effect of Acetylsalicylic Acid on Human Platelet Function in Normal Volunteers and in Women Using a Combined Oral Contraceptive Regime** — Baele G (Department of Medicine, University Hospital, B-9000 Ghent, Belgium), Thiery M, Vermeulen A, Barbier F — Thrombos Haemostas (Stuttgart) 36: 623-627 (Dec 31) 1976*

Six parameters related to the release reaction were measured simultaneously in 10 human volunteers prior to the intake of one single dose of 1 g acetylsalicylic acid and 1, 4, 5, 6 and 7 days later: ΔE with diluted collagen, ΔE with Thrombofax® and the serotonin-14C release by undiluted and diluted collagen, by Thrombofax and by bovine plasma. The duration of the inhibitory effect varied according to the test used. It was the most prolonged (through the 7th day) if serotonin-14C release by diluted collagen was measured.

A systematic investigation of the platelet release reaction in women taking a combined oral contraceptive was also performed. There were no statistically significant differences from a control group. No difference in acetylsalicylic acid sensitivity, measured 24 hours after intake of 1 g of aspirin, could be demonstrated.

**AB-3087-77**

**Effect of Aspirin on Platelet Phospholipids** — Valles J, Aznar J (Department of Clinical Pathology, Ciudad Sanitaria "La Fe", Valencia, Spain), Santos MT — Thrombos Haemostas (Stuttgart) 36: 628-633 (Dec 31) 1976*

It was observed that the acetylsalicylic acid "in vitro" (final concentration 10-4 M) as well as "in vivo" (1 g of aspirin) caused a platelet phospholipids variation which basically consisted of:

1. A diminution of the phospholipids/proteins rate of 22%.
2. A reduction of sphingomyelin "in vivo" of 27.66% and "in vitro" of 16.82%.
3. An increase in phosphatidyl choline "in vivo" of 12.24% and "in vitro" 10.28%.

The possible effects that these changes might have on the platelet function are evaluated.

**AB-3088-77**

**Spontaneous Thrombosis of an Intracranial Aneurysm During Treatment With Epsilon Aminocaproic Acid** — Scott RM (Department of Neurosurgery, Tufts-New England Medical Center Hospital, Boston, Massachusetts 02111), Garrido E — Surg Neurol 7: 21-23 (Jan) 1977*

The authors report spontaneous thrombosis of a middle cerebral artery aneurysm in a patient receiving the anti-fibrinolytic agent, epsilon aminocaproic acid. Intense vasospasm and profound neurologic deficit accompanied the initial hemorrhage. Follow-up angiography two, four and ten weeks following the initial hemorrhage demonstrated occlusion of the aneurysm and progressive thrombosis of a middle cerebral artery branch despite the disappearance of vasospasm. The patient had a persistent hemiparesis.

**AB-3089-77**

**The Nonlinear Responses of Cerebral Metabolism to Low Concentrations of Halothane, Enflurane, Isoflurane, and Thiopental** — Stullken EH Jr, Milde JH, Michenfelder JD (Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota 55901), Tinker JH — Anesthesiology 46: 28-34 (Jan) 1977*

The relationship between cerebral oxygen consumption (CMRO2) and anesthetic concentration has been assumed (based upon isolated measurements) to be approximately linear at concentrations less than 1 MAC. The shapes of the anesthetic dose-response curves for both CMRO2 and cerebral blood flow (CBF) were examined by multiple measurements made at small, progressive concentration increments from 0 to 2 MAC halothane (six dogs), enflurane (six dogs), and isoflurane (six dogs), and during a constant 23 mg/kg/hr infusion of thiopental (six dogs). The EEG was continuously recorded and changes in EEG patterns from "awake" to "anesthetic" were correlated with changes in anesthetic concentration, CBF, and CMRO2. The significance of changes in the slopes of regression lines for CMRO2 before, during and after changes in EEG patterns from "awake" to "anesthetic" was then determined.

Contrary to previous inferences, CMRO2 dose-response curves were found to be nonlinear at anesthetic concentrations less than 1 MAC for all anesthetics studied. CMRO2 decreased precipitously until a stable "anesthetic" pattern was observed on the EEG; thereafter, CMRO2 decreased at a markedly reduced rate. The onset of this change occurred at concentrations well below MAC for the inhalational anesthetics. With the thiopental infusion, CMRO2 decreased most rapidly during the first 25 minutes.

With halothane and enflurane, CBF was maximal during the period of transition in the EEG from an "awake" to an "anesthetic" pattern. CBF was elevated at all concentrations of isoflurane studied. CBF decreased rapidly during thiopental infusion until the EEG pattern changed from "awake" to "anesthetic" and then more slowly. The results demonstrate that the change in the EEG to an "anesthetic" pattern, which occurs at concentrations well below MAC, is accompanied by an abrupt metabolic depression. It is speculated that these events coincide with the onset of functional depression.

