SREE CHITRA TIRUNAL INSTITUTE
FOR
MEDICAL SCIENCES AND TECHNOLOGY
THIRUVANANTHAPURAM

PROJECT REPORT

NAME	:	DANDU RAVI VARMA
PROGRAMME	:	D.M. NEURORADIOLOGY
MONTH & YEAR OF SUBMISSION	:	NOVEMBER 2003

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TITLE OF THE PROJECT:

" NEUROLOGICAL COMPLICATIONS OF NEUROINTERVENTIONAL PROCEDURES "

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CERTIFICATE

I, Dr. Dandu Ravi Varma hereby declare that I have actually carried out the project **'Neurological Complications of Neurointerventional Procedures'** independently under supervision and guidance in the institution.

Thiruvananthapuram — Signature .....

November 2003 — Dr. Dandu Ravi Varma

Forwarded.

Dr. Dandu Ravi Varma carried out the above-mentioned project in the Department of Radiology, SCTIMST, Thiruvananthapuram.

Signature .....

Prof. A.K. Gupta
Head of the Department of Radiology
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ACKNOWLEDGEMENTS

At the outset, I thank the honorable Director of the institute, Dr. K. Mohandas for permitting me to conduct this study.

My Professor and Head of Department of Radiology, Dr. A.K.Gupta gave me the initial impetus to begin a study on this rarely broached topic. His vision and guidance at every step of the study were of great help in enabling me to complete this study. I am very much indebted to him for his help.

I sincerely thank Dr. T.R.Kapilamoorthy, Associate Professor of Radiology, Dr. C.Kesavadas, Associate Professor of Radiology, Dr.T.Krishnamoorthy and Dr. Bejoy Thomas, Assistant Professors of Radiology for their constant help, constructive criticism and encouragement during the study. I thank Dr.N.K.Bodhey, Ad-hoc consultant in Radiology for taking a lion's share of the work during our postings together, allowing me to work on this study.

I also sincerely thank my colleagues Dr.Sukalyan Purkayastha, Dr. Hemant Sonwalkar, Dr. Jayadevan, Dr.Rajesh Kannan and Dr.Vijaichandran for their help and constant support in carrying out the study.

I am grateful to our Radiology technologists including Mr.Hari Kumar, Mrs.Sharifa Beevi, Alex Jose, Johnson, Shitha, Seena, Sheeba, Shibu, Charlie and Biju for their whole hearted support and help to conduct this study. My special thanks to Cathlab nursing staff for their cooperation during the procedures.

I acknowledge the constant support, encouragement and love from my parents, wife and child throughout the residency programme.

Ravi Varma

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ABBREVIATIONS

ASITN	American Society for Interventional Therapeutic Neuroradiology
AVF	Arterio-Venous Fistula
AVM	Arterio-Venous Malformation
CCA	Common Carotid Artery
CCF	Carotico-Cavernous Fistula
CT	Computed Tomography
DWI	Diffusion Weighted Imaging
ECA	External Carotid Artery
GDC	Guglielmi Detachable Coils
ICA	Internal Carotid Artery
ICU	Intensive Care Unit
JRC	Judkin's Right Coronary catheter
MCA	Middle Cerebral Artery
MRI	Magnetic Resonance Imaging
n-BCA	n – Butyl Cyanoacrylate
PCA	Posterior Cerebral Artery
PVA	Polyvinyl alcohol
SAH	Subarachnoid Hemorrhage
TCD	Transcranial Doppler
VA	Vertebral Artery

Neurological Complications of Neurointerventional Procedures

1. INTRODUCTION

INTRODUCTION

Over the years, techniques in interventional neuroradiology have evolved from being procedures that were dangerous, often ineffective, and mostly considered "experimental"; to those that are safe, effective, and in certain conditions, the "treatment of choice". The first descriptions of catheterization of intracranial arteries using flow-directed balloon catheters by Kerber [1] and of liquid embolic agents by Berenstein [2] marked the beginning of modern interventional neuroradiology. The "Decade of the Brain" (1990-1999) saw a rapid evolution of the specialty, with the introduction of several new tools for clinical use - such as over-the-wire microcatheters, Guglielmi detachable coils, better detachable and nondetachable balloons, newer embolic agents and intra and extracranial vascular stents. Improvements in imaging, catheters and other devices, pharmaceuticals, advances in the understanding of natural history and pathophysiology of cerebrovascular diseases, and establishment of formal training programs in interventional neuroradiology have been largely responsible for the improvement in procedural safety and precision.[3]

With development of newer hardware and techniques, a wider range of neurological illnesses began to be treated by the interventional neuroradiologist, with an increase in the complexity of these procedures. This resulted in an increase in the potential to produce procedure related complications in patients undergoing neuro-interventions.

"**Complication**" has been defined by the Stedman's medical dictionary as " ***A morbid process or event occurring during a disease that is not an essential part of the disease, although it may result from it or from independent causes*** ". Although attempts should be made to achieve perfect outcomes (ie. 100% success, 0% complications), in practice, most neuro-interventional procedures fall short of this ideal to a variable extent.

Procedure related complications could occur at the site of intervention, at the site of vascular access or as systemic complications due to the injected contrast media and embolic agents. They can be broadly classified as neurological and non-neurological complications. Most neurological complications of neuro-interventional procedures are sudden and occur without warning. The appearance of fresh neurological deficit is often masked by the general anesthesia under which the procedure is usually performed. Considering the high sensitivity of the neural tissues to ischemic and other procedure related insults, it becomes essential for the neuroradiologist to avoid situations which may lead to complications, to recognize the occurrence of the event as early as possible and to institute corrective measures to limit and if possible, reverse the damage caused by it. This requires an extensive knowledge about the factors that are associated with an increased risk of complications, about the patho-physiological mechanisms operating during such events, therapeutic options available and about outcome of such salvage techniques.

Surprisingly, there has been very little information in literature dedicated to the incidence, risk factors and optimal strategies for management of complications, and most of what is available is conflicting. Differences in local referral patterns, management protocols and accuracy of follow up are largely responsible for the variability of results between reports from different institutions. In the absence of well-defined guidelines, each institution performing neuro-interventional procedures should carry out periodic review of its results and modify its techniques. This periodic audit of neuro-interventional procedures would ensure that attempts are made to achieve the best clinical results that are possible for that institution.

This study aims to review the various neuro-interventional procedures carried out at the Department of Radiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, during a 30-month period, in order to systematically analyze the complications.

2.AIMS & OBJECTIVES

AIMS OF THE STUDY

1. To describe the spectrum of neurological complications that occurred during and after the neuro-interventional procedures, those were carried out in the Department of Radiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology over a period of 30 months.

2. To critically analyze the roles of clinical presentation, angio-architecture of the lesion, details of the procedure and management protocol in relation to the occurrence and severity of complications.

3. To analyze the natural history of these adverse events and functional outcome of the procedure and various management and rescue strategies.

4. To suggest the changes in the current management protocols that would minimize the occurrence of procedure related complications.

3. REVIEW OF LITERATURE

REVIEW OF LITERATURE

One of the first studies on complications of neuro-interventional procedures using modern hardware and techniques was that of Halbach et al [4] in 1991. They encountered vascular perforations during fifteen (1.1%) of over 1500 endovascular procedures that they performed to treat vascular disorders of the brain and spinal cord. They grouped the vascular perforations as occurring due to three probable mechanisms: mechanical perforation of a normal vessel (6 patients), mechanical disruption of a dysplastic vessel or aneurysm (5 patients), and injection of fluids with pressure (4 patients). Treatment of the perforations included immediate reversal of anticoagulants (12 patients) and direct closure of the perforation site with coils (five patients). In addition, closure of the intra-vascular compartment adjacent to the perforation was achieved with coils (6 patients), liquid adhesives (4 patients), balloons (2 patients), or particles (2 patients). In 2 patients, a detachable balloon was placed across the perforation site for several minutes and deflated when no further extravasation was noted. Two patients succumbed to the massive subarachnoid hemorrhage, while two other patients had long-term neurological sequelae - including hydrocephalus and diabetes insipidus. Eleven other patients had a good outcome.The authors stressed that despite the development of better hardware, vascular perforations may occur and that prompt recognition and closure of the perforation results in a good outcome.

Most neurointerventions involve percutaneous catheterization of the craniocerebral vasculature. Since these procedures involve frequent use of guidewires for catheter exchanges, and require placement of large size of guide catheters for a prolonged time,

there is an increased risk of dissection of the target vessels. Cloft and coworkers [5] reported 12 cases of dissections in a total of 3112 angiographic and neurointerventional procedures, of which, 5 occurred during neurointerventional procedures. The causes of dissections included microcatheter advancement (n=2), guidecatheter exchange (n=2) and due to balloon catheter (n=1). The authors reported that dissections complicating neurovascular procedures are uncommon and have a benign clinical course. Review of five recent reports of procedure related dissections revealed a risk of transient neurologic deficit ranging from 0.55% to 2.2% and the risk of permanent neurologic deficit from 0.1% to 0.5% [6,7,8,9,10].

Most reports of complications have come from individual series and case reports. The clinical and procedural protocols were often dissimilar, with the attendant difficulties in comparing between different management strategies. The latter half of the last decade saw the emergence of several societies and associations that sought to standardize the science and practice interventional radiology. The American Society of Interventional and Therapeutic Neuroradiology (ASITN) was founded to further the development and improvement of endovascular neuro-radiological procedures. In an attempt to standardize the practice of interventional and therapeutic neuroradiology, the ASITN has recently designed practice guidelines for neuro-interventional procedures, which were published as a supplement in the American Journal of Neuroradiology [11]. These were developed by consensus opinion of multiple experts in the field of interventional neuroradiology, after thorough review of the relevant literature. Minimum thresholds for indications and technical proficiency and maximum thresholds for complications have been set for each

procedure. The ASITN suggested that when complication rates exceed the maximum threshold, an internal audit must be performed to determine the causes and implement any necessary changes. Furthermore, they cautioned that the development of universally acceptable thresholds is very difficult due to the wide variability of the referral pattern and each such institution has been urged to alter the thresholds as needed.

Neurological Complications of Embolization of Brain AVMs

Arteriovenous malformations of the brain consist of multiple tiny arteriovenous shunts, which are interspersed within the neuroparenchyma. The major endovascular technique used to treat these lesions involves transarterial superselective catheterization of the nidus and embolization using cyanoacrylate glue. However, considering the complex angioarchitecture, dysplastic nature of the vessels involved, and presence of the high flow arteriovenous shunts, these lesions may prove to be one of the most dangerous lesions to treat. The major complications associated with embolization of these lesions include intracerebral hemorrhage, cerebral ischaemia, microcatheter retention, and occlusion / dissection of the feeding artery.

In the largest analysis of its kind, Hartmann et al in 2002 [12]. reported the complications encountered in a prospective 8-year series of brain AVM embolization with independent assessment of the neurological outcome of the procedures. Of the 233 patients treated with 545 endovascular procedures, 200 patients (86%) experienced no change in neurological status after treatment. Thirty-three patients (14%) showed treatment-related neurological deficits. Among these, 5 (2%) had persistent disabling deficits (Rankin score

>2), and 2 (1%) died. None of the morphological characteristics of the AVM predicted treatment complications. Increasing patient age (odds ratio (OR) 1.04), number of embolizations (OR, 1.41), and absence of a pretreatment neurological deficit (OR, 4.55) were associated with new neurological deficits.

The n-BCA trial [13] was a prospective, randomized, multicenter trial that compared the results of brain AVM embolization in 104 patients, using n-BCA and PVA particles. Using a different definition of what constitutes a complication, these investigators found that 27 (50%) of the patients who underwent embolization, using n-BCA had at least one complication. 13% of patients in the n-BCA group and 29% of PVA group had hemorrhagic complications. These resulted in the death of one patient. The rest of the complications in this series were either asymptomatic (catheter adhesion, catheter blockage due to premature polymerization of glue, venous obstruction due to late polymerization, seizures etc.) or were transient and non-disabling.

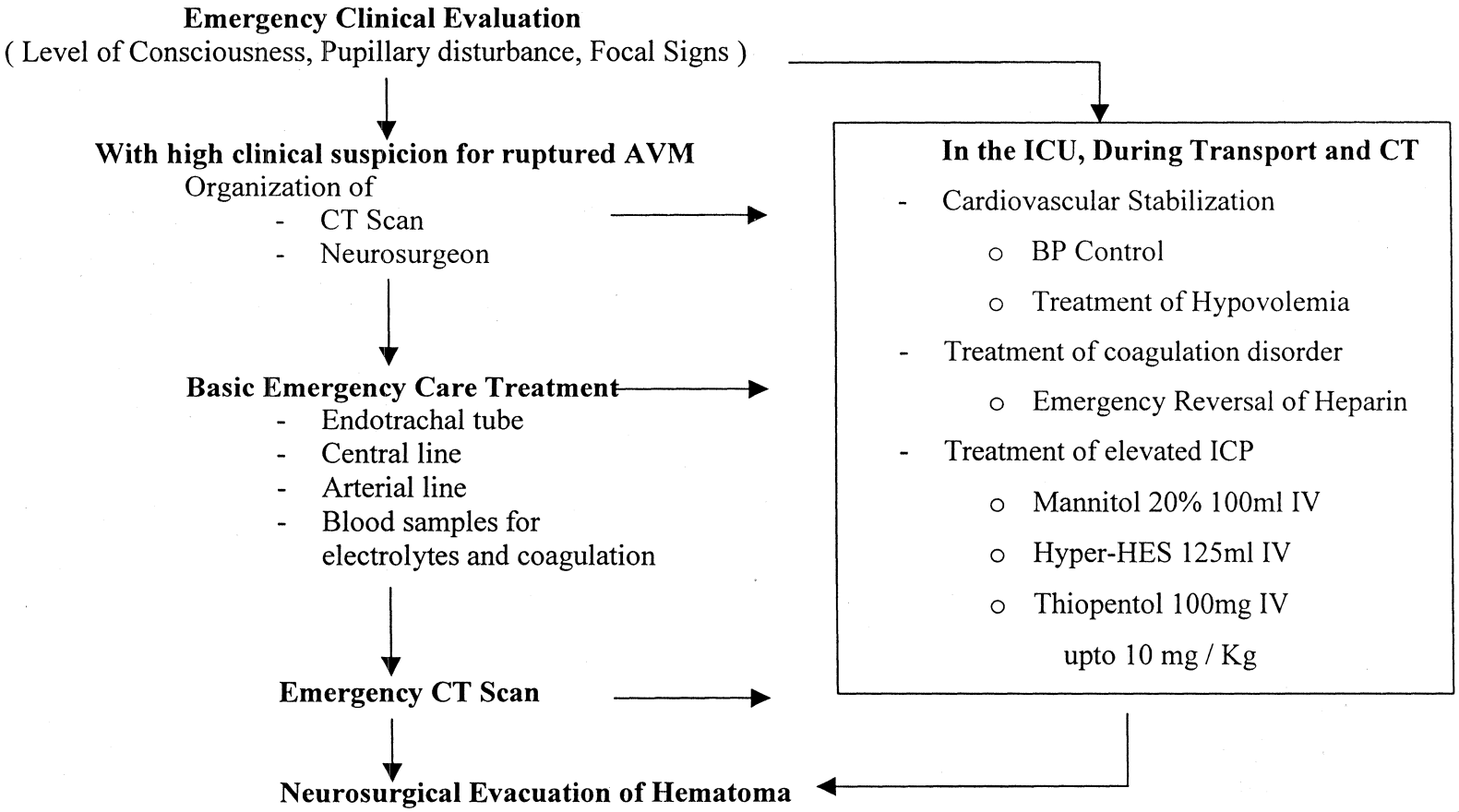
The development of newer microcatheters and the intracranial use of microguidewires have permitted the catheterization of even small caliber cortical arteries. However, wires introduce the potential risk of vessel perforation. In the series of Halbach and coworkers [4], six of the fifteen vascular perforations that occurred during neurointerventions were related to AVMs. The management included reversal of heparinization (6), coil embolization (2), transient balloon inflation across the perforation site (2), glue embolization (2), PVA particle embolization (2). One patient died before any therapy could be instituted, whereas, the rest recovered and were asymptomatic on follow up.

Gluing of the tip of the microcatheter in the nidus of the AVMs is particularly specific to the use of acrylic glue. This complication has been reported to occur in 3.1% of glue injections and rarely has neurological sequelae.[14] Most reports of glued catheters have occurred using isobutyl-2-cyanoacrylate (IBCA), or *n*-butyl cyanoacrylate (NBCA), and are secondary to technical mishaps, including overzealous embolization, too rapid polymerization, and excessive reflux of adhesive around the catheter tip.[15]

Intracerebral hemorrhage is the most dreaded of all the complications of brain AVM embolization. The hemorrhage occurs at arterial pressure with resultant rapid progression of neurological deficits. It has been reported to occur in 3.1 – 11% of the embolization procedures.[16] Considering the poor prognosis of untreated patients, expert ICU management and evacuation of the hematoma is recommended in these patients. Keller et al [16] in 2002 described their experience in managing procedure related bleeds. There were 24 episodes of intracerebral hemorrhage in 1066 procedures on 605 patients. Six patients had small bleeds (< 2.5cm), which presented with headache, without any major neurological deficits. 18 patients had large bleeds. Average Glasgow Coma Scale level at presentation was 4.2. Patients were managed using a special protocol that aimed to minimize the time between onset of symptoms and surgical evacuation, by avoiding the time consuming procedures that are not essential for management. The protocol suggested by the authors is depicted in Table 3.1. They reported 61.5% recovery in patients before 1998, where the average time between bleed and evacuation was 124 minutes. In contrast after 1998, using their protocol, they achieved a good outcome in all of the 5 patients who had procedure associated bleeds during this period.

Table 3.1 Algorithm for Emergency Management of patients with Embolization Induced Bleeding from Brain AVMs

(**Source:** Keller et al. Intensive care management of patients with severe intracerebral haemorrhage after endovascular treatment of brain arteriovenous malformations. Neuroradiology (2002) 44: 513–521



Apart from non-target embolization of normal cerebral vessels, procedure-induced ischemic stroke can also seen with catheter associated thromboembolic events. Ethanol is an extremely potent liquid embolic agent that can be used in selected cases of cerebral arteriovenous malformation. Ethanol causes both, a cytotoxic reaction in the neuroparenchyma as well as sclerosis of the arterial walls resulting in thrombosis[17]. Reflux of ethanol into normal arterial branches can result in intense spasm, resulting in infarction of the parenchyma supplied by that vessel.

The ASITN standards of practice [18] have set a threshold of 10% for major and minor permanent neurological deficits, 20% for transient neurological deficits and 5% for retained microcatheter fragments.

Neurological Complications of Endovascular Therapy for Aneuryms

Over the years, endovascular treatment of intracranial aneurysms has evolved from proximal balloon occlusion and intra-aneurysmal deposition of balloons, to packing the aneurysm with detachable platinum coils. Recent development of newer generation of coils and innovative techniques such as balloon remodeling technique and stent assisted coiling have increased the complexity of these procedures and thus worsened the risk of complications. Familiarity with the equipment and technique goes a long way in improving the safety of this mode of therapy.

Recently, Murayama et al [19] reported their 11-year experience in coiling 916 intracranial aneurysms in 818 patients. Technical complications occurred during the GDC placement

in 69 patients (8.4%). Nineteen patients (2.3%) had an intra-procedural aneurysm perforation; 20 patients (2.4%) had embolic events and 16 patients (2%) had parent artery occlusion during the procedure. Less frequent complications were - arterial dissection / spasm (0.7%), coil migration (0.5%), coil rupture (0.4%), and new mass effect (0.1%). The authors reported that complications occurred more frequently (11.3%) during the initial 5 years than the latter 6 years (7.3%)of the study period. Another recent report found that the rate of all complications in GDC coiling of unruptured aneurysms decreased from 53% in their initial cases to less than 10% as the experience of the treating physician increased.[20]

The most dreaded complication of coil embolization of intracranial aneurysms is intraprocedural rupture of the aneurysm. Diagnostic angiography itself is associated with a higher risk of re-rupture (1.4%) especially if performed within 6 hours after the initial SAH, and in the presence of worse levels of consciousness [21]. Rates of aneurysm perforation during coiling vary from 2.0% to 4.4% [22, 23, 24, 25]. A meta-analysis [26] of 14 major studies, revealed 51 aneurysmal ruptures in 2030 treated aneurysms (2.51%). Of these patients, 31 had a good outcome and 20 died (mortality rate 0.99%). Among the 1116 aneurysms that presented initially with SAH, 38 ruptured during the procedure, while only 1 of 458 aneurysms without previous SAH ruptured. This difference was significant ($p < 0.00022$), thus identifying previous SAH as a risk factor for procedure-related rupture. Cloft et al [27] in 2002, presented a similar meta-analysis of 17 published reports of perforation of the aneurysm during therapy. They reported an incidence of perforation in 2.7% of all aneurysms coiled. The rate was higher (4.1%) in ruptured

aneurysms and lower (0.7%) in unruptured aneurysms. For ruptured aneurysms, intraprocedural aneurysm perforation was associated with a 33% risk of death and a 5% risk of disability. For unruptured aneurysms, a 14% risk of death and a 14% risk of disability were noted. The morbidity and mortality rates with perforations caused by the coil (39%) and rates for those caused by the microcatheter (33%) were similar. The morbidity and mortality rates for microguidewire perforations were lower (0%) than the rates for coils and microcatheters, but this difference was not statistically significant (P = .16).

The commonest causes of intraprocedural rupture of aneurysms include - perforation due to inadvertent advancement of microcatheter while microguidewire was being withdrawn, due to inadvertent advancement of microguidewire or during placement of coils. Recently ruptured small aneurysms (smaller than 4mm), posterior fossa aneurysms, associated ventricular dilatation, massive cisternal hemorrhage have been associated with a high risk of intraprocedural rupture[23]. The fragility of small aneurysms was reported to be due to the larger surface area of the initial rupture for small aneurysms than for large aneurysms, or due to higher shape memory of small coils that measure 2–3 mm in diameter. Except for the 2-mm aneurysms, it was never the first coil that perforated the sac. Visual control during the placement of the first coil is not hampered by the presence of a coil mesh. Subsequently, as the coil mesh becomes denser, rupture may occur when a loop of the coil is forced between it and the aneurysm wall[26].

Coiling of intracranial aneurysms has a risk of thromboembolic complications, which constitute the commonest complications of this technique. Apart from the complexity and the multiple catheters involved in these procedures, the thrombogenecity of the platinum coils contributes to the risk of thromboembolic complications. The incidence of such complications has been reported to range from 2.5% to 28% [24,28]. There are many possible sources for embolic events during GDC treatment of cerebral aneurysms: friable plaques, iatrogenic dissection in the parent vessels, air bubbles, thrombus or fresh clots within aneurysms and catheters [29]. Wide necked aneurysms, protrusion of coil loops into the parent artery and use of balloon remodeling technique are risk factors for the occurrence of thromboembolic complications [30].

Monitoring with transcranial Doppler study has shown microembolic signals in 71% of patients who had ischemic complications and 21% of patients who did not have complications following coiling of aneurysms [31]. The authors emphasized the importance of TCD in decisions regarding post-procedural management with heparin and aspirin. In a study that used DWI to evaluate the thromboembolic events after GDC coiling, Soeda et al [29], have reported that hyperintense lesions occurred in DWI in 61% of their cases; 40% of which had neurological deficits. Ninety four percent of these patients recovered completely while one patient experienced permanent deficits. Thus, the absence of clinically identifiable neurological deficits does not exclude a thromboembolic event. These silent infarcts may be associated with a cognitive impairment, that would be difficult to detect unless a detailed neuropsychological evaluation is performed.

Unraveling and fracturing of GDCs occur with forceful manipulation, damaging the device and predisposing it to fracture[32]. They are estimated to occur in less than 2% of GDC embolization procedures [33]. They usually occur in three settings: 1) resistance to coil advancement or withdrawal while placing the final coils in an attempt to tightly pack an aneurysm, 2) folding over of a 0.010-inch coil during attempted deployment through a 0.018-inch microcatheter, and 3) in attempts to withdraw the introducing wire in instances of false coil detachment [34]

Phatouros et al [35], in 1999 described four cases in which migration of part of a coil into the parent artery occurred after completion of the procedure. They proposed that origin of an arterial branch from the aneurysm, presence of thrombus within the aneurysm sac, tendency of coils to revert back to their "memorized" shape, may predispose to post-procedural coil migration – especially in wide necked aneurysms, or when inappropriately sized coils are used.

The ASITN thresholds[36] for rates of complication during endovascular treatment of intracranial aneurysms are depicted in Table 3.2

Table 3.2 Complications Threshold in Endovascular Treatment of Intracranial Aneurysms
(Adapted from: ASITN Data, AJNR Sep 2001)

		Unruptured Aneurysms		Ruptured Aneurysms	
		Parent artery occlusion	Parent artery sparing	Parent artery occlusion	Parent artery sparing
Death in Hospital	Grade I	1	2	5	5
	Grade II			15	15
	Grade III			40	40
	Grade IV			60	60
	Grade V			75	75
All Neurological complications		10	8	8	8
Permanent Neurological complications		5	3	3	3
Inadvertent vessel occlusion		N/A	8	N/A	8
Vessel perforation		0.5	2	0.5	2
Aneurysm rupture during procedure		1	1	5	5
Aneurysm rupture after procedure		2	2	5	5

Neurological Complications of Embolization of Craniofacial Vascular Malformations and Head and Neck Tumors

Embolization of craniofacial vascular malformations and tumors is chiefly performed in the preoperative setting for devascularization of the lesion. The array of techniques used includes transarterial embolization using gelfoam and PVA particles, transarterial embolization using n-BCA and direct puncture embolization using n-BCA. The chief hazards of embolization in the external carotid territory lie in the supply to cranial nerves and in the presence of embryonic communications and collateral vessels to the intracranial compartment [37]. Every attempt must be made to assess the presence of such "dangerous anastomoses" before injection of any embolic agent. The caliber of these collaterals may be too small for adequate visualization on digital subtraction angiogram. Similarly changes in flow dynamics in the territory can result in the opening up of new channels during the course of embolization. The arterial territories with potential supply to neural structures and location of dangerous anastomoses are listed in Table 3.3

Most cases of cranial nerve palsies after embolization, are associated with the use of liquid embolic agents and small sized particles (<150 μm). Tumor embolization with ethanol may result in post embolization swelling. Embolization of intracranial tumors like meningiomas may produce headache due to increased mass effect or due to meningeal irritation. Preoperative steroids may help in minimizing this complication. Embolization of certain tumors like paragangliomas may be result in life-threatening vasomotor attack, due to catecholamine secretion.