**AB-3090-77**

**Light- and Electron-Microscopic Characteristics of Arterial Smooth Muscle Cell Cultures Subjected to Hypoxia or Car-
Smooth muscle cells from the tunica media of piglet aortae grown under hypoxic conditions undergo the following changes: First, they become modified by partial loss of myofilaments and proliferation of organelles, which are characteristics of young primitive cells. Second, an increase in number of pinocytotic vesicles at and below the cell surface, indicating increased extracellular uptake of material, can be detected. This is followed by accumulation of Oil Red O positive intracytoplasmic granules and vacuoles as well as the subsequent formation of mount-like protrusions. The latter consist of a core of extracellular material and necrotic debris covered with a cap of viable cells. A third feature of the cells subjected to hypoxia is a conspicuous rise in the number of lysosomes. This is considered to be a manifestation of a defense mechanism of the cells to remove undesirable material from cytoplasm. Cells exposed to an atmosphere rich in carbon monoxide exhibit basically the same alterations as those grown under hypoxic conditions; however, formation of mound-like aggregates is less prominent, while the rise in the number of lysosomes is more evident than in the hypoxic cells. The above alterations are similar to changes observed in smooth muscle cells of rabbit with experimental atherosclerosis. It is suggested that wherever the arterial smooth muscle cell is subjected to adverse conditions basically the same mechanism, consisting of dedifferentiation, increased permeability and lysosomal defense reaction, takes place.

AB-3091-77
Active Giant Cell Arteritis With Cerebral Involvement. Findings Following Four Years of Corticosteroid Therapy
Fulton AB, Lee RV, Jampol LM, Keltner JL, Albert DM (Massachusetts Eye and Ear Infirmary, Boston, Massachusetts 02114) — Arch Ophthalmol 94: 2068-2071 (Dec) 1976*

A patient had continued active giant cell arteritis proved on biopsy four years after the initial diagnosis of temporal arteritis. The patient had had erythrocyte sedimentation rates within the normal range and had been receiving corticosteroids without interruption for four years. Central nervous system symptoms occurred three and four years after the initial diagnosis.

AB-3092-77
The Relative Importance of the Major Risk Factors in Atherosclerotic and Other Diseases — Whyte HM (Department of Clinical Science, The John Curtin School of Medical Research, The Australian National University, Canberra, ACT 2601, Australia) — Aust NZ J Med 6: 387-393 (Oct) 1976*

Examination of results from the Framingham study shows considerable disparity between the various clinical conditions which are grouped together as atherosclerotic diseases in their relationship to systolic blood pressure, serum cholesterol level and cigarette smoking. The outstanding features are the dominance of blood pressure in cerebral conditions and the apparent unimportance of smoking in angina pectoris. Analysis of results from the National Cooperative Pooling Project shows that up to 70% of cardiac conditions in this category may be related to these three risk factors with each being of approximately equal importance. Further evidence suggesting that these factors are of greater importance than is commonly realised for non-cardiovascular deaths has been derived from results in the Chicago Peoples Gas Company Study.

Though not conclusive, it is hoped that this analysis adds perspective to considerations of pathogenesis and prevention.

AB-3093-77
Rationalisation of Hospital Services for Stroke Patients — Christie D (Department of Medicine, The Royal Melbourne Hospital, Victoria 3050, Australia) — Aust NZ J Med 6: 407-410 (Oct) 1976*

Over a six month period, the Royal Melbourne Hospital admitted 25% of the stroke patients expected from the north-west region of Melbourne. When the private sector and Mount Royal Hospital were included, a total of 57% of expected demand was met by the hospital services of the region. The natural history of stroke in hospital consisted of an early "active" phase with a high death rate and a high investigation rate. Three weeks after admission this activity had largely subsided and it is suggested that the acute general hospital was no longer providing the most appropriate care. Transfer of patients with stroke three weeks after admission to a lower cost care situation with more appropriate use of staff would enable the Royal Melbourne Hospital to meet the demand for hospitalisation from its region.

AB-3094-77

There appears to be a need to protect our young from an atherogenic way of life. The average male child today has one chance in three of a cardiovascular catastrophe before age 60. Atherosclerosis and the conditions which predispose appear to have their onset in childhood. Correctable precursors of cardiovascular disease have been identified, and their contribution to risk has been estimated not only for adults but for college students as well. An analysis of the combined impact of atherogenic risk factors indicates that they exert greater force early in life than later. Although the optimal time to begin prophylaxis is not established, there is evidence to suggest that measures instituted late in life when lesions are advanced is of only limited value. Prevention of atherosclerosis is best viewed as a family affair since the propensity to disease and contributing factors tend to be shared by family members.