The ASITN thresholds [38] for rates of complication during endovascular treatment craniofacial vascular malformations and head and neck tumors are depicted in Table 3.4

Table 3.3: Dangerous Vessels During Craniofacial Embolization

- Persistent primitive anastomoses and abnormal pattern of vascular arrangements
- Dangerous External Carotid – Internal carotid / Vertebral anastomoses
- Vessels supplying vital structures

Persistent primitive anastomoses and Abnormal vascular arrangements

- ***Carotid - Basilar Anastomoses***
    - Persistent Proatlantal Intersegmental Artery
    - Persistent Hypoglossal Artery
    - Persistent Otic Artery
    - Persistent Trigeminal Artery
- ***Abnormal Vascular Arrangements***
    - Origin of Ophthalmic artery from Middle meningeal artery - CN II
    - Origin of Ophthalmic artery from Accessory meningeal artery - CN II
    - Accessory meningeal artery dominance in cavernous sinus region – CN III, IV, V, VI, VII
    - Origin of Anterior Inferior Cerebellar artery from Accessory meningeal artery – Cerebellum
    - Origin of Posterior inferior cerebellar artery from Ascending pharyngeal artery – Cerebellum

Vessels Supplying Vital Structures

- ***Occipital Artery***
    - Direct supply to C1 and C2 nerves
    - Stylomastoid br. → Supply to CN VII
- ***Ascending Pharyngeal Artery***
    - Musculospinal artery → Supply to CN XII, C3, C4 nerves
    - Hypoglossal br. → Supply to CN XII
    - Jugular br. → Supply to CN IX, X, XI
    - Inferior tympanic br. → Supply to Jacobson's Nerve
    - Superior pharyngeal br. → Supply to Gasserian gang, Sympathetic plexus
    - Lateral spinal br. → Supply to cervical cord, medulla, C1 and C2 roots

- ***Posterior Auricular Artery***
    - Stylomastoid artery → Supply to CN VII
- ***Internal Maxillary Artery***
    - Middle Meningeal Artery → Supply to CN V, Gasserian ganglion
    - Recurrent br. of Middle Meningeal Artery → Supply to Ophthalmic division of trigeminal nerve
    - Accessory meningeal Artery → Supply to maxillary, mandibular and motor divisions of trigeminal nerve
    - Anterior tympanic artery → Supply to chorda tympani nerve
    - Infraorbital artery → Supply to Optic nerve
    - Artery to foramen Rotundum → Supply to mandibular divisions of trigeminal nerve
    - Vidian artery → Supply to Vidian nerve
- ***Posterior Cervical Artery*** → Supply to C2, C3 and C4 nerves

Dangerous Anastomoses

- External Carotid Artery
    - C4 collateral artery → • ← Cervical ICA
- Occipital Artery
    - C1 collateral → • ← Cervical ICA
    - C1 collateral → • ← Cervical ICA
- Ascending Pharyngeal Artery
    - C3 collateral artery → • ← Cervical ICA
    - Superior pharyngeal br. → • ← Petrous ICA (at skull base)
    - Inferior temporal br. → • ← Caroticotympanic artery ← Petrous ICA
    - Carotid br. → • ← Meningohypophyseal trunk ← Cavernous ICA
    - Clival br. → • ← Petrous ICA
      → • ← Meningohypophyseal trunk ← Cavernous ICA
      → • ← Meningeal br. ← Vertebral artery
- Posterior Auricular Artery
    - Stylomastoid br. → • ← Petrous ICA (at skull base)

- Superficial Temporal Artery
    - Medial frontal br. → • ← Supratrochlear br. ← Ophthlamic artery
      → • ← Dorsonasal br. ← Ophthalmic artery
    - Lateral frontal br. → • ← Supraorbital artery ← Ophthalmic artery
- Facial Artery
    - Angular artery → • ← Nasal br. ← Ophthalmic artery
- Internal Maxillary Artery
    - Middle and Accessory meningeal arteries
      → • ← Meningeal br. ← Posterior ethmoidal br. ← Ophthalmic artery
      → • ← Meningeal br. ← Lacrimal & Palpebral br. ← Ophthalmic artery
      → • ← Meningeal br. ← Inferolateral trunk ← Cavernous ICA
      → • ← Meningeal br. ← Vertebral artery
      → • ← Meningeal br.(Davidoff & Schechter) ← Posterior cerebral artery
      → • ← Pial collaterals between meningeal arteries and cortical arteries
- Deep Temporal Artery
      → • ← Lacrimal br. ← Ophthalmic artery
- Infraorbital Artery
      → • ← Lacrimal br. ← Ophthalmic artery
      → • ← Palpebral br. ← Ophthalmic artery
      → • ← Muscular br. ← Ophthalmic artery
- Sphenopalatine Artery
      → • ← Anterior ethmoidal br. ← Ophthalmic artery
      → • ← Posterior ethmoidal br. ← Ophthalmic artery
- Artery of Foramen Rotundum → • ← Inferolateral trunk ← Cavernous ICA
- Vidian Artery → • ← Mandibular br. ← Petrous ICA
- Thyrocervical trunk → Ascending cervical artery → • ← C2, C3 & C4 root anastomotic br. ← Vertebral artery
- Costocervical trunk → Deep cervical artery → • ← C2, C3 & C4 root anastomotic br. ← Vertebral artery

Table 3.4: Complication thresholds for Embolization of Craniofacial Vascular Malformations and Head and Neck Tumors (Adapted from: ASITN Data, AJNR Sep 2001)

Indicator	Craniofacial AVM Threshold (%)	Head and Neck Tumors Threshold (%)
Tissue Necrosis / Ulceration	-	>1
Nerve Palsy		
Transient	> 2	> 2
Permanent	> 1	> 1
Neurologic Deficit		
Major Permanent	>1	>1
Minor Permanent	>2	>2
Transient	>3	-
Death	0	> 0

Neurological Complications of Embolization of Extracerebral Arteriovenous Fistulae

Extracerebral arterio-venous fistulae such are carotico-cavernous fistulae and vertebral-venous fistulae are characterized by the presence of arteriovenous communications, resulting in symptoms due to elevated venous pressure. Depending on the location and angioarchitecture of these lesions, a wide array of techniques such as transarterial embolization by particles, transarterial embolization with detachable balloons and coils or transvenous embolization using coils are utilized in treating these patients.

Embolic episodes may complicate transarterial embolization procedures and can be prevented by meticuolous attention to periprocedural anticoagulation. Acute venous outflow obstruction caused by embolization may result in elevation of intraocular pressure and may cause deterioration of vision. Normal perfusion pressure breakthrough may complicate sudden closure of long standing high flow arteriovenous shunts. In report of 185 carotid and vertebral fistulas treated by Halbach and coworkers[39], 5 patients developed neurologic deficits after abrupt closure of their fistulas. All five patients had large, long-standing fistulas, ranging in duration from 9 to 32 years. The authors concluded that patients with carotid or vertebral fistulas of long duration, particularly those with cerebral steal symptoms, are at risk to develop neurologic deficits related to perfusion breakthrough if their fistulas are abruptly closed. The ASITN safety thresholds for embolization of indirect and direct carotico-cavernous fistulae are depicted in Table 3.5

Table 3.5: Complication thresholds for Embolization of Carotico-Cavernous Fistulae

(Adapted from: ASITN Data, AJNR Sep 2001)

Indicator	**Indirect CCF Threshold (%)**	**Direct CCF Threshold (%)**
Death	**0**	**1**
Major Complication (permanent deficit)	**1**	**3**
Minor Complication (permanent deficit)	**2**	**5**
Transient (>24 hour) deficit	**5**	**5**

Neurological Complications of Carotid Angioplasty and Stenting

The major complications associated with carotid stenting include embolism, arterial spasm and flow limiting arterial dissection, balloon rupture, failure of stent deployment, stent migration, post deployment stent compression, cerebral hyperperfusion syndrome, external carotid artery occlusion and rarely, vascular perforation and rupture.

Embolic events are by far the most frequent complications encountered in cerebrovascular recanalization procedures. Embolic events may be related to injection or release of clot, fibrin, air, or plaque debris. Several protection devices have been designed, to prevent the release of such debris into the intracranial circulation. However, the clinical effectiveness and safety have to be proved in large series before these can be introduced into routine clinical practice. Vasospasm can be due to mechanical irritation to the normal, nondiseased carotid artery segments that is produced by a guidewire, a stiff dilatation balloon, stent delivery device or protection devices. Spasm can also occur due to mechanical irritation to the vessel when the end of the stent is located in a vessel curve. Careful manipulation of these devices and placement of the stent so as to straighten the arterial curves may help prevent this complication. Dissection is not as much a problem with carotid stenting as it was with carotid angioplasty. The complications related to stent deployment and malpositions are on the wane, with development of newer designs of stents and delivery systems that permit more accurate and reliable deployment of the stent.

Intracerebral hemorrhage is a dreaded, but fortunately rare complication of carotid stenting. Three main mechanisms of hemorrhage have been described following carotid stenting.[40] Embolic insults can cause microvascular damage, with leakage of small amounts of blood into the parenchyma. This is usually clinically insignificant. Thrombolysis in the presence of an infarction can cause bleeding from large vessels, which can be devastating. The third mechanism is due to hyperperfusion syndrome, which has been described to occur both with carotid endarterectomy as well as carotid stenting. Severe carotid stenosis causes maximal dilatation of the capillaries in its distribution, with impairment of autoregulatory mechanisms. After carotid revascularisation, the high pressure in these unprotected capillaries disrupts the capillary endothelium, with breakdown of blood-brain barrier and formation of edema. Presence of any procedure related ischemic event, hypertension and concurrent administration of anticoagulants precipitates cerebral hemorrhage [41]

Wholey et al [40] have described their experience in managing the neurological complications in 450 patients who underwent carotid artery stenting. In their series, there were 14 (3.1%) TIAs, 10 (2.2%) minor strokes and 3 (0.7%) major strokes. There were 6 (1.3%) deaths, of which 4 (0.9%) were secondary to neurological events. More than a quarter of the TIAs and about 30% of the minor strokes occurred between the 2nd and 14th day after stenting. The authors described these neurological events to represent delayed embolic phenomena, related to dislodged plaque and / or thrombus from between the stent struts. The importance of this phenomenon lies in the possibility of occurrence of embolic episodes even after a successful stenting procedure with the help of protection devices.

In one of the largest series on carotid stenting, Vitek et al [42] reported their experience of stenting 451 vessels in 390 patients with >50% stenosis in symptomatic patients and > 70% stenosis in asymptomatic patients. The overall combined stroke and death rate at 30 days was 7.9%. Two patients died of neurological causes – due to rupture of the carotid artery during post dilation. There were a total of four (0.9%) major strokes - related to the acute stent thrombosis, procedure related embolism and two other due to cardiac causes. There were 25 (5.5%) minor non-disabling strokes of which 14 patients achieved complete recovery.

Neurological Complications of Percutaneous Vertebroplasty

The complication rate associated with percutaneous vertebroplasty is 1 to 3 percent for treatment of osteoporotic fractures and 7 to 10 percent for treatment of malignant neoplasms [43]. The majority of complications are transient and minor, and they include the following: hemorrhage from the site of puncture, rib or vertebral posterior element fracture, worsening of pain for several hours following the procedure caused by the heat generated by cement polymerization, nerve root irritation, cement embolization to the lungs via the paravertebral venous plexus, pneumothorax for thoracic lesions[44] and infection.

The biggest concern is foraminal or epidural cement leakage, which can lead to damage to the spinal cord and nerve roots and subsequent neurological deficit or pain. Leakage of the cement into the disc space appears to be inconsequential and in general leakage has been reported in up to 65% of the cases [45]. However, symptomatic leakage causing neurological deficits is fortunately rare.

The ASITN has not formulated any specific guidelines regarding vertebroplasty. The Society of Interventional Radiology has recently published Quality Improvement Guidelines for Percutaneous Vertebroplasty [46].The document identifies specific indications and contraindications for percutaneous vertebroplasty and described major complication rates of 1% and 5% as thresholds for complications in vertebroplasty of osteoporotic and neoplastic vertebrae respectively. It also points out that published rates for individual types of complications are highly dependent on patient selection and are based on series comprising several hundred patients, which is a volume larger than most individual practitioners are likely to treat.

Neurological Complications of Embolization of Vertebral lesions

Embolization of hypervascular tumors of the spinal axis is usually performed to decrease the surgical morbidity by reducing the blood loss, shortening the operative time, increasing the chance of complete resection, relieving intractable pain, reducing expected tumor recurrence, decreasing systemic toxicity of intra-arterial chemotherapy, stabilization of function, and sole treatment for a patient who is at poor risk for surgical therapy, radiation therapy, and/or chemotherapy. The hypervascular tumor types for which embolization may be indicated include benign tumors (hemangiomas, aneurysmal bone cysts, osteoblastomas, chondromas), malignant tumors (giant cell tumors, chordomas, osteogenic sarcomas, chondrosarcomas, hemangiopericytomas, lymphomas, multiple myelomas, plasmacytomas), metastatic tumors (renal cell carcinomas, thyroid carcinomas, other hypervascular metastases), and spinal cord tumors (hemangioblastomas).

The major complications associated with embolization in the spinal axis relate to non-target embolization of the vessels supplying the neural axis. This is more common towards the end of the embolization procedure, obliteration of the vascular bed results in significant reflux into the parent arteries. Increase in size of the mass lesion with resultant cord compression has been described with the use of absolute alcohol as an embolic agent.[47] The ASITN standards of practice [48] permit a maximal complication threshold of 5% for major neurological deficits, 10% for minor neurological deficits and 20% for transient neurological deficits in the presence of epidural component.

Neurological Complications of Embolization of Spinal Arteriovenous Malformations

The serious complications of spinal angiography and embolization are primarily related to ischemic injury to the spinal cord. Spinal angiography itself is associated with significant complications rates. In a retrospective analysis of spinal arteriography in 96 consecutive patients, Forbes et al [49] found transient neurologic deficits in 2.2% (two patients recovering within 24 hours and 1 patient within 1 week). Since the angio-architecture and techniques of treatment vary, the rate of complications associated with embolization of spinal dural arteriovenous fistulae, perimedullary fistulae and spinal cord arteriovenous malformations differs significantly.

Rodesch and coworkers [50] reported the results of embolization in 69 patients with spinal vascular malformations. These complications occurred rapidly (within the first 24 hours after the procedure). Transitory mild deficits were seen in 10 patients (14%), all of which resolved either spontaneously or rapidly with the administration of corticosteroids or anticoagulation. These were thought to be due to a local inflammatory reaction associated

with the glue, or due to venous congestion worsened by embolization. Nine patients (13%) had permanent deficits that occurred as a result of embolization. Complications causing severe neurological deficits occurred in 3 patients (4%). All of these complications occurred after embolization performed through the anterior spinal artery.

The ASITN standards of practice [48] permit a maximal major and minor complication rate of 2% and 5% for spinal dural arteriovenous fistulae, 5% and 10% for perimedullary fistulae and cord arteriovenous malformations.

Neurological Complications of Intraarterial Thrombolysis

The major neurological complication associated with intra-arterial thrombolysis is hemorrhagic transformation of the infarct. Aggregate data indicate an 8.3% risk of symptomatic brain hemorrhage with IA thrombolysis in the carotid territory and a 6.5% risk in the vertebrobasilar territory[51]. There is no evidence that the rate of symptomatic brain hemorrhage is lower with IA thrombolysis than with IV thrombolysis, but direct comparisons are difficult [52]. Hemorrhagic transformation has been reported to occur more commonly with t-PA (50%) than with urokinase(32%) [53]. Although brain hemorrhage complicating thrombolysis for acute stroke likely reflects reperfusion of necrotic tissue, no direct relation between recanalization and hemorrhage risk has been reported [54]. Early CT changes and severity of the initial neurologic deficit, are indicators of the extent of ischemic damage, and are the best predictors of the risk of hemorrhagic transformation [53, 55]. The dose of the thrombolytic agent [56], blood pressure[57,58], advanced age [58], prior head trauma [59], blood glucose >200 mg/dL [53,60] and longer time to recanalization [53] have been associated with hemorrhage after thrombolysis

Neurological Complications of Carotid Temporary Balloon Occlusion Test

Mathis et al [61] published the largest series of test balloon occlusion in 1995. They reported 16 (3.2%) complications among 500 patients. Among these, 8 patients were asymptomatic, while 6 (1.2%) had transient and 2 (0.4%) had permanent neurological deficits. On the other hand, complication rates as high as 8.3% have also been reported [62]. This variation in the rate of complications reflects the wide variations in the technique in practice. The ASITN standards of practice [63] permit a maximal rate of asymptomatic dissection in 4%, transient neurological deficit (5%) and permanent neurological deficit (5%).

Neurological Complications of Miscellaneous Neuro-interventions

Intracranial thrombolysis for cerebral venous sinus thrombosis

Intracranial hemorrhage is a feared and limiting complication of thrombolytic therapy. A review [64,65,66] of 45 cases of cerebral venous sinus thrombus treated with local thrombolysis (urokinase, n=33; tissue plasminogen activator, n=12) reveals 39 (87%) with a good recovery, 1 death, and 3 patients with worsening of intracranial hemorrhage without a fatal outcome. Four patients developed new hemorrhages (retroperitoneal bleed, n=2; intracerebral hemorrhage, n=1; subdural hemorrhage, n=1). Horowitz et al [65] described 13 patients, 4 of whom had pretreatment brain hemorrhages. No worsening was seen after thrombolysis. Frey et al [66] recently described 12 patients treated with recombinant tissue plasminogen activator (tPA), 7 of whom had pretreatment hemorrhages. Two of these patients showed nonfatal worsening of hemorrhage after thrombolysis, most likely due to a more rapid and more potent effect of tPA and the concomitant use of heparinization during thrombolysis.

Intracranial stenting for aneurysms / stenotic lesions

Lylyk etal [67] in 2002, reported their experience of stenting in 36 patients with intracranial stenotic lesions. In two patients, negotiation of the stent to the target was not possible. Unintended stent dislodgement during navigation occurred in two other patients. Friction induced by tracking of the stent through tortuous vessels could have induced stent dislodgement from the delivery catheter. Displacement of the stent after deployment, due to balloon deflation and withdrawl, occurred in three patients. The use of a stent with an inadequate diameter or underexpansion of the stent for fear of rupture could have accounted for this event. Three patients had sudden hypertension related to balloon inflation, and one had associated bradycardia. Two embolic events were successfully treated with selective urokinase infusion. The procedural morbidity rate was 12%. Neurological deterioration occurred in 2 (6%) cases, due to transient ischemic attacks.

Nakatsuka and coworkers[68] suggested that intracranial angioplasty is best performed with less invasive techniques, such as lowering inflation pressure (<3 atm), inflating the balloon catheter slowly, dilating fewer times (once or twice), and using a smaller balloon catheter (2.0 mm). They suggested that these techniques could minimize the intimal damage and prevent occlusion of the perforating artery and excessive dissection. The purpose of angioplasty should be to provide sufficient perfusion to reduce ischemic symptoms, not to achieve an angiographic cure, which is often associated with an unacceptable complication rate.

Mechanical and Pharmacological treatment of Vasospasm

Undesirable outcomes and effects are known to occur in association with both mechanical and chemical vasolytic therapy. Technical complications may be classified as minor or severe and may result in transient or permanent neurologic sequelae. The potential complications and their rates vary between balloon angioplasty and papaverine infusion. Treatment of a vessel harboring an unsecured, ruptured aneurysm may lead to aneurysm rupture. Treatment of a large vascular territory that is obviously infarcted is avoided if possible [69].

Pupillary dilation mimicking neurologic decompensation may occur with proximal ICA infusion below the ophthalmic artery [70]. Papaverine infusion below the posterior-inferior communicating artery may result in respiratory depression and is not recommended unless ventilatory support is available [71]. Similarly, reports of papaverine infusion causing increased intracranial pressure have been noted, and monitoring of intracranial pressure devices, when available, is recommended [72].

4. MATERIALS & METHODS

MATERIALS AND METHODS

For the purpose of this study, a "***neuro-interventional procedure***" was defined as any procedure performed under imaging guidance, that aims at evaluating and treating vascular and nonvascular abnormalities of the brain, spinal cord and head and neck; or has a reasonable potential to produce neurological deficits, either due to its proximity or due to common vascular supply with the neural axis. A "***procedure related neurological complication***" was defined as any untoward incident that occurs during the interventional procedure or within one month of the procedure, and which causes death, or major / minor neurological deficits, or prolongs the post-procedure stay of the patient in the hospital. The choice of one month as the limit for defining the post-procedural period is based on the assumption that adverse events caused due to procedure related alterations in neurovascular hemodynamics would manifest during the said period.

All patients who underwent neuro-interventional procedures at the Department of Radiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, during the period between 1st January 2001 and 30th June 2003 formed the study population. Patients who underwent multiple sittings of therapy, one or more of which were performed during the study period were also included in the study. Patients in whom, interventional procedures could not be performed due to unfavorable angioarchitecture, were also included in the study, as the risk of complication in this group of patients is high due to the prolongation of the procedure. Patients in whom death / deficits occurred in the post procedural period due to unrelated preexisting conditions (such as myocardial infarction in a known case of coronary artery disease) were excluded from the study.

All the procedures were performed under fluoroscopic guidance on a Advantx Digital Subtraction Angiography unit (GE, Milwaukee, USA) or HiSpeed CT Scan unit (GE, Milwaukee, USA). A wide variety of catheters, microcatheters, guidewires, embolic materials, and other materials were used, as was best suited for the individual procedure and angioarchitecture. Post procedural care was in the ward for procedures performed under sedation and local anesthesia, and in the neurological intensive care unit for patients who underwent procedures under general anesthesia.

The mode of presentation, relevant history and findings of neurological examination were obtained from the patient's medical records. Analysis of the preprocedural imaging and angio-architecture of the lesion, as well as the check-angiographic studies performed after the procedure, was performed by two experienced observers. Details of the procedure including the technique and hardware used were obtained from the patients' records and cath lab records. The details of post procedural hospital stay and neurological outcome were assessed from the clinical follow up records. Neurological status was assessed separately before and after each session of therapy. The severity of complication in relation and its effect on the outcome of procedure at the time of discharge was classified based on an six-point 'complication outcome' scale that was improvised by the department of Radiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum (Table 4.1). Rankin scale (Table 4.2) was used to study the functional status of the patient before and after the procedure. Duration of hospital stay after the procedure was compared with occurrence of complications. For patients in whom, multiple sittings of intervention were carried out during the same admission, or in

Table 4.1: Scale to measure the severity and functional outcome of the neurointerventional procedural complications
Improvised by the Department of Radiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum

Severity of Complication	Complication Outcome	Description
No Complication	0	No complication
Mild Complication	1	Minor technical complications (Not affecting the outcome)
	2	Transient neurological deficits necessitating prolongation of hospital stay or resolving within 1 month
Moderate Complication	3	Minor permanent neurological deficits
Severe Complication	4	Major permanent neurological deficits
	5	Vegetative state
	6	Death

Table 4.2: Rankin Score
(Adapted from Van Swieten et al. Interobserver agreement for the assessment of handicap in stroke patients. *Stroke* 1988;19:604–607)

0 = No symptoms at all

1 = Able to carry out all usual duties and activities

2 = Unable to carry out all previous activities but able to look after own affairs

3 = Requiring some help but able to walk without assistance

4 = Unable to walk without assistance and unable to attend to bodily needs

5 = Bedridden, incontinent, and requiring constant nursing care and attention

6 = Death

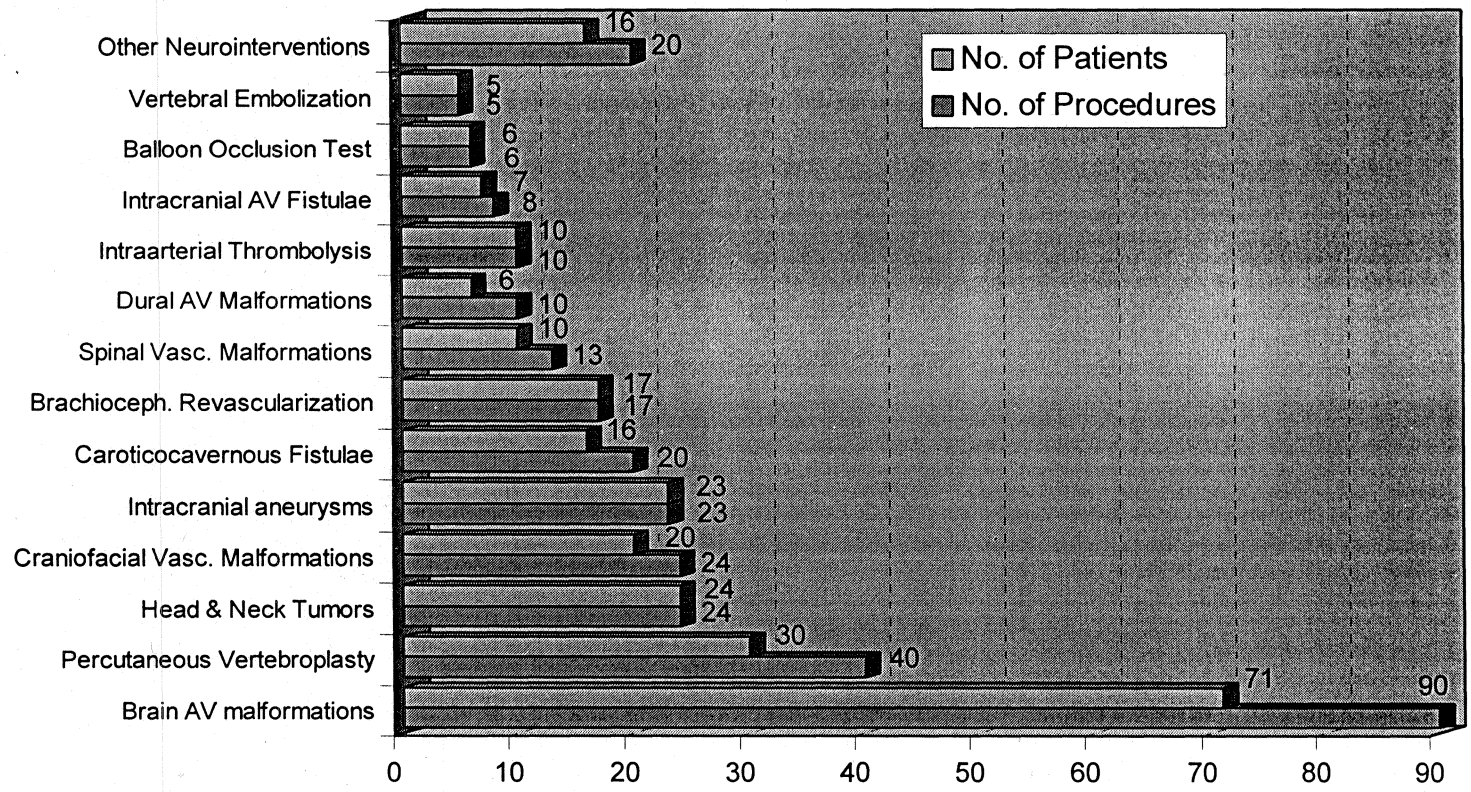


Figure 4.1: Neurointerventional procedures performed during the study period

whom, surgery was performed after the intervention, the duration of post procedural stay was taken as the time till next sitting of the procedure, surgery or discharge. For all patients who had complications during the procedures, up-to-date information on the functional status was gathered by outpatient review at last follow up.

Details of the procedures carried out are summarized in Figure 4.1

Cerebral Arteriovenous Malformations

71 patients with cerebral pial arteriovenous malformations were treated during the study period. These consisted of 52 male and 19 female patients. Average age at presentation for embolization was 31.1 years (range 8 – 59 years). Modes of presentation included chronic headache (n=44), seizures (n=42), intracerebral hemorrhage (n=33). Nine patients had progressive neurological deficits while 6 other patients presented with miscellaneous neurological symptoms including vertigo and cerebellar signs (3), dyskinesias (1), proptosis (1) and with benign intracranial hypertension (1). Among those who presented after intracerebral hemorrhage, mean duration between hemorrhage and embolization was 18.2 months (range 3 days to 10 years). The demographic profile of the patient population is summarized in Table 4.3 and Figure 4.2.

The angioarchitectural characteristics and details of AVM embolization of the patient population are summarized in Table 4.4. Angiographic evaluation revealed compact nidus in 56 patients and diffuse nidus in 34 patients. Medium sized (3-6cm) nidus was the commonest (66.7%). Most patients (n=52, 57.8%) had a complex angioarchitecture, with areas of microfistulae interspersed with areas of plexiform nidus. Pure plexiform nidus

Table 4.3 Demographic and Clinical Characteristics of Patients With Brain AVM Undergoing Embolization Therapy

Age in years Mean (range)	31.1(8 – 59)
Sex ratio M:F	52 : 19
Initial presentation, n (%)	
Intracranial hemorrhage	33 (46.5 %)
Seizures	42 (59.1 %)
Headache	44 (61.9 %)
Focal deficit	9 (12.7 %)
Other symptoms	6 (8.5 %)

Figure 4.2: Demographic characteristics of patients with brain AV malformations

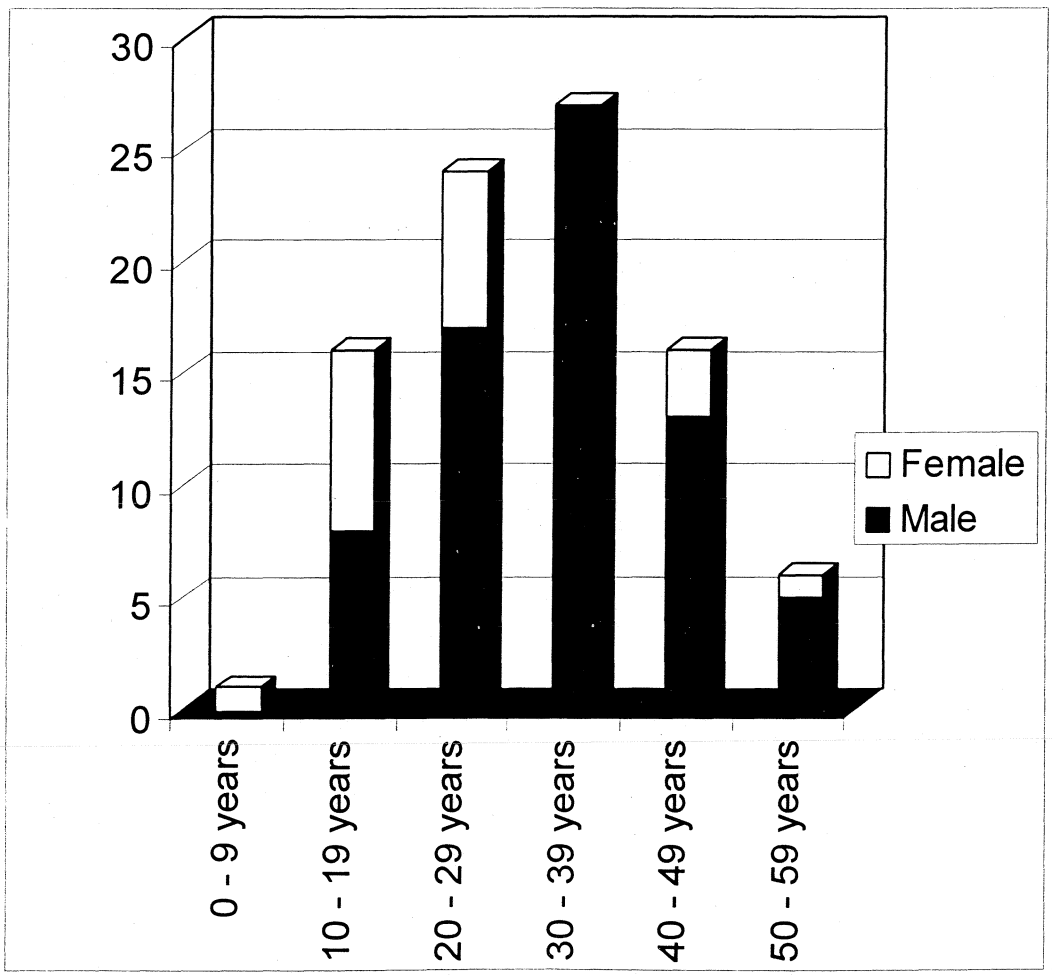


Table 4.4 Angioarchitectural Characteristics and details of AVM embolization

Angioarchitecture	
Feeders	
No. of cortical feeders mean (range)	3.48 (1 - 9)
Deep arterial feeders,* n (%)	28 (31.1%)
Concurrent arterial / nidal aneurysms,# n (%)	5 (5.5%)
Nidus, n (%)	
Compact nidus	56 (62.2%)
Diffuse nidus	34 (37.8%)
Arteriovenous shunt type, n (%)	
Predominant Plexiform nidus	32 (35.6%)
Plexiform nidus + Microfistulae	52 (57.8%)
Macrofistulae	6 (6.7%)
Venous drainage	
No. of cortical veins, mean (range)	2.96 (0 - 8)
Pure cortical venous drainage, n (%)	57 (63.3%)
Pure deep venous drainage, n (%)	7 (7.8%)
Aneurysmal dilatation of draining veins, n (%)	28 (31.1%)
Venous stenosis, n (%)	24 (26.7%)
Venous reflux, n (%)	21 (23.3%)
Spetzler-Martin criteria, n (%)	
Small AVM (diameter <3 cm)	16 (17.8%)
Medium AVM (3–6 cm)	60 (66.7%)
Large AVM (>6 cm)	14 (15.6%)
Deep venous drainage, n (%)	33 (36.6%)
Eloquent location,¶ n (%)	83 (92.2%)
Spetzler-Martin score, n (%)	
1	0 (0%)
2	23 (25.6%)
3	40 (44.4%)
4	19 (21.1%)
5	8 (8.9%)
Embolizations, n	90
Patients undergoing surgery after endovascular treatment, n (%)	4 (5.6%)
No of feeders embolized per procedure, n (%)	2.29 (0 - 5)
Percentage reduction in nidus per sitting of embolization	55.6 % (10-100%)

* Defined as penetrating branches of the major intracranial arteries of the circle of Willis.

Includes aneurysms on feeding arteries and intranidal aneurysms

¶ Includes location in the sensorimotor, visual, or language cortex; basal ganglia; internal capsule; brainstem; cerebellar peduncles; and deep cerebellar nuclei

was noted in 32 (35.6%) patients, while 6 (6.7%) patients had macrofistulae that were the chief contribution to the arteriovenous shunt. The lesions were supplied by a mean of 3.48 (range 1-9) named 2nd order branches of the circle of Willis. Contributions from the basal perforator arteries were seen in 28 patients, 7 of who had pure deep arterial supply. Flow related aneurysms were seen in 5 patients. The venous drainage was by a mean of 2.96 cortical veins (range 1-9). Deep venous drainage was seen in 33 patients, 7 of who had pure deep venous drainage. On the Spetzler-Martin grading system, most (44.4%) patients had grade 3 lesions, while grade 2, grade 4 and grade 5 lesions were seen in 25.6%, 21.1% and 8.9% respectively.

Embolization was carried out either as an isolated procedure for obliteration of small AVMs in one or more sittings (n=65, 91.5%), for preoperative embolization of inaccessible portions prior to surgical resection (n=4, 5.6%) or for volume reduction of nidus prior to stereotactic radiotherapy (n=2, 2.8%). The primary indication for treatment was to prevent hemorrhage from the AVM. In three patients, apart from this goal, embolization was performed to relieve benign intracranial hypertension (n=1), progressive neurological deficits (n=1) and dyskinesia (n=1). A total of 90 embolization procedures were performed. Superselective microcatheter cannulation of arteries feeding the AVM and injection of *N*-butyl cyanoacrylate or alcohol were used to occlude the fistulae. Decisions regarding choice of the sector of the AVM that was embolized and degree of shunt reduction per treatment session were made based on the angio-architecture of the lesion and the appearance of check angiogram.

The clinical details and angio-architecture of the AVM were analyzed as per the reporting terminology guidelines suggested by the Joint Writing Group of the ASITN, AANS, CNS and AAN [73]. The occurrences of any adverse event during the procedure or within the 1st month after the procedure were recorded. The presence of any predisposing / aggravating factor was also recorded and analyzed. Clinical presentation, angioarchitecture and procedural details of the 'complications' group were compared with those of the 'no complications' group, and the results were tabulated.

Intracranial Aneurysms

The demographic, clinical, angioarchitectural characteristics and procedure details are summarized in Table 4.5. Twenty-three patients (male - 13, female –10) with intracranial aneurysms were treated during the study period. Mean age at presentation was 51.5 years (range 24-66 years). Eleven of these aneurysms had ruptured at presentation, while 5 presented with cranial nerve palsies. Two patients had iatrogenic pseudoaneurysms of the cavernous segment of the ICA following trans-sphenoidal resection of pituitary adenoma (n=1) and functional endoscopic sinus surgery (n=1). One other patient had initially presented with a spontaneous CCF and had an angiographically documented Type A CCF. Elective balloon embolization of the fistula was planned 1 month later; but pre-procedural angiography revealed complete occlusion of the fistula, with a wide necked pseudoaneurysm at the site. Coil embolization of this lesion was performed.

Neurological examination revealed deficits in 8 patients. Systemic illnesses such as hypertension, diabetes and coronary artery disease were present in 18, 12 and 7 patients respectively. Among the 11 patients who presented with subarachnoid hemorrhage, most

Table 4.5 Demographic, Clinical, and Morphological Characteristics of Patients With Intracranial Aneurysms Undergoing Endovascular Therapy (n=23)

Age in years Mean (range)	51.5 years (24 - 66 years)
Sex ratio M:F	13 : 10
Initial presentation, n (%)	
Subarachnoid Hemorrhage	11 (47.8 %)
Cranial nerve deficits	5 (21.7 %)
Focal neurological deficits	4 (17.4 %)
Seizures	2 (8.7 %)
Headache	5 (21.7 %)
Ruptured aneurysms	
WFNS Grade 1	5 (45.4 %)
WFNS Grade 2	2 (18.2 %)
WFNS Grade 3	2 (18.2 %)
WFNS Grade 4	1 (9.1 %)
WFNS Grade 5	0 (0.0 %)
Unclassified (Patient under ventilation)	1 (9.1 %)
Location of aneurysm	
Anterior Circulation	12 (52.2 %)
Posterior Circulation	11 (47.8 %)
Type of aneurysm	
Saccular	21 (91.3 %)
Narrow neck (< 4mm)	7 (33.3 %)
Wide neck (> 4mm)	14 (66.6 %)
Fusiform	2 (8.7 %)
Intervention	
Aneurysm coiling	16 (69.5 %)
Parent artery occlusion	7 (30.5 %)
With aneurysm coiling	5 (21.7 %)
Without aneurysm coiling	2 (8.7 %)
Remodeling techniques	
Balloon remodeling	5 (29.4 %)
Stent assisted coiling	1 (5.9 %)

were in WFNS grade 1 (n=5), while 2 patients each were in WFNS grade 2 and 3 respectively, and 1 patient was in grade 4. WFNS classification could not be performed in 1 patient with a post-surgical rebleed of aneurysm remnant, as he was under mechanical ventilation. Angiographic evaluation revealed fusiform aneurysms (n=2), saccular aneurysms with narrow neck (n=7) or saccular aneurysms with wide neck – wider than 4 mm (n=14). Twelve aneurysms were located in the anterior circulation while 11 were located in the posterior circulation. A second unrelated aneurysm was noted in 3 patients.

Interventions were carried out in single sittings in all patients. Among patients with ruptured aneurysms, excluding one patient who presented to us with regrowth of aneurysm neck after undergoing coil embolization elsewhere, the mean duration between rupture of aneurysm and the procedure was 1.45 months (range: 3 days – 7 months). Seven patients underwent coil / balloon occlusion of the parent artery, with (n=5) or without (n=2) occlusion of the aneurysm; while 16 patients underwent coiling of the aneurysm sac with sparing of the parent artery. The causes for surgical exclusion included: anticipated surgical difficulty because of aneurysm size or location in 16 patients (69.6%); poor medical status in 5 patients (21.7%); failed surgical exploration in 1 patient (4.3%); and refusal of surgery by 1 patient (4.3%). All coiling procedures were performed under general anesthesia and carotid trapping procedures were performed under conscious sedation. A complete cerebral angiographic series was performed before each procedure to document the location, morphology of the aneurysm, neck-fundus ratio, presence of arterial vasospasm, and angiographic evidence of mass effect or

hydrocephalus. The microcatheter was positioned in the aneurysm by means of high quality fluoroscopy and roadmapping and GDC coils were sequentially delivered. The procedure was considered terminated when a solid cast of the aneurysm was achieved or when herniation of the last GDC coil in the parent artery was observed. Patients in whom coiling of the parent artery along with the aneurysm was performed underwent test occlusion of the artery prior to the procedure.

Intracranial Arteriovenous Fistulae

Seven patients with cerebral arteriovenous fistulae were treated in 8 sessions of embolizations. These included 4 cases of vein of Galen malformation, 2 cases of non-Galenic arteriovenous fistula, and 1 case of postoperative arteriovenous fistula.

The 4 cases of VOG malformations included 3 children (mean age 1.75 years) and one 28 year old adult. Two children with mural type of malformation presented with rapidly enlarging head circumference and one child was diagnosed to have the malformation antenatally. The adult patient presented with recurrent headache and one episode of sudden onset severe headache and loss of consciousness. Imaging revealed intraventricular hemorrhage and angiographic evaluation revealed a choroidal type of vein of Galen malformation. Embolization was performed in a single sitting in these lesions. In three patients, embolization of the sac with coils, followed by transarterial embolization of the fistulae with cyanoacrylate glue was performed. The venous sac was initially accessed through femoral venous access and a microcatheter was negotiated into the venous sac through the falcine sinus (2) or straight sinus (1). Large coils were deployed within the venous sac so as to achieve reduction in the arteriovenous shunt, as

documented by periodic check angiograms. Subsequently, a high concentration of cyanoacrylate glue was used to occlude the fistulae from the arterial side. In one other patient, transarterial embolization was carried out without prior venous sac coil embolization.

Two patients with non-galenic arteriovenous fistulae were treated in three sittings of embolization. One was a 6-month-old child who presented with hydrocephalus. Imaging and DSA revealed an arteriovenous fistula located ventral to the brainstem – fed by an enlarged thalamoperforator branch, and draining into the torcular. The lesion was embolized in two sittings with initial flow reduction using transarterial deposition of coils within the venous sac, followed by transarterial embolization with cyanoacrylate glue. The child did well for 7 months after the embolization procedure and died during a subsequent admission for surgical management of hydrocephalus. The second patient was a 9-year-old child who presented with history of frequent falls. Imaging and angiography revealed an arteriovenous fistula fed by the left middle cerebral artery and pericallosal branch of left anterior cerebral artery. Both vessels were embolized with 95% cyanoacrylate glue to achieve about 80% reduction of the arteriovenous shunt.

One 42-year-old male patient had been operated for a right temporal arteriovenous malformation. Postoperative check-angiogram revealed a high flow arteriovenous fistula at the operative site. The lesion was selectively catheterized and embolized with 95% glue to achieve complete occlusion of the fistula.

Carotico-Cavernous Fistulae

Sixteen patients with carotico-cavernous fistulae were treated in 20 endovascular procedures during the study period. These included 11 male and 5 female patients with a mean age of 30.9 years (range: 20 – 61 years). The aetiological factors included trauma (n=10), spontaneous (n=5) and one iatrogenic fistula (following rupture of cavernous segment of the internal carotid artery during attempted stenting of an atherosclerotic stenosis). The duration of symptoms prior to presentation ranged from 15 days to 5 years (mean – 6.1 months). Clinical presentation with orbital symptoms (proptosis, cheimosis and conjunctival injection) was seen in 15 patients. 12 patients had abnormalities of eye movement, while 9 patients had deterioration of vision. Angiographic evaluation was performed to delineate the location, type and caliber of the fistula, major arterial feeders and venous drainage patterns. Angiographic types included Type A (11 patients), Type C (1 patient) and Type D (4 patients).

Thirteen patients were treated in single sessions, while two patients required two sessions due to failure of first session (n=1) or incomplete closure of fistula (n=1). In one patient, repeated deflation of the detachable balloon necessitated embolization in three sittings. Embolization was performed through the arterial route (n=16) or venous route (n=4). In two patients, attempts at combined embolization through arterial and venous routes failed. The treatment strategies included closure of fistula (n=8), trapping of the internal carotid artery (n=4), embolization of external carotid feeders (n=2) or coil embolization of cavernous sinus (n=6). Two procedures were abandoned. One was a post-traumatic CCF in which, failure of detachable balloon to reach the site of the fistula, necessitated trapping of the internal carotid artery in a subsequent sitting. The other patient had

undergone internal carotid ligation elsewhere and failure of the microcatheter to negotiate through the posterior communicating artery led to abandoning the procedure. Complete obliteration of the fistula could be achieved in 12 procedures, while near total and partial obliteration could be achieved in 4 and 2 procedures respectively.

The occurrences of complications and the factors predisposing to / aggravating the complication were analyzed.

Dural Arteriovenous Malformations

Six patients with dural arteriovenous malformations were treated in 10 sessions. All patients were male and the mean age at presentation was 34.1 years (range 18 - 46 years). The clinical presentation included tinnitus (n=6), recurrent headache (n=6), deterioration of vision (n=3), proptosis and cheimosis (n=2), memory impairment (n=1), gait difficulties (n=1) and behavioural changes (n=1). The duration of symptoms ranged from 3 months to 11 years. Angiographic evaluation revealed dural arteriovenous malformations involving the transverse-sigmoid junction (n=3), transverse sinus (n=2) and posterior third of superior sagittal sinus (n=1). Angiographic types, based on the pattern of venous drainage[74], included Type II a+b (n=4), Type I (n=1) and Type IV (n=1). Embolization of three cases of Type II a+b and one Type IV fistula were performed by coil embolization of the venous sinus, using burr hole (n=3) or jugular venous access (n=1). In addition, the Type I fistula and the arterial feeders of Type II a+b fistula were embolized with particles and glue. Extent of reduction of the arteriovenous shunt ranged from 20% with transarterial embolization; to 90-95% using combined transvenous - transarterial embolization.

Craniofacial Vascular Malformations

Twenty patients with craniofacial vascular malformations underwent embolization in 24 procedures. While 18 patients underwent embolization in single sittings, one patient with a mandibular AVM required two sittings of embolization prior to surgery and one child with a sinus pericranii was treated in four sittings. The mean age of the patient population was 22.5 years (6-46 years). The pathological substrates were arteriovenous malformations in 17 patients (cirsoid aneurysm – 6, cheek – 5, neck – 2, shoulder – 2, lip – 1, tongue – 1); hemangiomas in 2 patients and sinus pericranii in 1 patient. The duration of symptoms were 7 months to 20 years (average – 76.2 months).

While all patients presented with swelling, only one reported a rapid increase in the size of the lesion. Eight patients had pain over the swelling. Skin changes such as ulcerations and bleeding were seen in 7 patients each. Though three patients with scalp vascular malformations had history of headache ipsilateral to the lesion, none had any neurological deficits. Six patients were operated previously for the swelling and had experienced recurrence of the lesion.

Of the 24-embolization procedures, 18 were performed with a goal of preoperative devascularization of the mass, 3 were performed as definitive procedures and 3 were performed as palliative measures. Nineteen procedures were performed under local anesthesia, while 5 were performed under general anesthesia. Transarterial embolization was carried out in 15 patients, using PVA particles and gelfoam (n=6), only PVA particles (n=6), and glue (n=3). Percutaneous direct puncture embolization using glue (20% – 50%) was performed in 9 patients. Embolization procedures resulted in 40% – 100% reduction in the vascularity of the lesions.

Head & Neck Tumors

Twenty-four patients with head and neck tumors underwent preoperative embolization in 24 procedures. The mean age of the patient population was 28.1 years (2-65 years). The duration of patients' symptoms was for 1 month to 10 years (average – 30.6 months). Fourteen patients presented with external swelling, while 10 other patients had intracranial extra-axial lesions. Six patients had pain over the swelling. Skin changes such as ulcerations and bleeding were seen in 2 patients each. An additional mode of presentation was epistaxis (n=4), ear bleed (n=2) and tumor bleed (n=1). All ten patients with intracranial mass lesions had history of neurological symptoms ranging from headache to lower cranial nerve palsies and pyramidal symptoms. Two patients had coexisting diseases – rheumatic heart disease and systemic hypertension.

On angiographic evaluation, while all lesions had feeders from the external carotid artery (mean number of feeders – 2.3), additional feeders from the internal carotid system were seen in 9 patients (ophthalmic artery (n=2), meningeal branch of posterior cerebral artery (n=2), meningeal branches from cavernous part of internal carotid artery (n=2), anterior cerebral artery (n=1) and anterior and posterior inferior cerebellar arteries (n=2). Features of arteriovenous shunting were seen in 17 patients.

Apart from embolization of an extremely vascular parotid tumor in a 2-year-old child, all procedures were performed under local anesthesia. Transarterial embolization was carried out in 20 patients, using PVA particles (n=13), PVA particles and gelfoam (n=6), and only gelfoam (n=1). Protection of the internal carotid artery using a Berman balloon (Arrow therapeutics, USA) was performed during particulate embolization of external carotid feeders in two patients. Percutaneous direct puncture embolization using glue

(20% – 50%) was performed in 4 patients. Embolization procedures resulted in 60% – 100% reduction in the vascularity of the lesions (mean - 85%).

Carotid and Subclavian Angioplasty and Stenting

17 patients (13 male and 4 female) with hemodynamically significant stenotic lesions of the brachiocephalic vessels (internal carotid (n=11), subclavian (n=5) and common carotid (n=1)), underwent revascularization procedures during the study period. To be considered for stenting, symptomatic patients required at least 50% stenosis and asymptomatic patients required at least 70% diameter narrowing by angiographic North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria.