It is also difficult to implement effectively preventive measures which include dietary changes, weight control, ex-
Exercise and restriction of cigarettes for one family member without involving the rest of the family.

Optimal levels of the correctable precursors of cardiovascular disease are not established for children. However, the rise in serum lipids, blood pressure, weight and blood sugar observed in transition from childhood to adult life is not inevitable, or desirable. Paediatricians can alter the appalling cardiovascular mortality statistics by not allowing the process or the habits and conditions which promote it to reach an irreversible stage.

Cardiovascular disease may well begin in childhood with "medical trivia" such as a tendency to obesity, moderate cholesterol and blood pressure elevations, lack of exercise and the cigarette habit. In some respects a heart attack at age 45 can be regarded as a failure of the paediatrician.

Awaiting proof of the efficacy of the indicated prophylactic measures is not acceptable since this will be a long time in coming. We must learn how to correct risk factors effectively in childhood as soon as they appear. We must establish goals based on optimal as distinct from usual levels of risk factors. Paediatricians' resolve about prevention of atherosclerosis in childhood needs to be strengthened and we must develop a sense of urgency about this.

\[ AB-3095-77 \]

**Effect of Propranolol on Platelet Function** — Weksler BB (Department of Medicine, New York Hospital-Cornell Medical Center, New York, New York 10021), Gillick M, Pink J — *Blood* 49: 185–196 (Feb) 1977*

Excessive reactivity of blood platelets may contribute to atherosclerotic vascular disease. Hence drugs which alter platelet function may be protective. Prompted by findings that propranolol therapy normalized hyperactive platelet aggregation in patients with coronary artery disease, we studied propranolol in vitro to assess its action on platelets. At concentrations similar to those achieved in vivo (0.1–1 μM), propranolol raised the thresholds for aggregation of some normal platelets by adenosine diphosphate (ADP). At higher concentrations (10–50 μM), propranolol abolished the second wave of platelet aggregation induced by ADP and epinephrine, and inhibited aggregation induced by collagen, thrombin, and the ionophore A23187. Propranolol blocked the release of ³H-serotonin from platelets, inhibited platelet adhesion to collagen, and interfered with clot retraction. Propranolol blocked ionophore-induced uptake of ⁴⁴Ca by platelets. Inhibition appeared unrelated to beta-adrenergic blockade, as d(+) propranolol (which lacks beta-blocking activity) was equipotent with l(−) propranolol. Moreover, practolol, a beta-blockading drug which is nonlipophilic, did not inhibit platelet function. These studies suggested that propranolol, like local anesthetics, decreased platelet responsiveness by a direct action on the platelet membrane, possibly by interfering with calcium availability. Modulation of platelet function by propranolol may occur at concentrations achieved at usual clinical doses of the drug.

*Authors' abstract*

\[ AB-3096-77 \]


The incidence of myocardial infarction and death from coronary heart disease was studied in defined samples of 45 to 68 year old Japanese men in Japan, Hawaii and California. The incidence rate was lowest in Japan where it was half that observed in Hawaii \((P < 0.01)\). The youngest men in the sample in Japan were at particularly low risk. The incidence among Japanese men in California was nearly 50 percent greater than that of Japanese in Hawaii \((P < 0.05)\). A striking increase in the incidence of myocardial infarction appears to have occurred in the Japanese who migrated to the United States; this increase is more pronounced in California than in Hawaii.

\[ AB-3097-77 \]


Various risk factors were evaluated to explain a significantly greater incidence of coronary heart disease in men of Japanese ancestry resident in Hawaii compared with men resident in Japan. The independent predictors of incidence of coronary heart disease in both Japan and Hawaii were systolic blood pressure, serum cholesterol, relative weight and age. These factors appeared to influence incidence similarly in both areas because in each case the correlation coefficients for Japan and Hawaii did not differ significantly.

The hypothesis that the greater incidence in Hawaii could be attributed to differences in levels of these risk factors was tested with the Walker-Duncan method. The four variable multiple logistic function describing the probability of coronary heart disease in Japan was applied to the cohort characteristics observed in Hawaii. The estimated incidence thus obtained was not significantly different from that actually observed in the men resident in Hawaii. Therefore the increased coronary risk profile in Hawaii compared with Japan can account for the greater incidence of coronary heart disease in the former.

Current cigarette smoking was significantly related to the risk of coronary heart disease in Hawaii but not in Japan. This difference requires further investigation.
Abstracts

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