The patients' ages ranged from 32 to 69 years (mean – 51.6 years). Eighty eight percent of the stenotic arteries were symptomatic: 41 % presented with transient ischemic attack, 12 % with relatively recent stroke, and 12 % with amaurosis fugax. Five patients had arm pain secondary to subclavian stenosis, 3 of them had features of vertebrobasilar steal. Coexistent systemic illnesses included hypertension (n=12), diabetes (n=9), symptomatic coronary artery disease (n=8), dyslipidemia (n=7) and aortoarteritis (n=1). In two patients, the procedure was performed to salvage the artery from posttraumatic dissection (n=1) or postoperativve residual stenotic lesio with intimal flap (n=1). In two patients, the procedure was performed to improve the cerebral blood flow in a case of contralateral carotid occlusion for cavernous ICA aneurysm (n=1) and to improve inflow in a case of ICA-MCA reversed-saphenous-vein graft.

All patients were referred for neurologic examination to document the preprocedural clinical neurologic status. Symptomatic patients or patients with history of stroke or transient ischemic attacks and those with abnormal results of the neurologic examination underwent CT or MR imaging (if this has not recently been performed) to document preprocedural changes. Antiplatelet therapy was started 3 days before the procedure: aspirin (325 mg administered daily), and clopidogrel (75 mg administered once a day), were administered. Angiography and stenting were performed with the patient under local anesthesia in all patients except the two patients in whom carotid stenting was performed as an adjunct to management of intracranial aneurysms. All procedures were carried out after careful assessment of the intracranial hemodynamics and collateral channels. Predilation was employed in 12 (70.5%) cases, which had tight stenotic lesions. The procedures involved only angioplasty (n=4) or deployment of self-expanding stents (n=12) or balloon expandable stents (n=1). The diameters of the stents used, ranged from 6 mm to 14 mm. Protection devices (Guard wire Plus Temporary Occlusion and Aspiration System, Medtronic AVE, USA) were used in two patients.

The procedural details were reviewed for the occurrence of any technical mishaps. The details of pre-procedural neurological examination were compared with that in the post procedural follow up or at 1-month follow up to document any procedure related deterioration of neurological status.

Percutaneous Vertebral Interventions

Percutaneous vertebral interventions were performed in 30 patients. Percutaneous vertebroplasty was performed at 38 vertebral levels in 28 patients (male –8, female-20) with low back pain. The average age of the patients at the time of the vertebroplasty was

46 years (range 27–77 years). The procedures were performed using the fluoroscopically guided trans-pedicular / paravertebral approaches under local anesthesia and conscious sedation. Disposable Murphy bone biopsy needles (Cook, USA) were placed at the junction of anterior and mid thirds of the vertebral body. 11G needles were used in 26 procedures and 13G needles were used in 12 procedures. Before injecting the PMMA, venography was conducted in 2 patients to evaluuate the venou drainage pattern. CMW 2 bone cement (DuPuy, Johnson and Johnson, USA) was additionally opacified using sterile barium sulfate powder and injected using hand injection technique.

In 2 patients injection of embolic agent into the posterior elements was performed under CT guidance to treat the posterior elements involved by vertebral tumors (hemangioma (n=1) and osteoclastoma (n=1)). In the patients with the hemangioma involving the posterior elements, 4 ml of absolute alcohol was injected through an 18G spinal needle under fluoroscopic guidance. In the patient with the osteoclastoma, CT guided injection of bone cement was performed. Both patients had good filling of the tumor with the agents.

Occurrence of complications and their severity and outcome were correlated with the underlying pathology, clinical symptomatology, presence and degree of vertebral collapse, integrity of the posterior cortex of the vertebral body, presence and degree of epidural component and cord compression, pre-procedural neurological status, use of unilateral or bilateral access, position of the needle tip, volume of PMMA injected, percentage of cast achieved and leakage of PMMA into extravertebral tissue planes.

Vertebral Embolizations

Embolization of hypervascular vertebral tumors was performed in 5 patients. The pathological substrates included vertebral hemangiomas (n=4) and giant cell tumors (n=1). Four patients presented with focal back pain referable to the site of the lesion, while one patient with significant compression of the spinal cord presented with progressive paraparesis.

A diagnosis of vertebral tumor was made on the basis of findings at MR imaging and other imaging studies in all patients. All patients underwent angiography and embolization under local anesthesia and with intravenous sedation. Selective angiography of the suspicious arterial branches supplying the tumor was then performed. No provocative testing was attempted. In total, 9 arteries were embolized. Embolic agents that were employed included polyvinyl alcohol particles (n=6) and PVA particles and gelfoam (n=3).

Spinal Vascular Malformations

During the study period, 10 patients (8 male and 2 female) with angiographically proven spinal vascular malformations underwent endovascular management of the lesions in 13 procedures. Mean age of the patients was 35.5 years. The underlying malformations consisted of spinal dural arteriovenous fistulae (n=4), perimedullary fistulae (n=5) and spinal cord arteriovenous malformation (n=1). The patients had been symptomatic for an average of 24.3 months prior to presentation (4months - 6 years). Four patients presented with sudden onset of paraparesis while other 6 presented with gradual deterioration of lower limb power.

All patients were treated under general anesthesia to minimize patient motion and optimize digital subtraction angiography (DSA) and roadmapping. Comprehensive selective spinal angiography was performed, including injection of vertebral, thyrocervical, costocervical, intercostals and lumbar arteries and internal iliac arteries. When the malformation and feeder were identified, superselective catheterization was performed using a microcatheter (Prowler -10, Cordis, USA or Spinnaker - 1.5, Boston Scientific Corporation, USA) with the aid of digital fluoroscopy and roadmapping. DSA runs were performed to improve visualization of the fistula and to exclude the presence of anterior or posterior spinal arteries. The microcatheter was positioned as close to the lesion as possible and angiographic flow characteristics were analyzed to determine the type of embolic material that be could safely used.

Pre- and post intervention spinal MR examinations and follow-up spinal angiograms were retrospectively reviewed. If unavailable, information was obtained from available reports. Motor, sensory, gait, micturition and bowel disabilities were recorded prior to any intervention and at last follow-up.

Intracranial Thrombolysis for Procedure Related Complications

Ten patients underwent thrombolysis for procedure related thromboembolic complications. The precipitating events included thromboembolism following coronary angiography (n=3), intracranial aneurysm coiling (n=2), intracranial AVM embolization (n=2), craniofacial tumor embolization (n=1), carotid stenting (n=1) and subclavian

stenting (n=1). Time from onset of occlusion and thrombolysis ranged from 10 minutes to 4 hours.

Thrombolysis was performed under general anesthesia in the four patients in whom the event occurred during intervention for intracranial AVM or aneurysm, and thus the pre-thrombolysis neurological status could not be evaluated. The symptoms at presentation in the other 6 patients included confusion and dysphasia (n=3), rapid deterioration of vision (n=1) and loss of consciousness due to basilar top occlusion (n=1), while one patient was asymptomatic. The dose of urokinase used ranged from 1 lakh to 7.5 lakh units (mean – 3.9 lakh units). The vascular territories involved by the embolus included MCA (n=7), ACA (n=1), basilar top (n=1), posterior choroidal artery (n=1), ophthalmic artery (n=1) and external carotid artery (n=1).

Temporary Balloon Occlusion Test

Temporary balloon occlusion test was performed for testing adequacy of collateral circulation in five patients with giant aneurysms of the cavernous and caroticoophthalmic segments of the internal carotid artery and in one patient with recurrent pseudoaneurysm of the internal carotid artery. In 3 patients, the study was done as a part of an interventional procedure for treating the aneurysm - including attempted balloon assisted coiling of the aneurysm (n=1) and trapping of the internal carotid artery (n=2). In 3 other patients, the test was performed prior to clipping of the aneurysm and surgical trapping of the internal carotid artery respectively. Total duration of the study was a maximum of 90 minutes. Induction of hypotension was not employed.

Table 4.6 Details of Miscellaneous Neurointerventional Procedures performed during the study period

	Patients	Procedures
- Intracranial thrombolysis for cerebral venous thrombosis	2	4
- Extracranial Arteriovenous fistulae	2	4
- Intracranial stenting for aneurysms / stenotic lesions	4	4
- Stenting for aneurysms of brachiocephalic vessels	3	3
- Chemical angioplasty for vasospasm	3	3
- Intravascular foreign body retrieval in cranial circulation	2	2
Total:	16	20

Miscellaneous Neuro-interventional Procedures:

This group includes those types of neuro-interventional procedures, of which less than or equal to 5 cases have been performed during the study period. The details of these procedures are depicted in Table 4.6

Intracranial thrombolysis for cerebral venous – sinus thrombosis

Two female patients, with cerebral venous-sinus thrombosis underwent two sittings of thrombolysis each, during the study period. Both were young female patients, and had presented with headache and progressive deterioration of level of sensorium. Imaging revealed extensive superficial venous sinus and straight sinus thrombosis in both patients. They were referred for thrombolysis after a mean of 45 days after onset of symptoms. One patient had bilateral hemorrhagic cerebral infarcts at the time of starting thrombolysis. One patient tested positive for anti-phospholipid antibodies. Both patients had extensive occlusion of the superior sagittal sinus and transverse-sigmoid sinuses. Thrombolysis was performed by transfemoral venous access using mechanical thrombolysis and urokinase. The microcatheters used included Tracker-18 (Boston Scientific, USA) and Microferret (Cook, USA). Bolus doses of local urokinase (mean dose – 3.17 lakh units) were injected in the cath lab and followed by local infusion of urokinase at the rate of 30,000 to 50,000 units per hour (mean dose – 6.21 lakh units) in the ICU. Concomitantly, heparin infusion was continued at 500 units per hour. Periodic check angiography and repositioning of the microcatheter was performed for effective thrombolysis. Procedure was terminated in both cases when no further effective angiographic recanalization could be achieved

Extracranial Arteriovenous Fistulae

Two patients with extracranial arteriovenous fistulae were embolized in four procedures. One 40-year-old woman had a spontaneous vertebrovenous fistula. Initially, transarterial balloon embolization of the fistula was peformed. Subsequent deflation of the balloon needed direct puncture of the venous sac and coil embolization during a second sitting. The other patient was a 35-year-old man who presented with progressive deterioration in level of consciousness, with right-sided hemiparesis about 1 year after a penetrating injury to the neck. Imaging and angiographic studies revealed a left sided carotico-jugular fistula with reflux of venous drainage into the intracranial venous system. Occlusion of the left common carotid artery and the left internal jugular vein was performed using balloons and GDC coils. Near complete obliteration of the fistula was obtained, with minimal residual shunt from retrograde flow through the distal internal carotid artery. Attempts at embolization of this residual shunt months later, using a retrograde access through the posterior communicating artery were unsuccessful.

Intracranial stenting for atherosclerotic stenosis / wide necked aneurysms

Stenting of intracranial vessels was performed in four patients during the study period. In two patients, the procedure was performed for symptomatic stenosis of the internal carotid artery. The first patient had two stenotic lesions in the left cavernous and supraclinoid ICA causing 50% and 70% stenosis respectively; with complete occlusion of the right ICA. The second patient had a symptomatic 50% stenosis of the right cavernous ICA and an asymptomatic 90% stenosis of the left distal common carotid artery with reformation of the internal carotid artery through external carotid branches.

In two other patients, stenting was performed to assist in coiling of intracranial aneurysms. The third patient had undergone a left ICA sacrifice with a ICA - MCA, reversed saphenous venous bypass graft for a giant cavernous segment ICA aneurysm in 1994. After being asymptomatic for 9 years, she had an episode of spontaneous left insular bleed. Diagnostic angiogram revealed a small wide necked aneurysm at the junction of the saphenous vein graft and inferior division of the MCA. Stent assisted coiling was performed in this patient. The fourth patient had a spontaneous Type A carotico-cavernous fistula that was confirmed by angiography. 4 months later, review angiograms performed before proposed balloon occlusion of the fistula revealed complete spontaneous obliteration of the fistula, with a residual pseusdoaneurysm in the cavernous segment of the ICA. The aneurysm was initially coiled using GDC coils. Subsequent coils were seen to prolapse into the parent artery- and hence, it was decided to place a stent across the neck of the aneurysm and then pack the aneurysm with coils.

The stents used these cases included Be stent (Medtronic, CA, USA) (3.5 x 12mm), AVE (Medtronic, CA, USA) (2.5 x 8mm), AVE (2.75 x 12mm) and AVE (2.5 x 9mm) stents. One stent had to be retrieved from the cavernous ICA due to malposition.

Endovascular management of aneurysms of the brachiocephalic vessels

Three patients with aneurysms of the brachiocephalic vessels were treated. The first patient had a traumatic pseudoaneurysm of the ICA, which was treated by deployment of a stent graft The second patient had a pseudoaneurysm of the ICA which was earlier

treated by surgery and then by stent graft placement. Recurrence of the pseudoaneurysm necessitated occlusion of the ICA with balloons and coils. The third patient had a left subclavian artery pseudoaneurym. A nitinol stent was deployed through a left brachial access.

Mechanical and Chemical angioplasty for vasospasm

In three patients, intraarterial papaverine infusion was performed to relieve vasospasm. All three patients had undergone clipping of intracranial aneurysms and had deterioration of neurological status following surgery. In two patients, good opening of the spastic segment could be achieved, while in one patient with multiple intracranial aneurysms, a clip placed across the neck of an ICA bifurcation aneurysm was seen to mechanically kink the M1 segment of the middle cerebral artery. This did not respond to papaverine infusion, and required subsequent surgical repositioning of the clip

Intravascular foreign body retrieval in cranio-cerebral circulation

Retrieval of intravascular foreign body in the cranio-cerebral circulation was performed in two patients. The first patient was a 38-year-old male patient, in whom, the tip of a 7F Judkins Right coronary catheter broke during the course of a coronary angiographic study and lodged in the left vertebral artery. Using bilateral femoral arterial access, a 8F Zeppelin balloon catheter was used to achieve flow reduction, while the foreign body was retrieved with a vascular loop. The second patient was a 55-year-old woman, who underwent stenting assisted coiling of a wide necked cavernous ICA aneurysm. During deployment, the stent migrated proximally and lay within the C4-C5 segments of the

cavernous ICA. Repeated attempts to expand the stent further by overinflation of the balloon were unsuccessful. As the stent was lying freely mobile at this position, it was decided to retrieve the stent. The balloon of the stent deployment system was re-negotiated into the stent and inflated to firmly hold the stent. The whole assembly including the guide catheter, stent system and the stent was withdrawn smoothly into the abdominal aorta. A vascular snare was navigated into the abdominal aorta through an 8F sheath placed in the contralateral femoral artery. The stent was captured by the snare and retrieved.

In both cases, adequate heparinization was ensured so as to maintain an activated clotting time between 250 and 300 seconds during the procedure. Heparinization was continued for 48 hours after the procedure. Periodic monitoring of neurological status and coagulation parameters were performed.

5. RESULTS

RESULTS

A total of 310 neuro-interventional procedures were performed during the study period. These included embolization of brain AVMs (n=90), endovascular therapy for intracranial aneurysms (n=23), embolization of carotico-cavernous fistulae (n=20), intracranial arteriovenous fistulae (n=8), dural AV malformations (n=10), craniofacial vascular malformations (n=24), head and neck tumors (n=24), spinal vascular malformation (n=13), brachiocephalic revascularization (n=17), percutaneous vertebral interventions (n=40), balloon occlusion tests (n=6), intraarterial thrombosysis (n=10), embolization of hyper-vascular vertebral tumors (n=5) and miscellaneous neuro-interventions (n=20). The complication rates associated with various neuro-interventional procedures, timing of the complications, complication outcome and neurological outcome of these procedures are summarized in Table 5.1

Brain Arteriovenous Malformations

Of a total of 90 procedures of brain AVM embolization, 6 (6.7%) procedures resulted in significant neurological complications that caused permanent deficits or death (complication outcome ≥ 3). Five (5.6%) patients had transient neurological deficits that completely resolved before discharge (complication outcome – 2) . Thirteen other patients had minor technical complications that did not alter the management of the patient and did not require additional duration of hospital stay (complication outcome -1). The occurrence rates of complications in the present series are compared with the ASITN thresholds in Table 5.2.

Table 5.1: Comparison of Minor and Major Complications of Different Neurointerventional Procedures (Present series)

	Brain AVMs	Aneurysm	Cerebral AVFs	CCF	Dural AVMs	Craniofacial Embolization		Brachio-cephalic Revascularisation	Vertebroplasty etc.	Vertebral embolizations	Spinal Vasc Malf	Misc	**Total**
						Vasc. Malf.	Tumor						
No. of Procedures	90	23	8	20	10	24	24	17	40	5	13	20	**310**
No. of Complications	24 (26.7%)	7 (30.4%)	3 (37.5 %)	1 (5 %)	1 (10 %)	1 (4.2%)	5 (20.1%)	2 (11.8 %)	11 (28.9%)	0	3 (9.1 %)	5 (25 %)	**52 (16.7%)**
Outcome													
Death	2 (2.2%)	1 (4.3%)	2 (25%)	-	1 (10%)	-	-	-	-	-	-	1 (5%)	**7 (2.25%)**
Vegetative State	-	1 (4.3%)	-	-	-	-	-	-	-	-	-	-	**1 (0.32%)**
Major Neuro Def	2 (2.2%)	-	-	-	-	-	1 (4.2%)	-	-	-	-	-	**3 (0.97%)**
Minor Neuro Def	2 (2.2%)	2 (8.7%)	-	-	-	-	-	-	-	-	1 (7.7%)	1 (5%)	**6 (1.93%)**
Prolonged Stay / transient deficit with full recovery	5 (5.5%)	-	-	-	-	-	1 (4.2%)	1 (5.9%)	-	-	-		**7 (2.25%)**
Minor Technical	13 (14.4%)	3 (13.1%)	1 (12.5%)	1 (5%)	-	1 (4.2%)	3 (12.5%)	1 (5.9%)	11 (28.9%)	-	2 (6.3%)	3 (15%)	**39 (12.6%)**
Timing													
Intraprocedural	20	7	3	1	1	-	5	2	1	-	3	5	47
Post Procedural	4	-	-	-	-	1	-	-	-	-	-	-	5
Mean change on Rankin Score per complication	0.54	0.86	3.33	0	2	0	0.60	0	0	0	0	0.60	

Table 5.2 : Embolization of Brain Arteriovenous Malformations

Clinical Complications

	ASITN Thresholds	Present Study
Death	0 %	2.2 %
Major (Permanent Neuro. Deficits)	10 %	2.2 %
Minor (Permanent Neuro. Deficits)	10 %	2.2 %
Transient Neurological Deficits	20 %	5.6 %
Other Clinical Embolization related complications	10 %	10 %
Alopecia	10 %	*

* Not assessed

Technical Complications

	ASITN Thresholds	Present Study
Failure to obtain proper consent	0 %	0 %
Retained catheter fragment	5 %	4.4 %
Device failure directly contributing to an untoward clinical outcome	5 %	2.2 %
Operator error directly contributing to an untoward clinical outcome	5 %	
Inadvertent arterial occlusion and / or dissection	10 %	2.2 %

Timing of complications

The complications occurred chiefly during catheterization of the nidus prior to embolization (n=6) and during injection of glue (n=7). Thromboembolic complications and complications due to absolute alcohol occurred in two patients each. Four patients had fresh onset of neurological deficits due to post procedural hypertension. Three patients had complications, which occurred due to unrelated causes such as power failure during glue embolization (n=1) or attempted retrieval of a glued microcatheter.

Severity of complications

Procedure related deaths

Two patients (case no. 10 and 31, Table 5.3) died due to procedure related complications (complication outcome – 6). One patient with a parietal AVM had a feeder artery rupture during cannulation. Though immediate reversal of anticoagulation and embolization of the feeder with cyanoacrylate glue was performed, there was a large parietal lobe hematoma. Neurosurgical evacuation was not performed due to hesitation on the part of the neurosurgeon to perform the evacuation, in the presence of residual AVM in a recently anticoagulated patient. Though aggressive external ventricular drainage was performed and cerebral decongestant and cerebral protection therapy was instituted, the neurological status of the patient rapidly deteriorated and resulted in death. The second patient was a 46-year-old female patient with a left temporo-occipital AVM. Partial embolization of the lesion – two feeders with glue and one feeder with 35ml alcohol was performed. The post procedure period was complicated by hypertension that had large fluctuations despite aggressive management with parenteral sodium nitroprusside and nitroglycerin. The patient's drowsiness and restlessness was attributed the large dose of

Table 5.3 Details of Complications Associated with Embolization of Cerebral Arteriovenous Malformations

No.	Age Sex	Location	Complication	Neurological Deficits	Rankin Score*		Functional § Outcome
					Pre	Post	
Catheterization and Microcatheterization Related Complications							
11	20F	Rt Temporoparietal	Dissection and contrast extravasation from 3rd feeder – glue inj.	Nil	1	1	1
26	57M	Lt Temporal	Microcatheter rupture during catheterization – feeder occlusion	Nil	2	2	1
31	38M	Lt Medial Parietal	Feeder perforation during microcatheterization – glue inj.	Death	1	6	6
39	23M	Lt Post. Temporal	Perforation of feeder artery during cannulation – glue inj	Nil	1	1	1
56	16 F	Rt parietal	Nidus rupture during microcatheterization – glue inj	Nil	2	2	1
64	34M	Lt Temporal	Guidecatheter induced ICA spasm – NTG infusion	Nil	1	1	1
Complications Related Embolization and Glue							
6	23M	Rt Occipital	Basilar, PCA spasm on pulling glued microcatheter – papaverine	Nil	1	1	1
29	30M	Rt Frontoparietal	Microcatheter retention – Cut at groin	Nil	3	3	1
36	53M	Lt Frontoparietal	Venous escape of glue - Venous stasis	Hemiparesis	1	2	2
37	26F	Rt Temporal	Significant venous escape of glue – settled in straight sinus	Nil	2	2	1
43	23M	Rt Motor Strip	Feeder rupture during glue embolization – sealed with glue	Nil	2	2	1
45	16M	Rt Parietal	Microcatheter retention	Nil	1	1	1
69	39M	Lt Temporoparietal	Venous escape of glue – Lt temporoparietal hematoma	Sensory aphasia	1	1	2
Thromboembolic Complications							
28	34M	Cerebellar	Paraesthesias and mild weakness – 6 hrs after embolization	Hemisensory	3	3	3
61	16M	Rt Parietal	Embolic occlusion of M3 segment of MCA - Thrombolysed	Nil	1	1	1
Complications Related to use of Absolute Alcohol							
3	22F	Lt B.G., Thalamic	Spasm of feeder, Thromboembolism - Lt parietooccipital artery	Upgaze palsy	2	2	2
7	54M	Rt Parietal	Spasm of feeder due to reflux of alcohol – Infarction	Lt Hemiplegia	1	3	4
Complications Related to Post Procedural Hypertension							
10	46F	Lt Temporooccipital	Hypertension 4 hrs after alcohol embo – Parietooccipital bleed	Death	2	6	6
19	46M	Rt Temporoparietal	Hypertension 24 hrs after embolization	Mild facial palsy	1	1	2
30	59M	Lt Frontoparietal	Hypertension 2 hours after embolization	Hemiparesis	1	1	2
41	17M	Rt Motor strip	Hypertension 24 hours after embolization	Hemiplegia	2	3	4
Unrelated causes							
32	16F	Rt Occipital	Power failure during glue inj – occlusion of parietooccipital & calcarine artery	Lt homonymous hemianopia	1	2	3
49	32M	Rt Parietal	Spasm of ICA during retrieval of retained microcatheter	Nil	3	3	1
55	23M	Rt Motor Strip	Spasm of ACA during retrieval of retained microcatheter	Nil	2	2	1

*** Rankin Score:** 0 = No symptoms at all, 1 = Able to carry out all usual duties and activities, 2 = Unable to carry out all previous activities but able to look after own affairs, 3 = Requiring some help but able to walk without assistance, 4 = Unable to walk without assistance and unable to attend to bodily needs, 5 = Bedridden, incontinent, and requiring constant nursing care and attention, 6 = Death

§ Functional Outcome of Neurointerventional Procedure: 1 - Minor technical complications (Not affecting the outcome), 2 - Transient neurological deficits necessitating prolongation of hospital stay or resolving within 1 month, 3 - Minor permanent neurological deficits, 4 - Major permanent neurological deficits, 5 - Vegetative state, 6 - Death

alcohol. About two hours after the procedure, she had an intracerebral bleed and rapidly deteriorated.

Procedure related neurological deficits

Four patients (case no. 7, 28, 32 and 41, Table 5.3) had permanent neurological deficits one each, caused by thromboembolic episode, related to alcohol, related to post procedural hypertension and due to non target embolization. These resulted in minor permanent neurological deficits in 2 patients (hemisensory dulling, hemianopia) and major permanent neurological deficits in 2 patients (hemiparesis, brachial monoparesis)

Minor Technical Complications

Complications, the occurrence of which did not necessitate any change in the management strategies, chiefly occurred during the embolization procedure (during catheterization (n=5) and during glue embolization (n=5). Potentially dangerous complications in 5 patients (feeder rupture (n=2) and feeder perforation (n=1) during microcatheterization, rupture of microcatheter (n=1), feeder rupture during glue embolization (n=1)) were recognized immediately and immediate corrective measures were instituted, resulting in good outcome without any neurological deficits or prolongation of hospital stay.

Neurological Outcome of the procedures :Rankin Score

By baseline Rankin score, 63.3% of the patients had no functionally relevant neurological deficit (Rankin score= 1), 31.1% of patients were capable of looking after themselves (Rankin score =2) and 5.7% of patients needed some form of assistance (Rankin score =3). After the procedure, apart from the two patients who died, four patients had deterioration of neurological status (Rankin score 1 → 2 (n=2), 1 → 3 (n=1) and 2 → 3 (n=1)). By Rankin scale, 93.3% patients experienced no change in neurological status after treatment. The pre-procedure and post-procedure Rankin scores of the patients who underwent embolization of brain AVMs are summarized in Table 5.4

Comparative Analysis of procedures without and with complications

The mean age and sex ratio of both groups was not significantly different (Table 5.5). Analysis of modes of clinical presentation revealed that patients in the 'complications' group more frequently presented with hemorrhage and with progressive neurological deficits, than the 'no complications' group. They also had a worse preprocedural neurological status, with Rankin score → 2 in 45.8% as compared to 33.3% in the 'no complications' group.

Procedures that were complicated involved embolization of fewer feeders (mean: 1.79 vs 2.47), but resulted in more nidal obliteration than uncomplicated procedures (mean: 61.5% vs 53.5%). Procedures that were associated with complications resulted in a longer duration of post-procedural hospital stay as compared to uncomplicated procedures (5.56 vs 3.31 days).

Table 5.4 : Rankin Scores* of Patients Undergoing Endovascular Brain AVM Treatment Before and After Completed Embolization Therapy

Rankin Score	Baseline		Post Embolization	
	n	%	n	%
0	-	-	-	-
1	57	63.3 %	56	62.2 %
2	28	31.1 %	25	27.8 %
3	5	5.6 %	7	7.8 %
4	-	-	-	-
5	-	-	-	-
6	-	-	2	2.2%

* Rankin 0 = no symptoms at all; 1 = able to carry out all usual duties and activities; 2 = unable to carry out all previous activities but able to look after own affairs; 3 = requiring some help but able to walk without assistance; 4 = unable to walk without assistance and unable to attend to bodily needs; 5 = bedridden, incontinent, and requiring constant nursing care and attention; and 6 = death

Table 5.5 : Comparison of AVM Embolization procedures – Without and With complications (Including major and minor technical complications)

	Without Complication	With Complication	p value
Number, n (%)	66 (73.3 %)	24 (26.7 %)	
Mean Age years	30.9	31.5	
Sex Ratio M : F	53 : 13	18 : 6	
Presentation, n (%)			
Hemorrhage	29 (43.4 %)	12 (50.0 %)	0.61
Seizures	40 (60.6 %)	14 (58.3 %)	0.84
Headache	44 (66.7 %)	16 (66.7 %)	1.00
Progressive neurol. deficits	9 (13.6 %)	5 (20.1 %)	0.29
Preprocedural Rankin Score, n (%)			
1	44 (66.7 %)	13 (54.2 %)	0.28
2	20 (30.3 %)	8 (33.3 %)	0.77
3	2 (3.0 %)	3 (12.5 %)	0.11
Eloquent Location, n (%)	59 (89.4 %)	24 (100%)	0.10
Size, n (%)			
< 3 cm	9 (13.6 %)	7 (29.2 %)	0.09
3 – 6 cm	48 (72.3 %)	12 (50.0 %)	0.04
> 6 cm	9 (13.6 %)	5 (20.1 %)	0.29
Deep venous drainage	26 (39.4 %)	7 (29.2 %)	0.26
Spetzler Martin grade			
Grade 1	0 (0 %)	0 (0 %)	1.00
Grade 2	16 (24.2 %)	7 (29.2 %)	0.64
Grade 3	30 (45.5 %)	10 (41.7 %)	0.75
Grade 4	15 (22.7 %)	4 (16.7 %)	0.53
Grade 5	5 (7.5 %)	3 (12.5 %)	0.36
Angioarchitecture			
Plexiform Nidus	18 (27.3 %)	14 (58.3 %)	0.006
Plexiform nidus + microfistulae	42 (63.6 %)	10 (41.7 %)	0.06
Macrofistulae	6 (9.1 %)	0 (0 %)	0.14
Average no. of feeders embolized	2.47	1.79	
Percentage of nidal obliteration	53.5 %	61.5 %	
Duration of post procedural hospital stay	3.31 days (Excluding 4 patients who underwent surgery in the same sitting)	5.56 days (Excluding 2 patients who died in the post procedural period)	

Intracranial Aneurysms

Among 16 patients who underwent coil embolization of saccular aneurysms, one patient with a basilar top aneurysm and severe triple vessel coronary disease and peripheral occlusive vascular disease had a massive myocardial infarction on the 3rd day after successful coil embolization of the aneurysm using remodeling technique. One other patient with a postoperative leak from a remnant anterior communicating artery aneurysm, and underwent uneventful coil embolization, died in the post procedure period due to septicemia. These patients were excluded from analysis. The details of the rest of the patients with complications during treatment of aneurysms are summarized in Table 5.6.

The clinical and angiographic details in patients without (complication outcome – 0) and with complications (complication outcome – 1 to 6) during treatment of aneurysms are summarized in Table 5.7. Three patients had intraprocedural technical complications that were successfully managed. These included one patient with occlusion of proximal segment of P1 during coil embolization of a wide necked basilar top aneurysm, one patient with a malpositioned stent during stent-assisted coiling of a cavernous ICA aneurysm and one patient who had an embolic episode during coiling, that was successfully managed by thrombolysis.

Two patients had intraprocedural rerupture of the aneurysm. One patient had bilateral posterior communicating aneurysms, of which one had ruptured. Previous attempt at surgical clipping of the aneurysm was unsuccessful. During coil embolization of the

Table 5.6: Complications in Endovascular Therapy for Intracranial Aneurysms

Patient No	Age Sex	Location	Rupture	WFNS Grade	Complication	Rankin Score*		Functional Outcome§
						Pre	Post	
1	57F	Lt PCA (P2 segm)	No	-	Memory disturbances after coil occlusion of P2 segment of PCA	1	2	3
8	44M	Rt PCA (P3 segm)	No	-	Memory disturbances after coil occlusion of P3 segment of PCA	1	2	3
15	39M	Rt Paraclinoid	No	-	Occlusion of MCA by clot – Thrombolysed	1	1	1
16	44M	Basilar Top	Yes	4	Rupture of aneurysm during coiling	4	6	6
17	45M	Basilar Top	Yes	3	Occlusion of origin of PCA – Good flow through Pcom.	1	1	1
21	55F	Cavernous ICA	No	-	Malposition of stent during attempted stent assisted coiling - retrieved	1	1	1
23	49F	Pcom aneurysm	Yes	3	Dislodgement of luminal clot and occlusion of supraclinoid ICA. Thrombolysis . MCA territory infarction	3	5	5

Table 5.7 : Clinical and Angiographic Characteristics of Complicated and Uncomplicated Procedures for endovascular management of intracranial aneurysms, in the present series

	(n)	Complication Outcome						
		0 (No Complication)	**1**	**2**	**3**	**4**	**5**	**6**
Unruptured Aneurysms								
Parent Artery Preservation	6	4	2	-	-	-	-	-
Parent Artery Occlusion	6	4	-	-	2	-	-	-
Ruptured Aneurysms								
Parent Artery Preservation	8	5	1	-	-	-	1	1
Parent Artery Occlusion	1	1	-	-	-	-	-	-
Ruptured Aneurysms								
WFNS Grade I	4	4	-	-	-	-	-	-
WFNS Grade II	2	2	-	-	-	-	-	-
WFNS Grade III	2	-	1	-	-	-	1	-
WFNS Grade IV	1	-	-	-	-	-	-	1
WFNS Grade V	0	-	-	-	-	-	-	-
Anterior Circulation								
Narrow Neck Saccular	4	2	1	-	-	-	1	-
Wide Neck Saccular	7	6	1	-	-	-	-	-
Posterior Circulation								
Narrow Neck Saccular	2	2	-	-	-	-	-	
Wide Neck Saccular	6	3	1	-	1	-	-	1
Fusiform Aneurysms	2	1	-	-	1	-	-	

aneurysm, dislodgement of intra-aneurysmal clot and embolism with occlusion of the supraclinoid segment of internal carotid artery. Lysis of the clot with 1 lakh units of urokinase was performed, along with balloon angioplasty and administration of nitroglycerin. Check angiogram revealed a small non-perfused area in the superior parietal lobule. Post procedure CT scan revealed a large hyperdense area suggesting SAH, which was actually the postoperative enhancement; which was confirmed on repeat CT after 24 hours.

After a prolonged and stormy course in the hospital, the patient was discharged in a vegetative state on tracheostomy. The second patient was a 44-year-old male patient who underwent coil embolization of a recently (5 days) ruptured wide necked basilar top aneurysm. Two Sentry occlusion balloons were placed across the neck of the aneurysm to remodel the neck, and the aneurysm was partially coiled. Check angiogram after deployment of the first coil revealed non-filling of the distal basilar and proximal left posterior cerebral artery. The balloon placed in the left posterior cerebral artery was withdrawn. Check angiogram revealed leak from the aneurysm sac. Despite immediate reversal of heparinization and expeditious coiling of the aneurysm sac, the patient had a massive subarachnoid bleed and succumbed to it.

Among 7 patients who underwent parent artery occlusion for intracranial aneurysms, two patients had complications. Both patients had aneurysm from the P2 (n=1) or P3 (n=1) segments of the posterior cerebral artery. Subsequent to coil occlusion of the aneurismal sac along with the parent artery, both patients developed memory disturbances

Table 5.8 : Comparison of Complications in the present series with ASITN Thresholds (Adapted from: ASITN Data, AJNR Sep 2001)

		Unruptured Aneurysms				Ruptured Aneurysms			
		Parent artery occlusion		Parent artery sparing		Parent artery occlusion		Parent artery sparing	
		ASITN Thresholds	Present Study	ASITN Thresholds	Present Study	ASITN Thresholds	Present Study	ASITN Thresholds	Present Study
Death in Hospital	Grade I	1	0	2	0	5	0	5	0
	Grade II					15	-	15	0
	Grade III					40	-	40	0
	Grade IV					60	-	60	100
	Grade V					75	-	75	-
All Neurological complications		10	2	8	0	8	0	8	25
Permanent Neurological complications		5	2	3	0	3	0	3	25
Inadvertent vessel occlusion		N/A	N/A	8	0	N/A	N/A	8	0
Vessel perforation		0.5	0	2	0	0.5	0	2	0
Aneurysm rupture during procedure		1	0	1	0	5	0	5	25
Aneurysm rupture after procedure		2	0	2	0	5	0	5	0

(anterograde amnesia) despite angiographic demonstration of adequate perfusion of posterior cerebral territory from middle cerebral artery pial collaterals.

The incidence of complications in ruptured aneurysms (3 complications in 9 patients) was similar to that in unruptured aneurysms (4 complications in 12 patients). Among patients with ruptured aneurysms, patients with higher WFNS grades had a higher rate of complications (Table 5.7). The complications in the present series are compared to the ASITN thresholds in Tables 5.8.

Intracranial Arteriovenous Fistulae

Among the 7 patients with cerebral arteriovenous fistulae that were treated in 8 procedures during the study period, two patients with vein of Galen malformations had intraventricular hemorrhage during the periprocedural period. The first patient was a 28-year-old man who had history of recurrent headaches since childhood and presented with intraventricular hemorrhage. Angiographic evaluation revealed a choroidal type of malformation. The venous sac was partially coiled using GDC coils to achieve flow reduction. One of the arterial feeders was kept catheterized for embolization using glue. Extravasation of contrast from one of the feeders was detected. This was immediately sealed off using cyanoacrylate glue. A post procedure CT scan was performed, which revealed extensive intraventricular and subarachnoid hyperdensities, which were due to leaked blood mixed with contrast medium. External ventricular drainage was performed immediately, but the patient succumbed to the bleed 3 days later. The second patient was a 3-year-old child who presented with hydrocephalus. He had undergone ventriculo-peritoneal shunt placement at the age of one year. Angiographic evaluation revealed a

mural type of malformation fed by posterior thalamoperforator and thalamogeniculate branches. Initially, using a transarterial access, one of the feeders was cannulated and a Berenstein liquid coil (Target Therapeutics, Fremont, CA) was released to reduce the flow through the fistula and prevent the migration of the glue to venous side. Subsequently, through a femoral venous access, the venous sac was cannulated and 7 vein of Galen coils (Target Therapeutics, Fremont, CA) were deployed. Then the thalamogeniculate branch feeding the fistula was cannulated and embolized using 15% Histoacryl. The patient was electively ventilated after the procedure. About 3 hours after the procedure, the child developed pupillary asymmetry. CT scan of the brain revealed extensive intraventricular and subarachnoid bleed and ventriculomegaly. In spite of immediate placement of external ventricular drainage and institution of barbiturate coma, the patient succumbed to the bleed 2 days later.

One other child with a non-Galenic cerebral arteriovenous malformation was embolized in two sittings using detachable coils and glue. During the first sitting, during deployment of GDC coils in the venous sac, there was non-detachment of the GDC coil. Manual rotation of the delivery wire resulted in fracture of the wire. Subsequently, a mechanically detachable coil (Cook) was negotiated into the venous sac. Fracture of the delivery wire occurred during attempted detachment of this coil also. Both coils were not retrieved, as they did not limit the flow in the cerebral circulation.

Four other procedures of cerebral arteriovenous fistula embolization were uneventful.

Carotico-cavernous Fistulae

Among 16 patients with carotico-cavernous fistulae who were treated in 20 endovascular procedures during the study period, 1 patient (6.25 %) had a technical complication. This patient had a traumatic Type A fistula, which showed persistent filling despite detachment of two detachable balloons within the venous sac. Attempts at cannulation of the inferior petrosal sinuses were unsuccessful. So coiling of the venous sac using GDC and mechanically detachable coils (Cook, USA) was performed from the arterial route. The last coil did not detach despite repeated attempts. So the delivery wire was cut at the level of the groin. The patient did not suffer from any morbidity secondary to this event, and remains asymptomatic on follow up.

In two patients, unfavorable architecture prevented adequate access to the site of fistula and therefore embolization could not be performed. However, there were no procedure related complications in these patients.

Dural Arteriovenous Malformations

Six patients with dural arteriovenous malformations were treated in 10 sessions.

Nine procedures were uncomplicated. One patient with a type IV dural arteriovenous fistula had intracerebral hemorrhage that complicated the embolization procedure. This 46-year-old hypertensive male patient underwent partial embolization of the arterial feeders in 1990 and subsequently in 2 sittings during the study period. However, persistence of the high flow shunt resulted features of cerebral venous hypertension. At the time of last embolization, the patient was bed bound, with slow cognition, impaired comprehension and reduced fluency. DSA had revealed occlusion of the left sigmoid

sinus and internal jugular vein and a type IV dural AV fistula draining into the right transverse and sigmoid sinuses. Embolization of the venous sac through a burr hole access, using 5ml of 50% cyanoacrylate glue and subsequent embolization of the three arterial feeders with glue was performed, resulting in about 90% reduction of flow. Post procedural CT scan revealed persistence of hydrocephalus, which was managed by external ventricular drainage. Despite attention to all the metabolic complications he developed, the patient continued to deteriorate and expired on the 3rd day after embolization.

Craniofacial Vascular Malformations

Among the 24 patients who underwent 24 embolization procedures, that were performed to devascularize craniofacial vascular malformations, 23 procedures could be performed without any periprocedural adverse event. One child with a sinus pericranii underwent percutaneous direct puncture injection of cyanoacrylate glue. This child had medication (Inj. Serenace) related dystonia during the post-procedural period. The child showed immediate improvement with symptomatic treatment. There were no procedure related neurological deficits in any patient.

Head and Neck Tumors

Among the 24 patients who underwent 24 procedures of head and neck tumor embolization, there were 2 complications. The first patient was a 20-year-old male patient with a recurrent hyper-vascular thyroid mass, with retrosternal extension. Embolization was advised prior to subtotal thyroidectomy. Angiographic evaluation revealed abundant supply from superior thyroid arteries from both sides. Embolization of the lesion was

performed after distal cannulation of the involved arteries with a 4F catheter, using 250-355μ, 355-500μ and 500-710μ PVA particles – resulting in total devascularization of the lesion. Towards the end of the procedure, the patient progressively became disoriented. Clinical evaluation revealed aphasia and right-sided hemiplegia. Immediate check angiogram showed a perfusion defect in the left posterior frontal – parietal regions. Post procedure CT scan revealed an infarct in the affected regions. Subsequent management in the immediate post procedure period with heparin and mannitol and subsequently with physiotherapy and speech therapy helped the patient recover markedly within 2 weeks. He had residual mild facial palsy and had limitation of naming. Word output, comprehension, repetition, reading and writing and memory were normal. He subsequently underwent successful resection of the tumor.

The 2nd patient was an 11-year-old male patient with a hyper vascular right sided hard palate mass fed by terminal branches from the internal maxillary artery. Preprocedural internal maxillary injection did not reveal any "dangerous anastomoses". Since the vessels supplying the lesion were of small caliber, small sized PVA particles (<106μ and 106 - 212μ) were used to embolize the lesion. In the post procedure period, the patient complained deterioration of vision in the right eye, on the same side as the embolization. Check angiography revealed stasis in the ophthalmic artery and complete occlusion of the external carotid artery. Thrombolysis using 5 lakh units of urokinase resulted in opening up of all external carotid branches, but the patient had residual dimness of vision and could perceive hand movements at 3 feet. Subsequent ophthalmological examination revealed features of central retinal artery occlusion.

Three other patients had dissection of one of the feeder arteries during attempted distal cannulation of extremely tortuous feeders (n=2) or manipulation of microguidewire (n=1). These patients remained asymptomatic and since they were being embolized in the preoperative setting, occurrence of this technical complication aided in further devascularizing the lesion.

Excluding 12 patients who were operated in the same sitting as embolization, the mean duration of post-procedural hospital stay in the 10 uncomplicated procedures was 1.4 days, while in the complicated procedures was 4 days.

Carotid and Subclavian Angioplasty and Stenting

One patient with severe triple vessel disease and significant carotid stenosis underwent carotid artery stenting as a preoperative procedure before a coronary bypass surgery. He developed a massive myocardial infarction, a few hours after the procedure and succumbed to it. This patient was excluded from analysis.

Two of the other 16 patients who underwent revascularization procedures, had procedure related complications. Both these patients had stenotic lesions of the 1st part of the subclavian artery. One patient had a retrograde dissection of the artery during a failed attempt at angioplasty from transfemoral and transaxillary routes. One other patient with a right subclavian stenosis underwent successful angioplasty and stenting. Subsequent check angiograms revealed occlusion of the parieto-occipital branch of the left middle cerebral artery, which was presumed to be thromboembolic origin. The patient was disoriented and developed global aphasia without any limb weakness. Thrombolysis of the lesion was performed using 7.5 lakh units of urokinase. The patient recovered completely within 24 hours.

None of the patients who underwent carotid stenting had any procedure related neurological deficits.

Percutaneous Vertebroplasty & Other Percutaneous Vertebral Interventions

Minor complications were associated with 11 (28.9%) of the 38 vertebroplasty procedures. In one patient with collapse of D12 vertebra experienced a transient worsening of lower limb power after vertebroplasty. Immediate decompressive laminectomy was performed, and resulted in complete recovery of motor function. All other complications were intraprocedural minor technical complications and did not cause deterioration of the neurological status in any patient (complication outcome - 1). Analysis of the occurrence of these technical complications revealed no statistically significant correlation with the vertebral level of the procedure ($p=0.769$), underlying pathological substrate ($p=0.233$), volume of cement injected ($p=0.408$) or percentage of filling of vertebra. ($p=0.518$) (Tables 5.9 – 5.12)

Both procedures of percutaneous injection of embolic agent into posterior elements involved by tumor were uneventful. There were no procedure related technical complications or post procedural neurological deficits.

Vertebral Embolizations:

Of the five patients who underwent transarterial embolization of hypervascular vertebral body tumors, none had any procedure related technical complications or deterioration of neurological status. Apart from one patient who was operated in the same sitting as the embolization, rest of the patients were discharged at an average of 2.5 days after the procedure.

Table 5.9: Percutaneous Vertebroplasty - Nature of complications

Individual complications	No of procedures	%
Prevertebral venous filling	3	7.9
Paravertebral venous filling	3	7.9
Prevertebral soft tissue extravasation	0	0
Paravertebral soft tissue extravasation	4	10.5
Epidural leak	2	5.3
Disc space filling	3	7.9
Injury to cord	0	0
Pain during injection	1	2.6
Needle track and subcutaneous hematoma	0	0
Local infection	0	0
Pulmonary embolism	0	0
Systemic embolism	0	0

Table 5.10: Percutaneous Vertebroplasty - Correlation of occurrence of complication with level of the procedure

Level		No of procedures	
		Complication	No complication
D1 - D6	no. (%)	2 (40 %)	3 (60 %)
D7 - D12	no. (%)	6 (70 %)	14 (30 %)
L1 - L5	no. (%)	3 (23.1 %)	10 (76.9 %)

Table 5.11: Percutaneous Vertebroplasty - Correlation of occurrence of complications in different pathologic groups:

Lesion type	No of procedures	
	Complication	No complication
Osteoporotic collapse (n=15)	10 (66.7%)	5 (33.3%)
Hemangioma (n=17)	11 (64.7%)	6 (35.3%)
Tumor (n=6)	6 (100%)	0

Table 5.12: Percutaneous Vertebroplasty - Correlation of occurrence of complication with volume of cement injected:

Complication	No	Vol. of cement injected (ml)		Percentage filling of vertebra	
		Mean (ml)	Std. error mean	Mean (%)	Std. Error mean
No Complication	27	6.07 (± 2.57)	0.495	86.3 (± 9.67)	1.861
Complication	11	5.36 (± 1.75)	0.527	83.9 (± 11.5)	3.478

Spinal Vascular Malformations

Among the 13 procedures of attempted spinal vascular malformation embolization that were performed during the study period, 3 patients had procedure related complications. Two of these were minor technical complications that did not alter the management of the patient in any way. Two patients were young individuals with low flow spinal vascular malformations (cord AVM (n=1) and perimedullary fistula (n=1)), which had small caliber feeding arteries. Attempts at catheterization of the feeder to a safe location for embolization resulted in spasm of the artery. Relief of the spasm was achieved by infusion of nitroglycerin and followed by embolization in one patient, while this led to abandonment of the procedure in the other. Both patients did not suffer any neurological deficits related to these events (complication outcome– 1).

One patient with a spinal perimedullary fistula at the level of the first lumbar artery had a long feeder artery, that originated from the 8^{th} intercostal artery, took an acute hairpin bend at the level of the 5^{th} lumbar vertebra and ascended up to the level of the fistula. Super selective catheterization of the feeder and embolization of the feeder resulted in deterioration of the patient's lower limb power from grade 3 to grade 1. With prolonged stay in the hospital that was complicated by bedsores and urinary tract infection, and physiotherapy, the power in the lower limb mildly improved to grade 2 at discharge.

Intracranial Thrombolysis for Procedure Related Complications

Among the 10 procedures of intra-arterial thrombolysis, two patients had procedure related complications. The first patient was 78-year-old male patient in whom; coronary angiography resulted in embolic occlusion of the basilar artery, posterior cerebral and superior cerebellar arteries. Thrombolysis with 5 lakh units of urokinase resulted in

complete opening up of the vessel within 4 hours of onset of symptoms, but post procedure CT scan showed hemorrhagic conversion of multiple infarcts in the territories of both posterior cerebral arteries and left posterior inferior cerebellar artery. However, there were no further deficits as compared to the pre procedure neurological status.

The second patient was a 49-year-old female patient who had undergone unsuccessful attempt at clipping of a partially thrombosed posterior communicating aneurysm. During coil embolization of the aneurysm, the intraluminal thrombus was dislodged and caused embolic occlusion of the main trunk of the middle cerebral artery. Subsequent thrombolysis using 1 lakh units of urokinase and angioplasty using a 2mm balloon resulted in reopening of the vessel. Check angiogram after thrombolysis revealed a small perfusion defect in the superior parietal region. After a prolonged and stormy course in the hospital, the patient was discharged in a vegetative state on tracheostomy.

The thrombolytic procedures in all other patients, (including embolism complicating coronary angiography (n=2), intracranial aneurysm coiling (n=1), intracranial AVM embolization (n=2), craniofacial tumor embolization (n=1), carotid stenting (n=1) and subclavian stenting (n=1)) were uncomplicated

Temporary Balloon Occlusion Test

Of the six patients who underwent temporary balloon occlusion test during the study period, four tolerated the procedure well, while two other patients had features of cerebral ischemia, which manifested as transient disorientation that resolved completely, soon after deflation of the balloon. There were no procedure related complications, either at the site of balloon occlusion or due to thrombo-embolic phenomena. (Table 5.14)

Table 5.14 : Complication thresholds for Carotid Balloon Occlusion Test

(Adapted from: ASITN Data, AJNR Sep 2001)

Indicator	Threshold (%)	Present Study (%)
Asymptomatic arterial dissection	4	0
Transient Neurological Deficits Persisting after balloon deflation	5	0
Permanent Neurological Deficits	5	0
Death	0	0

Miscellaneous Neurointerventional Procedures

Of 20 miscellaneous neurointerventional procedures, 15 procedures were not associated with any complications. These included intracranial thrombolysis for cerebral venous thrombosis (n=4), extracranial arteriovenous fistulae (n=4), intracranial stenting for aneurysms / stenotic lesions (n=1), stenting for aneurysms of brachiocephalic vessels (n=1), chemical angioplasty for vasospasm (3) and intravascular foreign body retrieval in cranial circulation (n=2).

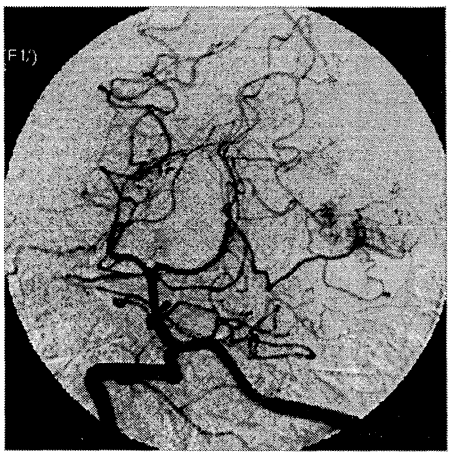
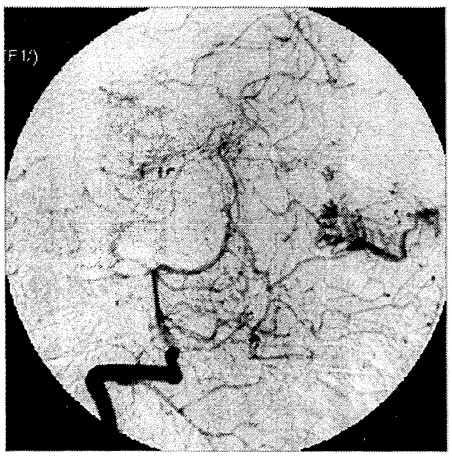
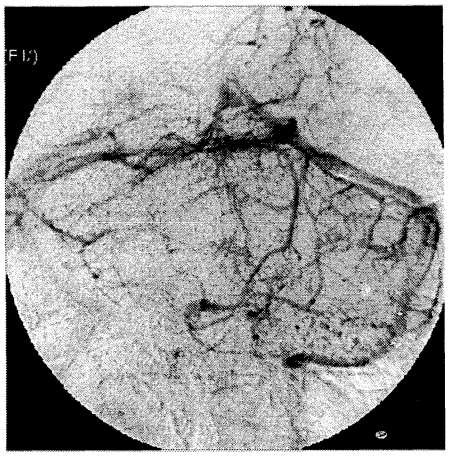
Among 4 patients who underwent stenting of intracranial vessels, 3 had technical complications. In 1 patient with a wide necked cavernous ICA aneurysm, the stent could not be negotiated into its intended location and was lying loosely within the proximal larger caliber artery. This had to be captured using the stent deployment balloon and with drawing the assembly as a whole into the abdominal aorta. A contralateral femoral puncture, a large caliber (8F) sheath and a wire loop was used to retrieve the malpositioned stent. One other patient underwent stenting across the neck of a saphenous vein graft – MCA junction aneurysm. However, due to lack of support from the venous walls, the stent slipped back and had to be repositioned. An the 3rd patient, rapid balloon inflation during deployment of the stent caused rupture of the cavernous ICA and resulted in the formation of a carotico-cavernous fistula. This was subsequently embolized using coils from transvenous access. The patient did not have any major neurological deficits except diplopia due to lateral rectus palsy.

Two patients who underwent stenting of the brachiocephalic vessels had complications . The first patient who had stent graft placement to treat a traumatic pseudoaneurysm of the

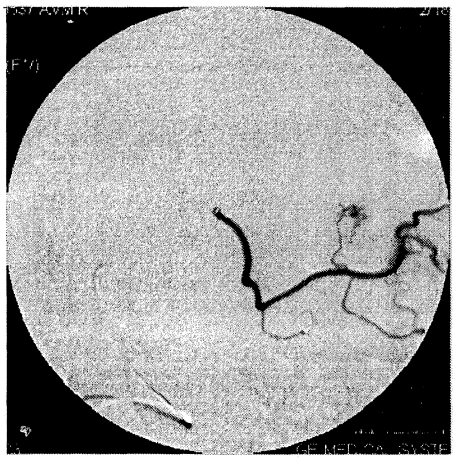
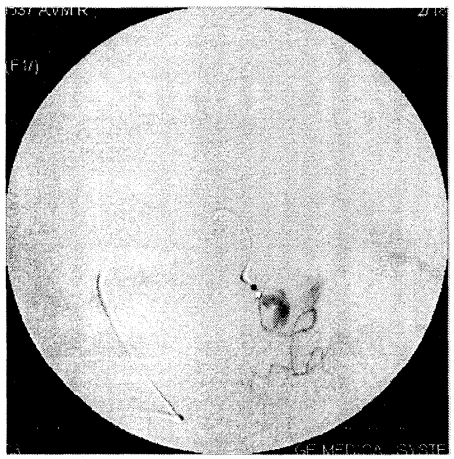
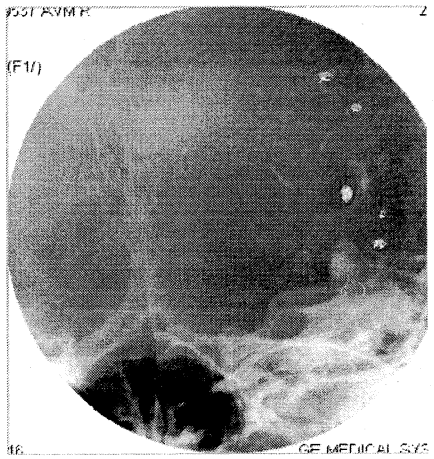
cervical internal carotid artery had embolic occlusion of the middle cerebral artery. This was thrombolysed and did not result in any neurological deficits. The second patient had embolic complications during placement of a stent for a pseudoaneurysm of the subclavian artery and succumbed to them.

6. ILLUSTRATIVE CASES

ILLUSTRATIVE CASE No: 1

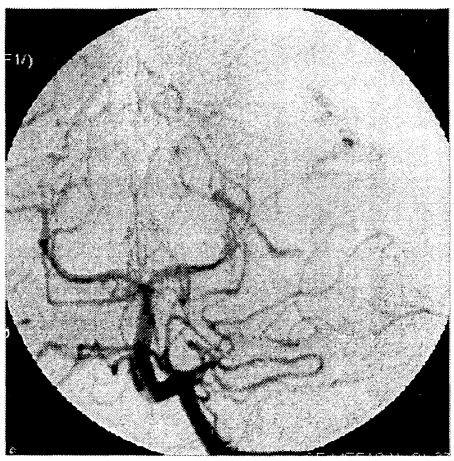
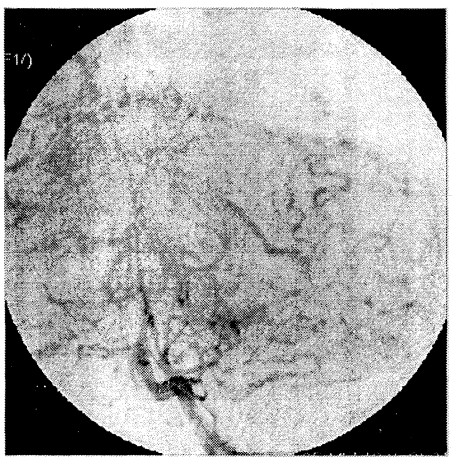
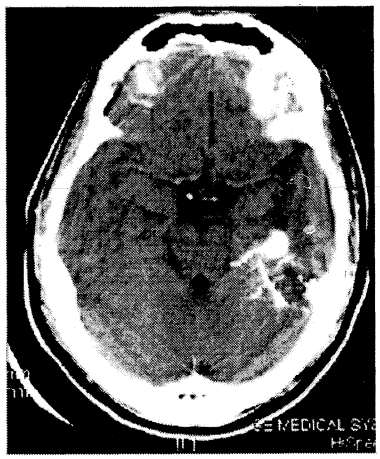
DIAGNOSTIC ANGIOGRAM

FEEDER ANGIOGRAM

FEEDER PERFORATION DURING CATHETERIZATION

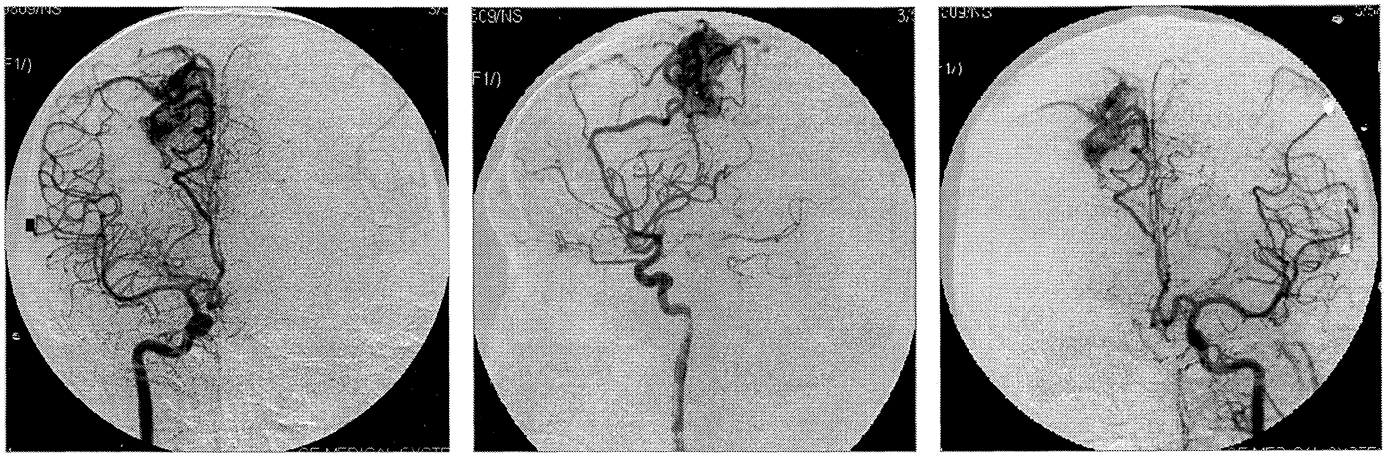
POST EMBOLIZATION

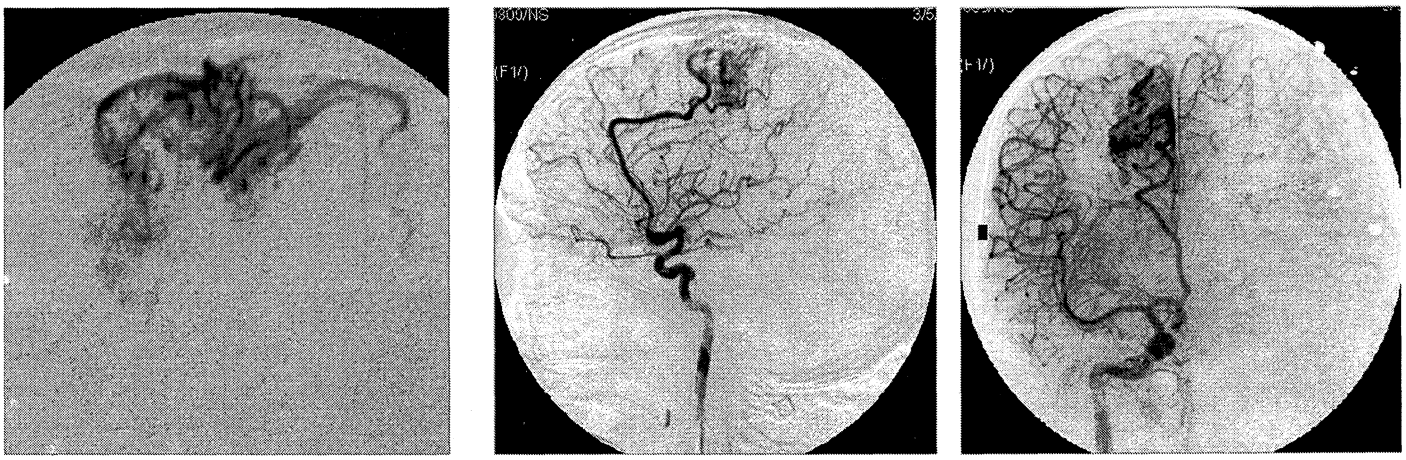
CHECK ANGIOGRAM

POST PROCEDURE CT SCAN

ILLUSTRATIVE CASE No: 2

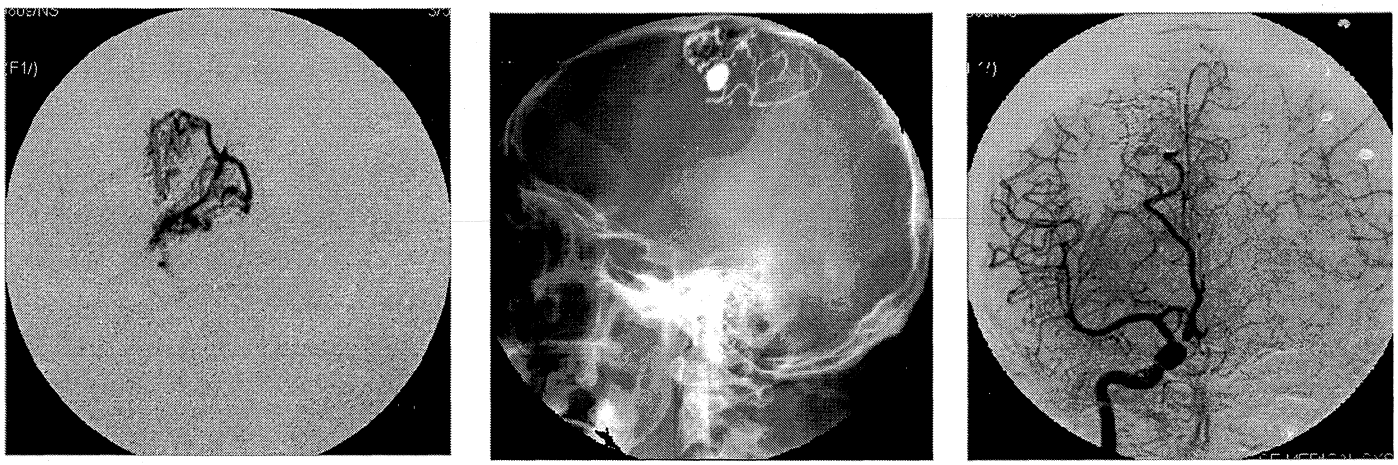


DIAGNOSTIC ANGIOGRAM



FIRST FEEDER

POST EMBOLIZATION - FIRST FEEDER

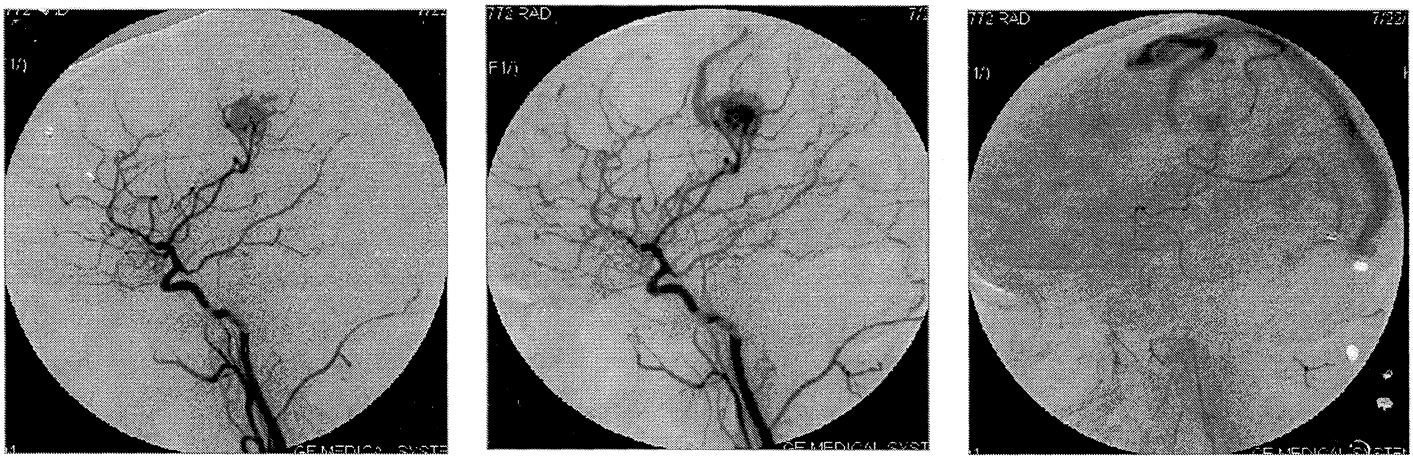


SECOND FEEDER

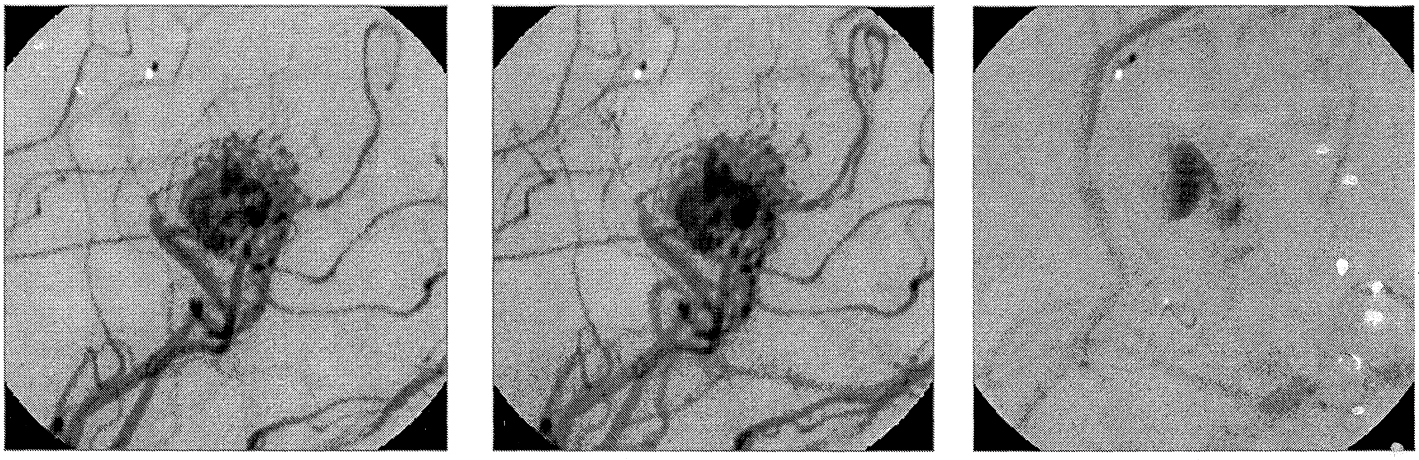
FEEDER PERFORATION DURING GLUE INJECTION

CHECK ANGIOGRAM

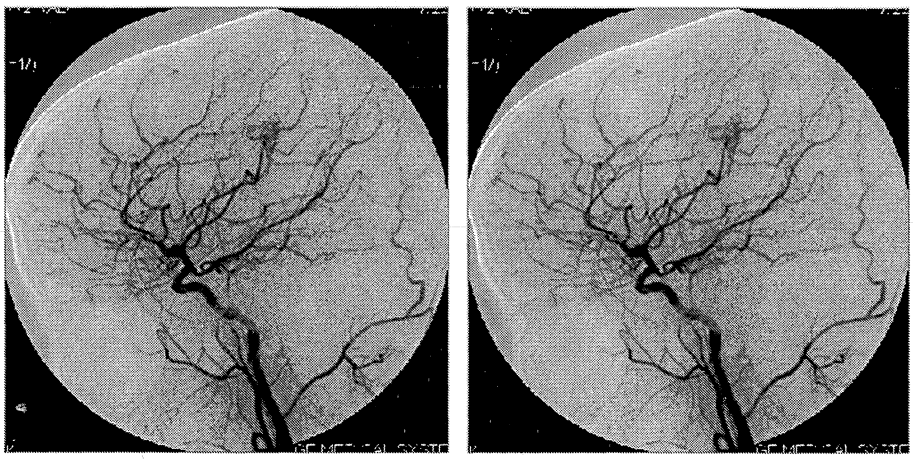
ILLUSTRATIVE CASE No: 3



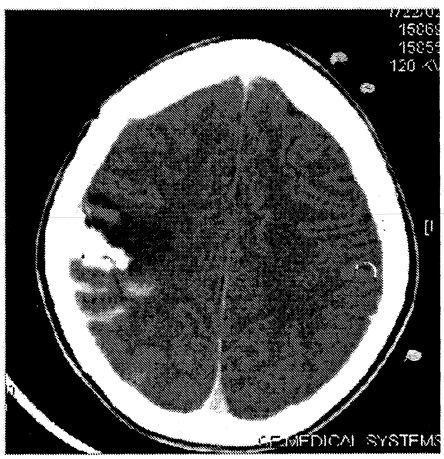
DIAGNOSTIC ANGIOGRAM



RUPTURE OF NIDUS DURING SUPERSELECTIVE CATHETERIZATION

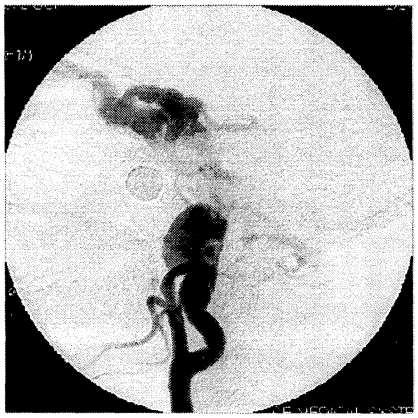
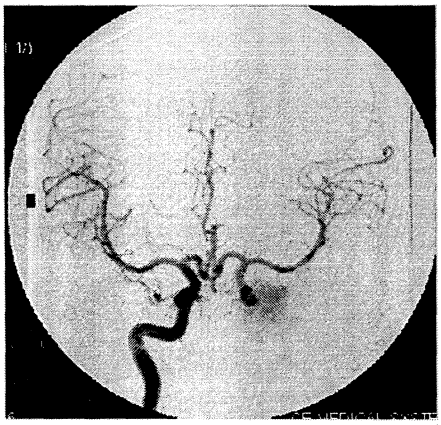


CHECK ANGIOGRAM

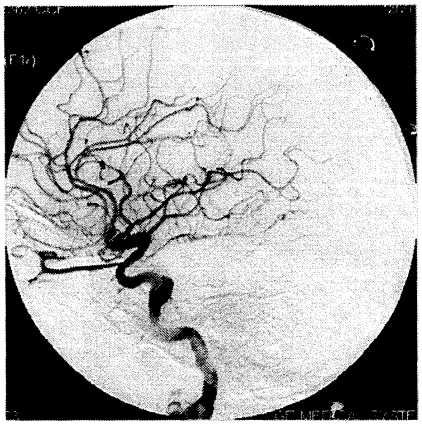


POST PROCEDURE
CT SCAN

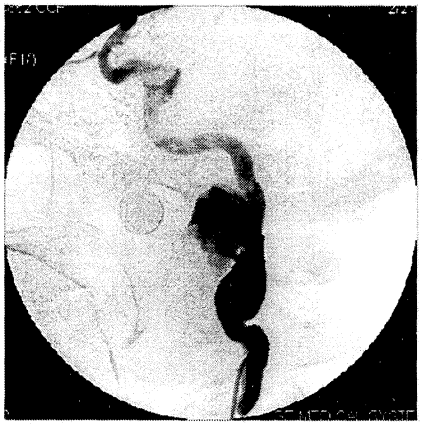
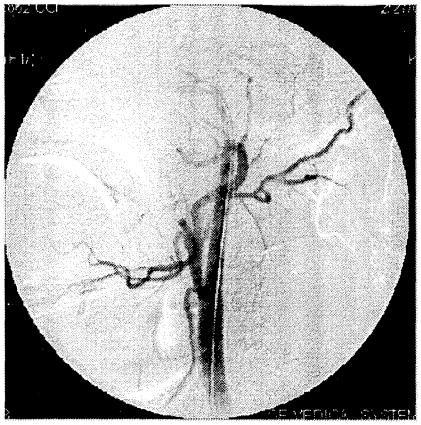
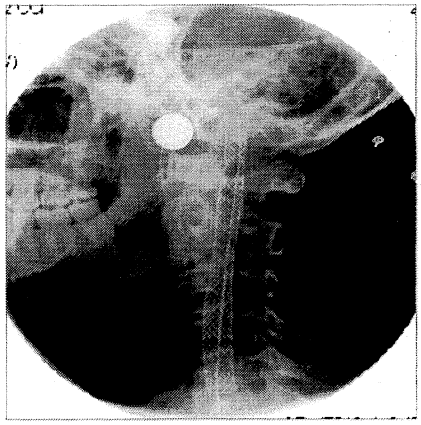
ILLUSTRATIVE CASE No: 4

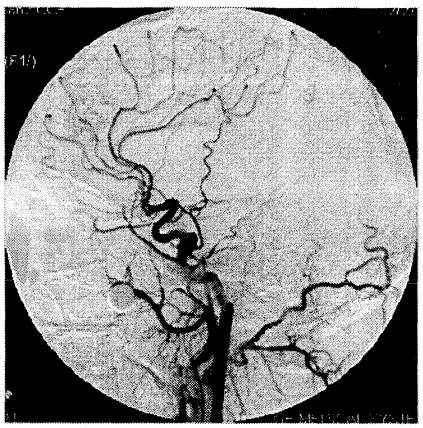
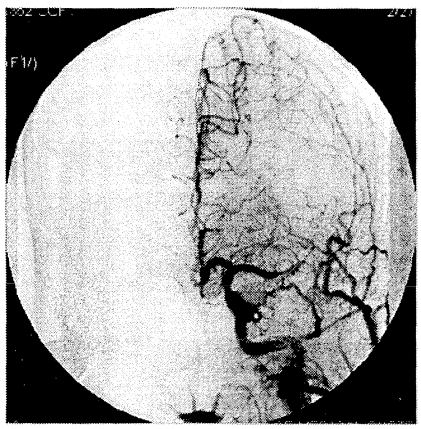
DIAGNOSTIC ANGIOGRAM



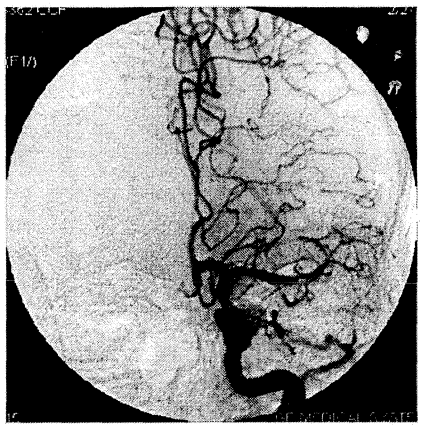
BALLOON EMBOLIZATIO

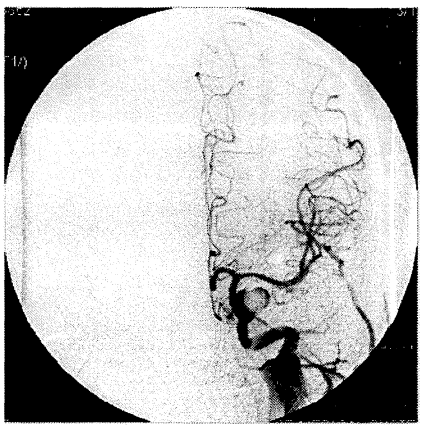
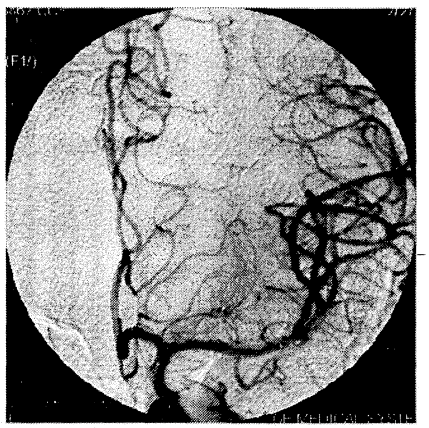
STENT GRAFTING OF CERVICAL ICA ANEURYSM

EMBOLIC OCCLUSION OF MCA

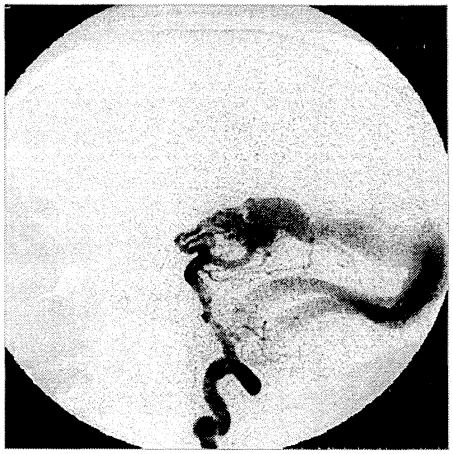


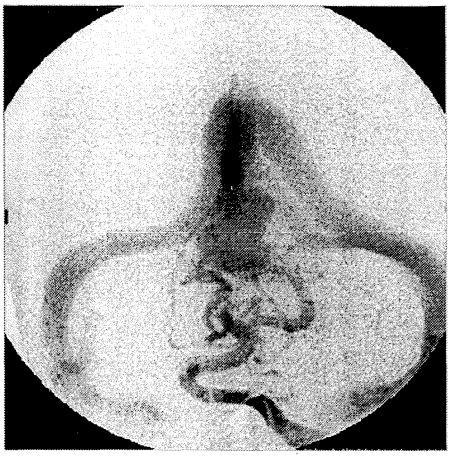
THROMBOLYSIS

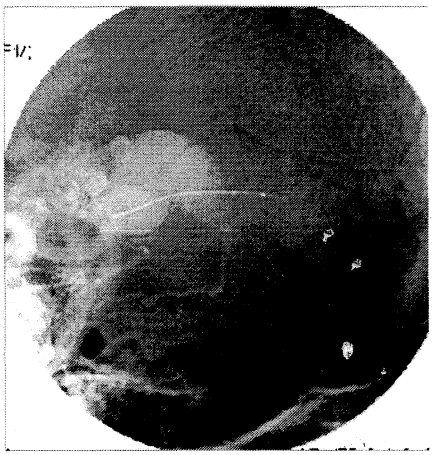
POST
THROMBOLYSIS

ILLUSTRATIVE CASE No: 5

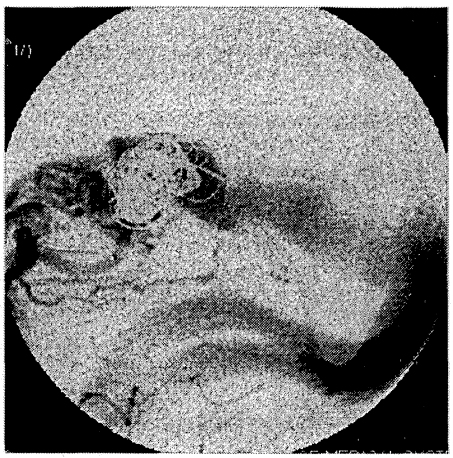




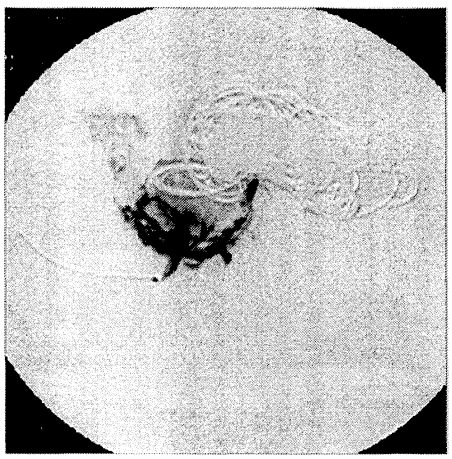
DIAGNOSTIC ANGIOGRAM

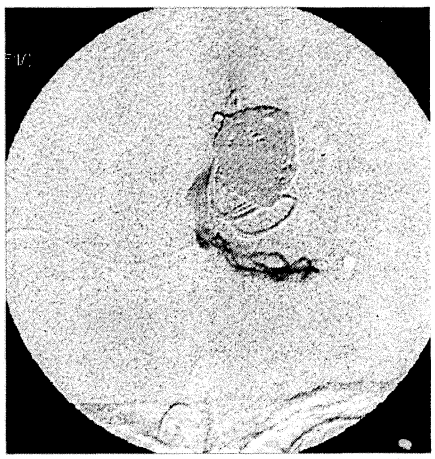


COIL EMBOLIZATIO
OF VENOUS SAC

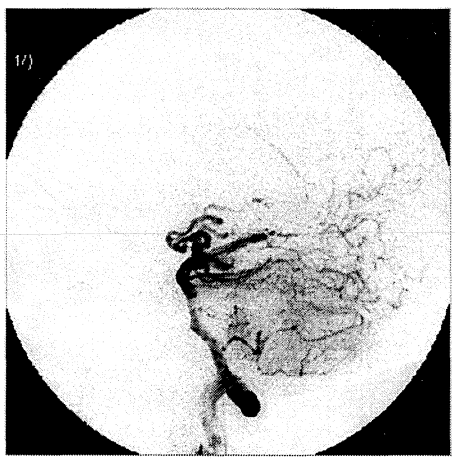


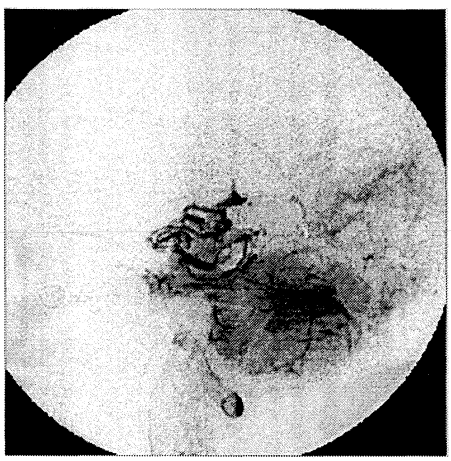
POST - COIL
EMBOLIZATION



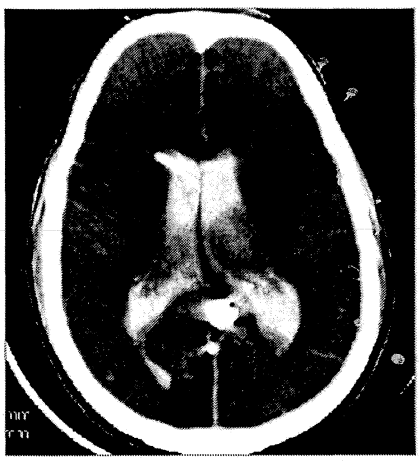


CONTRAST EXTRAVASATION
DURING FEEDER CATHETERIZATION





CHECK ANGIOGRAM



POST PROCEDURE
CT SCAN

ILLUSTRATIVE CASE No: 6

ILLUSTRATIVE CASE No. 6:

HISTORY:

23 year old male patient with history of headache, proptosis and tinnitus following a road traffic accident. Angiography revealed a Type A high flow right sided caroticocavernous fistula

PROCEDURE:

Balloon embolization of the fistula through arterial route was attempted initially, but fistula was filling despite placement of two detachable balloons. Coil embolization of the venous sac through arterial route was performed, which resulted in near total occlusion of the fistula.

COMPLICATION:

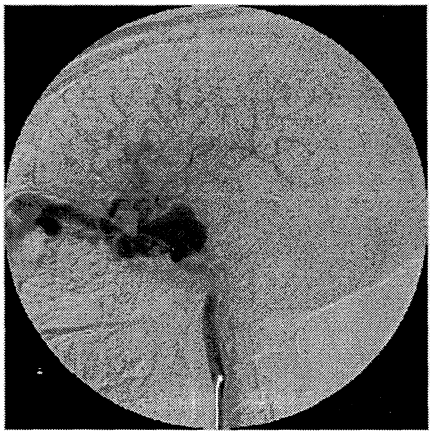
Failure of detachment of the last mechanically detachable coil (Cook, USA)

MANAGEMENT OF COMPLICATION:

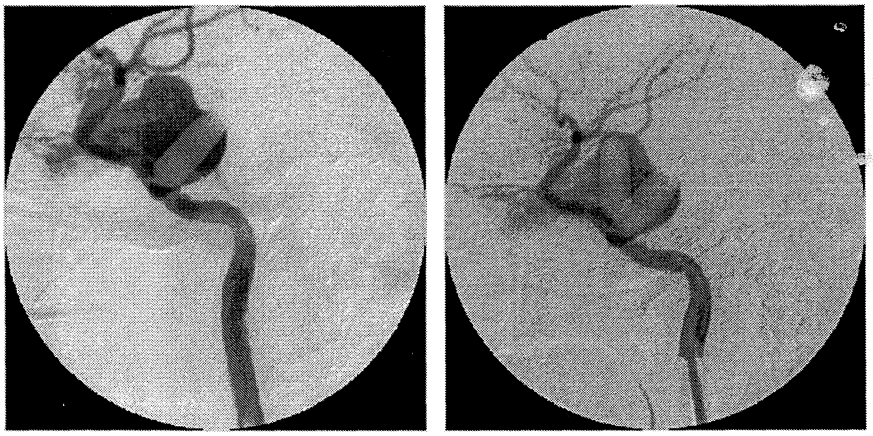
Repeated attempts at mechanical detachment failed. Attempts at withdrawl of the coil also were unsuccessful, and were resulting in movement of the whole coil mass.
The delivery wire of the coil was stretched, was cut at the groin level, and was allowed to retract into the iliac artery. The patient was put on antiplatelet agents in the post procedure period

FOLLOW UP & OUTCOME:

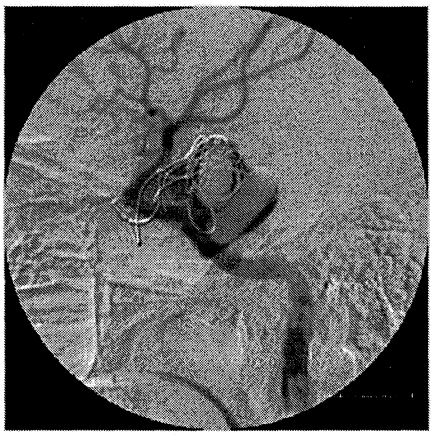
No procedure related complications. Patient asymptomatic on follow up

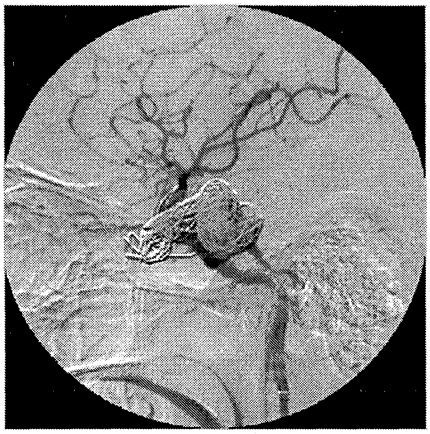


DIAGNOSTIC ANGIOGRAM

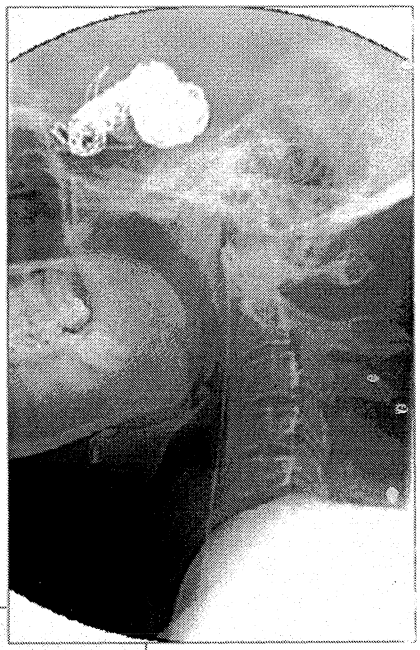


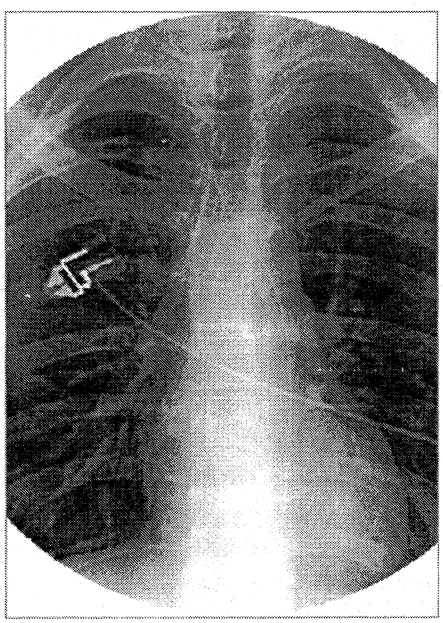
BALLOON EMBOLIZATION

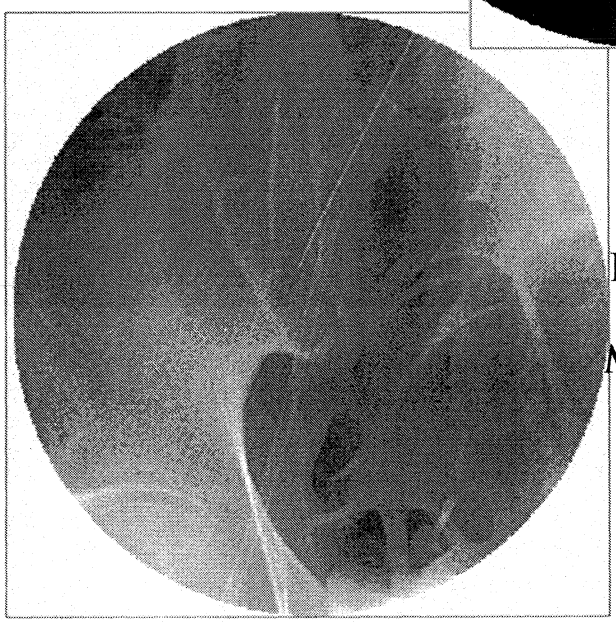




TRANS-ARTERIAL
COIL EMBOLIZATION



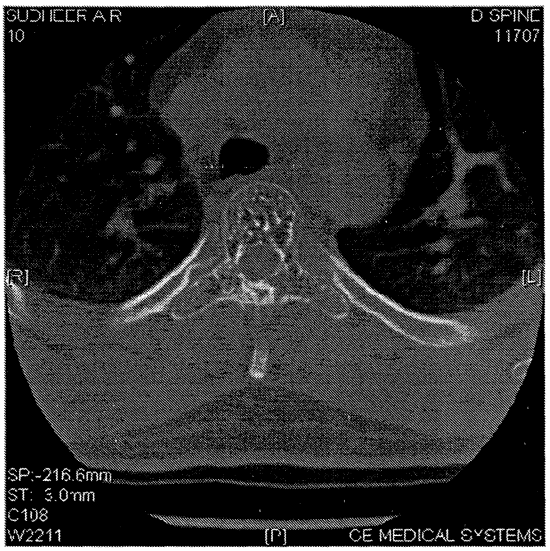




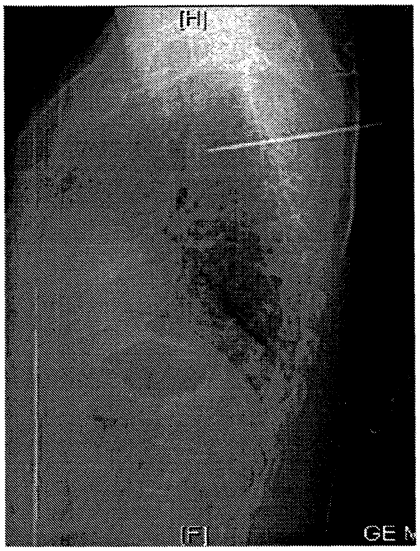
RETAINED
DELIVERY WIRE
OF
MECHANICALLY
DETACHABLE
COIL SYSTEM

ILLUSTRATIVE CASE No: 7

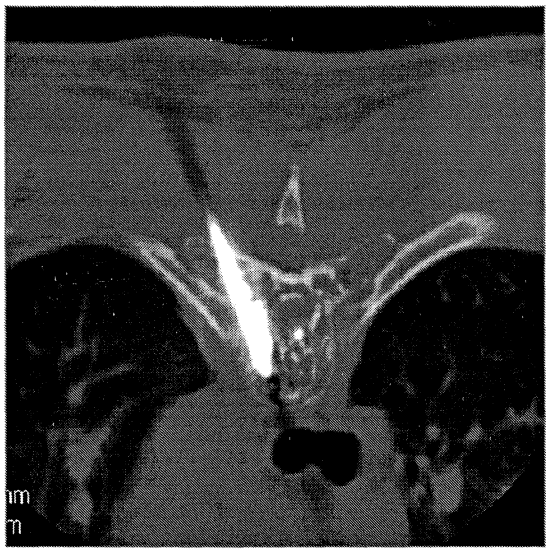
ILLUSTRATIVE CASE No: 7

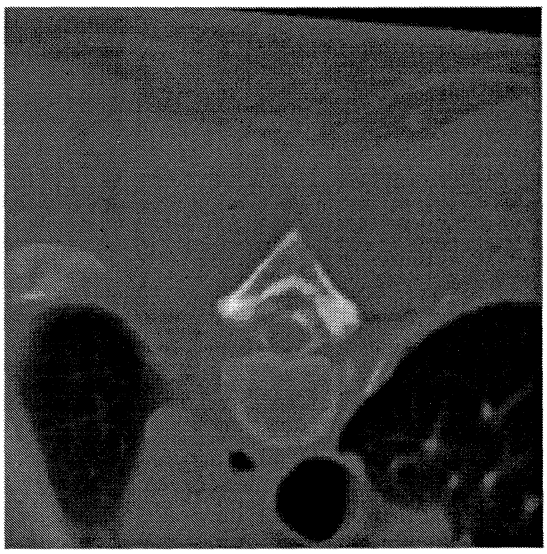


PRE-PROCEDURE
CT SCAN

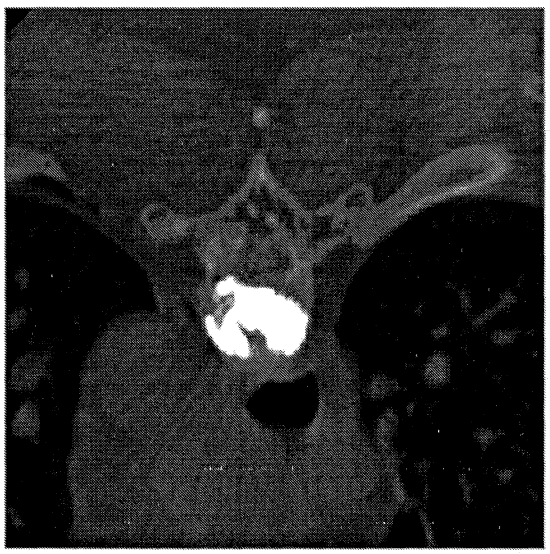


PERCUTANEOUS
VERTEBROPLASTY





CT SCAN DURING INJECTION OF CONTRAST MEDIUM
THROUGH VERTEBROPLASTY NEEDLE



POST PROCEDURE
CT SCAN

ILLUSTRATIVE CASE No: 8

ILLUSTRATIVE CASE No. 8:

HISTORY:

58 year old female patient with history of proptosis and cheimosis of right eye after a road traffic accident. Imaging and Angiographic studies performed elsewhere were suggestive of a Type A caroticocavernous fistula. Preprocedural angiogram before a planned balloon occlusion of CCF revealed spontaneous closure of the CCF with a residual cavernous ICA pseudoaneurysm

PROCEDURE:

Coil embolization of the wide necked cavernous ICA pseudoaneurysm. Towards the later part of the coil embolization, the coils were seen to prolapse into the parent artery lumen. A bare stent was planned to be deployed across the neck of the aneurysm.

COMPLICATION:

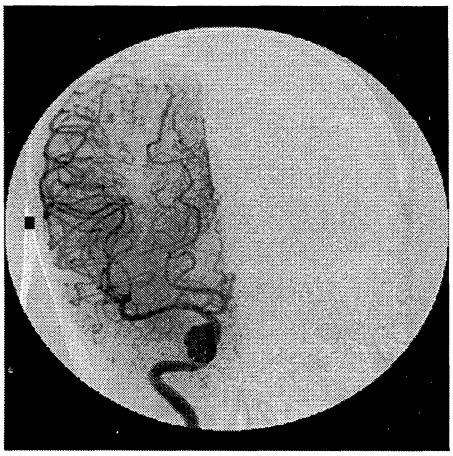
There was proximal migration of the stent during deployment, due to tight bend in the cavernous segment of ICA. The stent was lying loose in the precavernous ICA.

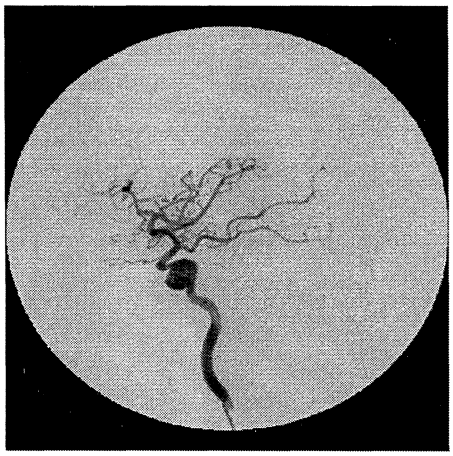
MANAGEMENT OF COMPLICATION:

The malpositioned stent was recaptured using the stent deployment balloon, and the stent - balloon – guidecatheter assembly was withdrawn as a unit into the abdominal aorta. Subsequently, the stent was retrieved using a vascular snare through a contralateral femoral approach.

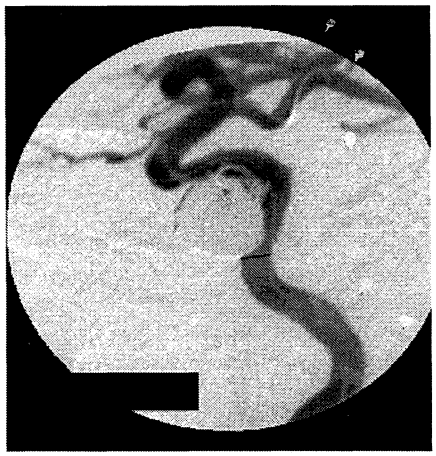
FOLLOW UP & OUTCOME:

No procedure related complications. Patient asymptomatic on follow up

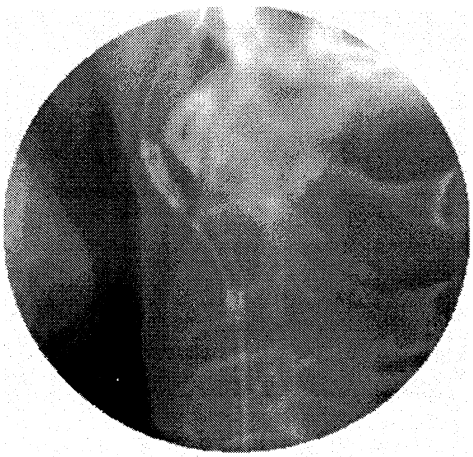


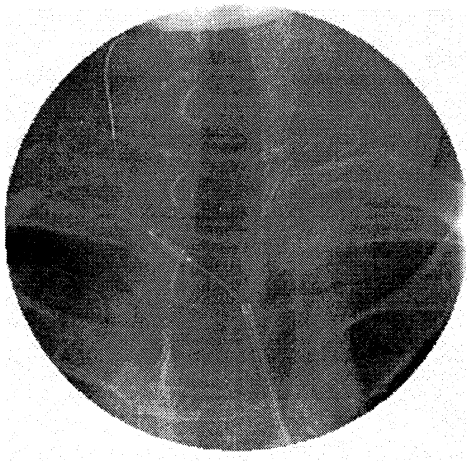


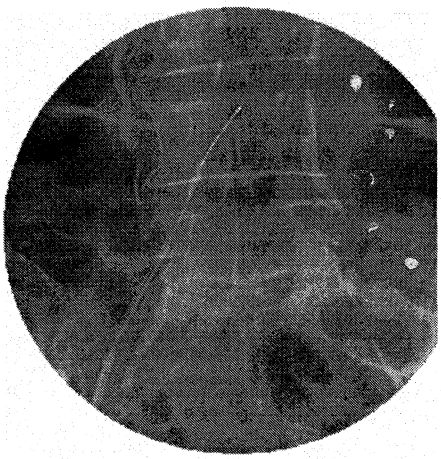
DIAGNOSTIC ANGIOGRAM



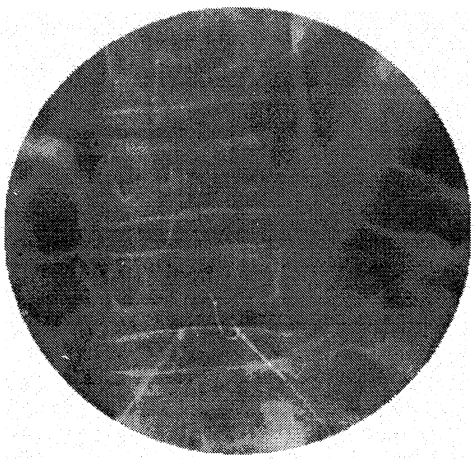
PROLAPSE OF COILS
INTO PARENT ARTER

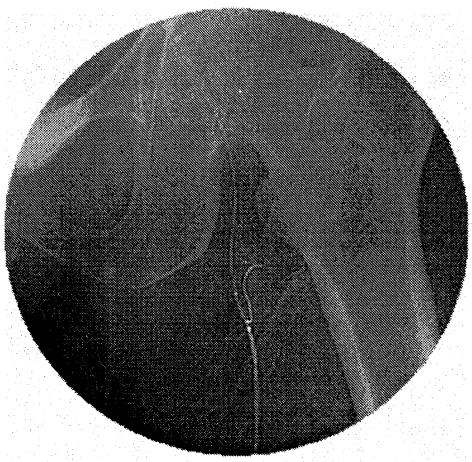




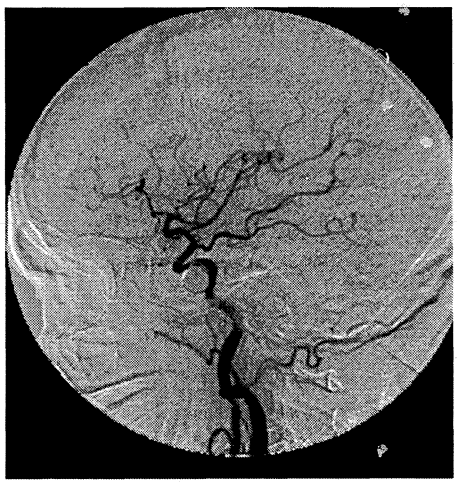


CAPTURE OF MALPOSITIONED STENT AND
NAVIGATION INTO ABDOMINAL AORTA





RETRIEVAL OF STENT THROUGH
CONTRALATERAL FEMORAL PUNCTURE



CHECK
ANGIOGRAM

7. DISCUSSION

DISCUSSION

Complications during endovascular navigation of the cerebral vasculature can be rapid and dramatic and may require interdisciplinary collaboration. Often lesions that can be best treated by endovascular means are also treatable by neurosurgical techniques. In such situations, close interaction with the neurosurgeon is essential to ensure that the safest mode of therapy can be chosen for the patient. Similarly, after the occurrence of complications such as intracerebral hemorrhage, literature has shown that emergent evacuation of the hematoma goes a long way in preventing / minimizing the morbidity and mortality associated with the complication. As neurovascular pathological lesions are extremely susceptible to changes in cerebral hemodynamics, close interaction with the neuroanesthetist is essential to ensure the highest standards of safety during and after the procedure. Training of the technical staff and nursing staff in the intensive care unit about the special requirements of patients after neurovascular interventions may help in minimizing the post procedural complications.

Procedure related complications have widespread and important consequences for not only the patient, but also for the treating physician and for hospital resource utilization. Thus a review of complications has wide ranging implications as outlined in Table 7.1.

CRITICAL ANALYSIS OF COMPLICATIONS DURING NEUROINTERVENTIONAL PROCEDURES:

Rate of Occurrence and Severity of Complications:

Analysis of complications among the neurointerventional procedures performed at our institution during the study period (Table 5.1) revealed maximum occurrence of major complications (complication outcomes: 4, 5 and 6) with embolization of intracranial aneurysms (17.4 %), cerebral AVMs (6.6 %) and dural AV malformations (10%).

Minor complications (complication outcome: 1 and 2) occurred most frequently during percutaneous vertebroplasty, brain AVM embolization, aneurysm coiling and embolization of head and neck tumors. These typically did not result in deterioration of neurological status of the patients at discharge. The rate of complications with

Table 7.1: Implications of the present study

1. **Review of interventional practices and techniques**
    a. Preprocedural Considerations
        i. Procedural risk stratification to offer a safe mode of treatment for the patient
        ii. Explanation of risks of procedure to patient for informed consent.
    b. Procedural Considerations
        i. Ensuring availability of all hardware for rescue from complication
        ii. Modification of technique to prevent complications
        iii. Monitoring for early signs of complications
        iv. Prompt and correct management of the complication
    c. Post procedural Considerations
        i. Modification of post procedural monitoring protocols – eg; longer ICU stay
        ii. Modification of post procedure therapeutic protocols
        iii. Avoidance of risky situations eg; Rigorous control of BP in AVMs

2. **Identification of areas deficiency in response to complications, to teach technicians and other paramedical staff to react in emergencies**

3. **Risk stratification**
    a. Identification of low-risk neurointerventions for performance and high-risk neurointerventions for avoidance during training and initial phase of practice for inexperienced operators
    b. Performance of high-risk interventions for performance by more experienced operators

4. **Optimizing Resource Utilization**

embolization of intracranial AV fistulae (including Galenic and Non Galenic) (25 %) and miscellaneous neurointerventions (10 %) is likely to be falsely elevated due to the small number of such procedures performed during the study period of the present study.

Analysis of the neurological impact of occurrence of complications revealed maximal change of Rankin score for complications associated with embolization of intracranial arteriovenous fistulae, intracranial aneurysms and cerebral AVMs. Thus, these interventions can be identified as high-risk interventions and should be performed by experienced operators only.

Timing of Occurrence of Complications:

In our experience, except embolization of brain AVMs in which 16.7% of complications occurred in the post procedural period, complications associated with all other neurointerventional procedures have occurred during the interventional procedure. This does not undermine the importance of post procedure monitoring and care in other procedures, but only underlines the extra care that is necessary in post procedural management of cerebral AVMs.

Risk Factors for Complications:

Patient Factors

The role of preexisting systemic diseases such as hypertension, diabetes and coronary artery in the occurrence of complications is limited in our study. Two patients, who died in the post procedural period due to acute myocardial infarction, were excluded from the study, as they had preexisting severe triple vessel disease and were deemed to be poor risk candidates for any sort of treatment. Though, in general, older patients are thought to be predisposed to occurrence of periprocedural complications due to atherosclerosis[(6)], in our study, we did not find any correlation between age and complication rate. Similarly, the rate of occurrence of complications was not related to the sex of the patient.

Clinical presentation and Angioarchitecture

Clinical presentation and lesional angioarchitecture had a variable role in producing complications.

Among Cerebral AVMs, a slightly higher proportion of embolization procedures that had complications, involved AVMs that primarily presented with hemorrhage. Also, patients who had complications had poorer Rankin scores at presentation. Analysis of angioarchitecture revealed that small AVMs, AVMs associated with plexiform nidus, and AVMs in which a higher percentage of nidal reduction was achieved were associated with complications.

Hartmann and coworkers[12], found no relation between Spetzler-Martin grade of the AVM and occurrence or outcome of complications. In our study, however, the percentage of AVM nidus that was embolized during the intervention had a weak correlation with outcome. This relation was stronger with large AVMs as compared to small AVMs. Medium sized AVMs had no correlation between percentage of nidal reduction and outcome (Figure 7.1). A similar strong correlation was found between percentage of AVM embolization in grade 5 and grade 2 Spetzler-Martin lesions, while grade 3 and grade 4 lesions showed no such correlation (Figure 7.2). Analysis of frequency of complications in relation to the number of feeders embolized, revealed an increase in poorer outcomes with increase in number of feeders that were embolized in small AVMs. No such relation was found in medium sized and large AVMs (Figure 7.3).

Prior to embolization, large AVMs have low pressure flow within. Embolization of a considerable portion of the lesion elevates the pressure within the AVM and thus predisposes to rupture. A similar phenomenon of elevation of intranidal pressure may account for the higher number of complications with subtotal embolization of small AVMs. Since size of the AVM is a major factor in the Spetzler-Martin scoring system, the same trends may be reflected in the analysis using Spetzler-Martin grades.

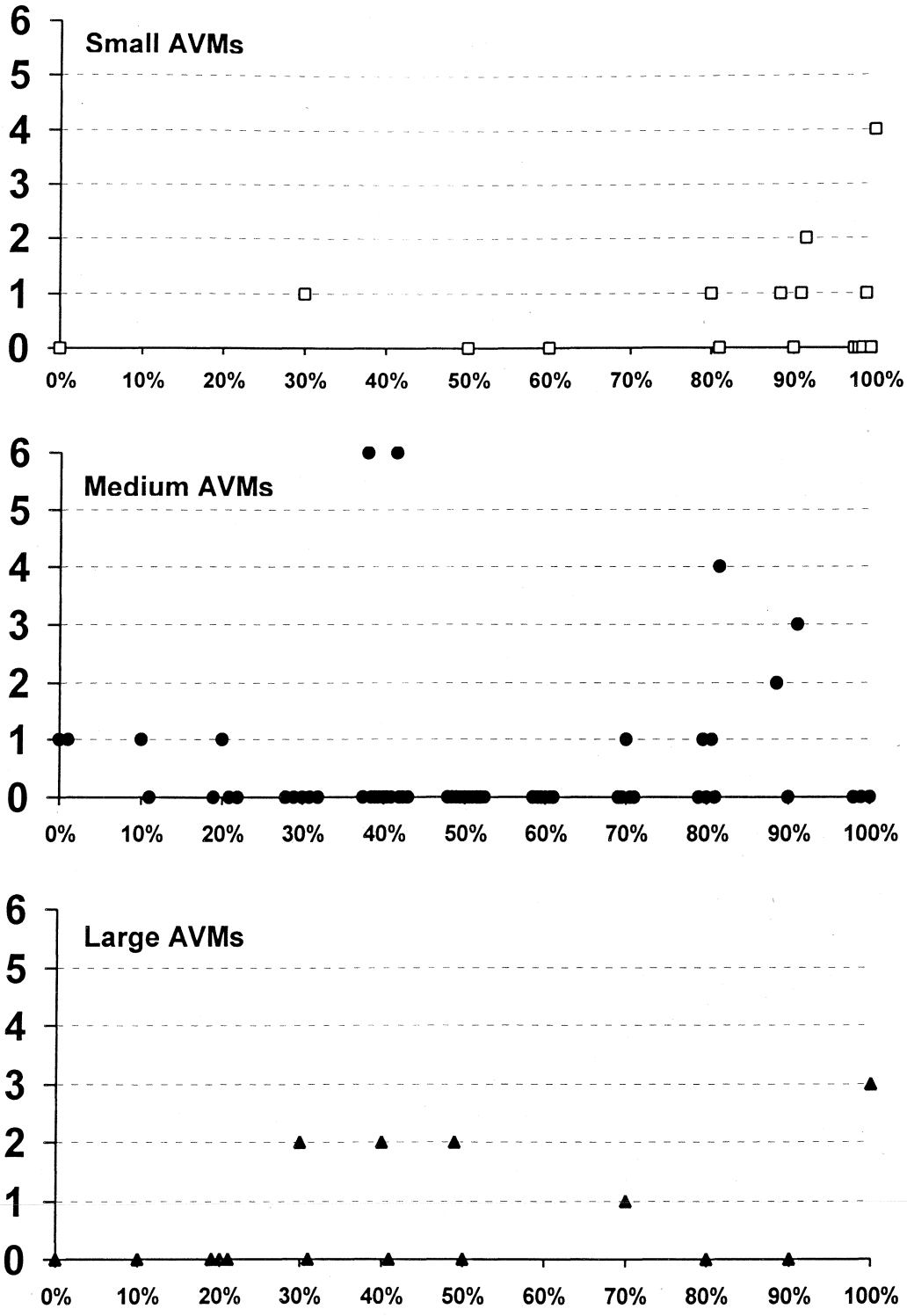


Figure 7.1: Complications of Brain AVM Embolization.

Relationship of percentage of nidus embolized per procedure, with procedural outcome, in small (top), medium (middle), and large (bottom) AVMs

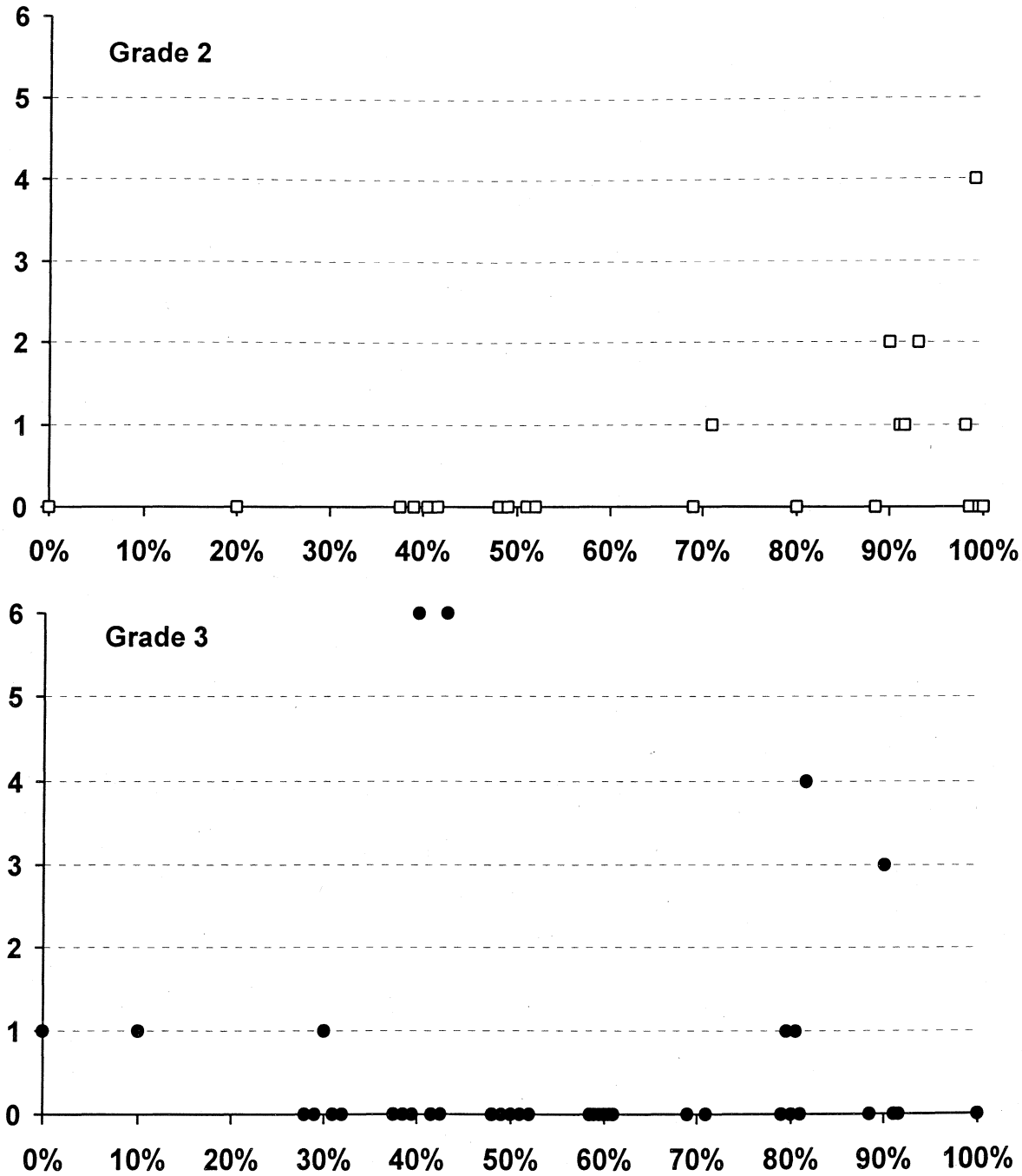


Figure 7.2 : Complications of Brain AVM Embolization

Relationship between percentage of embolization and complication outcome of the procedure in Spetzler-Martin Grade 2 (top) and 3 (bottom) lesions

(Contd ...)

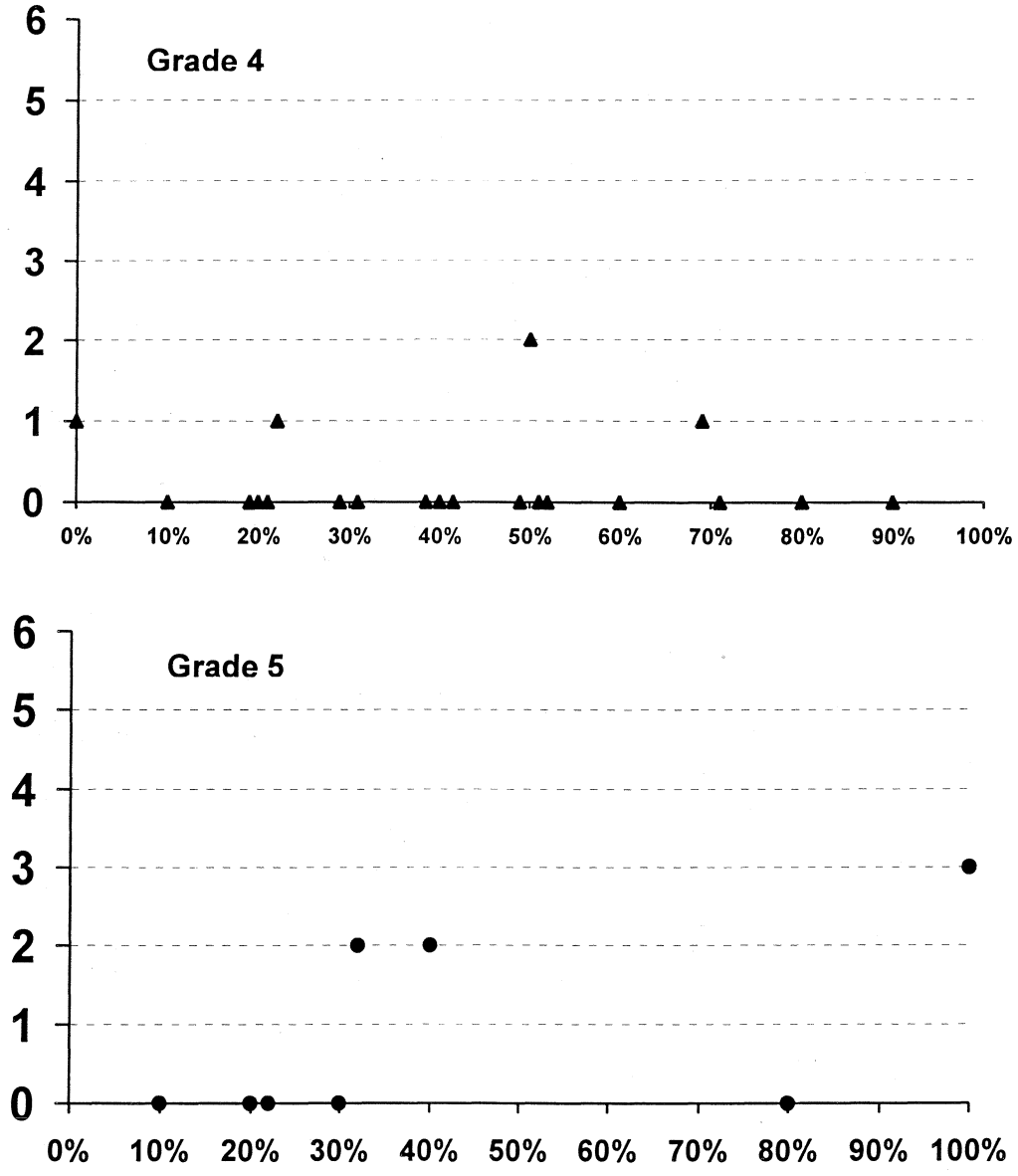


Figure 7.2 (Contd) : Complications of Brain AVM Embolization

Relationship between percentage of embolization and complication outcome of the procedure in Spetzler-Martin Grade 4 (top) and 5 (bottom) lesions

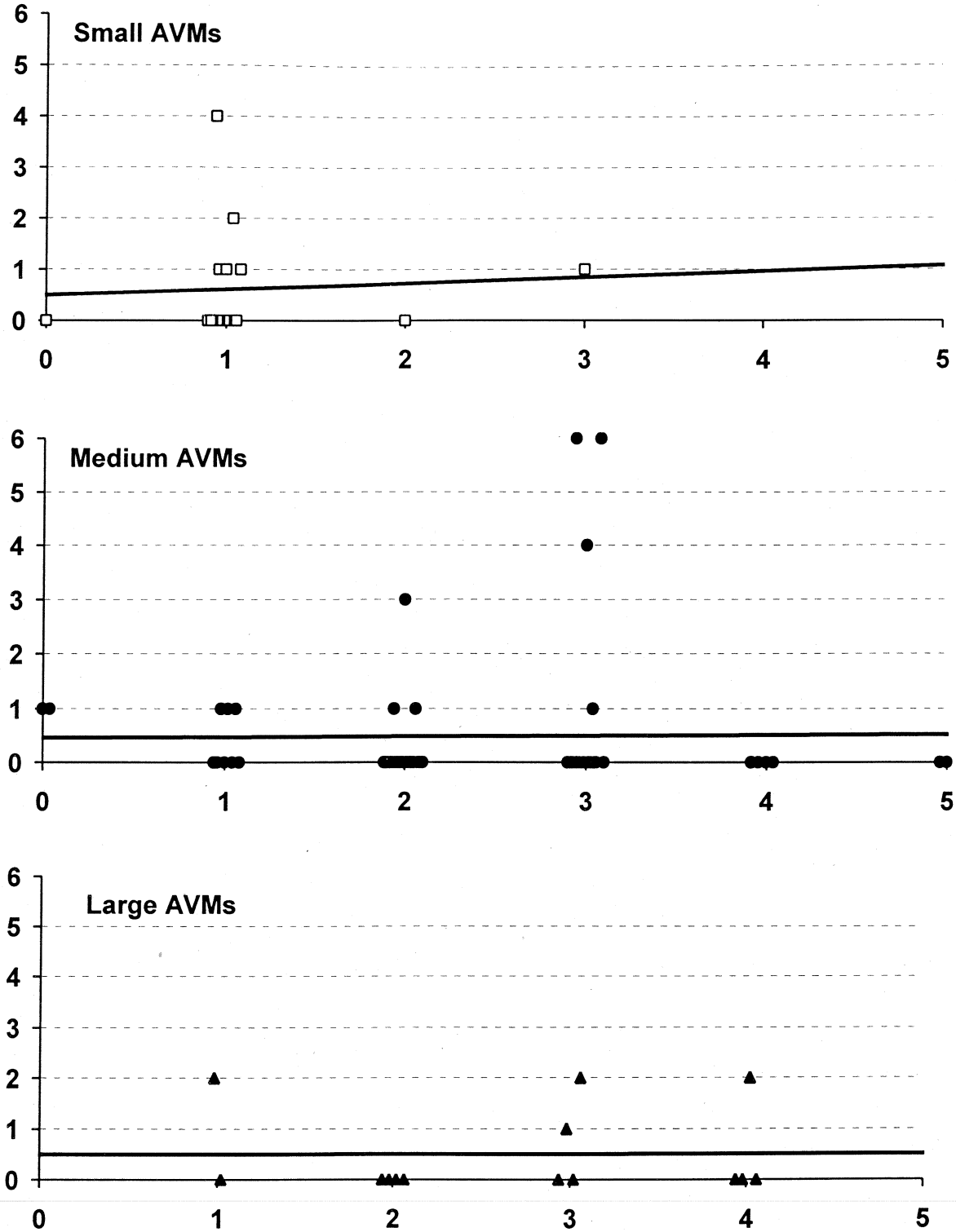


Figure 7.3 : Complications of Brain AVM Embolization

Relationship between number of feeders embolized per sitting and complication outcome of the procedure in small (top), medium (middle) and large (bottom) lesions

Both complications, which occurred during intracranial AV Fistula embolizations in our study, were directly related to the clinical presentation and adverse angioarchitecture. One patient with a vein of Galen malformation had presented with intraventricular hemorrhage and had dilated subependymal veins on imaging. The second patient with a vein of Galen malformation had a bulbous projection from the anterior end of the venous sac, at the confluence of the feeder arteries. Embolization of the malformations through combined transvenous and transarterial routes resulted in intraventricular and subarachnoid hemorrhage respectively.

On the other hand, no relation was found between occurrence of complications and the angioarchitecture in intracranial aneurysms, caroticocavernous fistulae, carotid and subclavian atherosclerotic stenoses, and craniofacial lesions.

Procedural Factors

Procedure related factors range in severity from potentially fatal incidents like rupture of AVM nidus during cannulation or rupture of aneurysm during coiling, to minor technical complications such as spasm of parent artery or perivertebral leaks in percutaneous vertebroplasty. In certain aspects, these complications are easier to manage than post-procedural complications, as the vascular access is available and most material needed for salvage is available in the cath lab, resulting in faster institution of corrective measures. In general, events that occur during the later part of the procedure have better prognosis than events occurring in the early part. Bleeding from aneurysms that have been partially coiled is not as massive as with uncoiled aneurysms. Similarly, availability of premixed cyanoacrylate glue has played a large role in successful salvage of a ruptured AVM nidus in one of our patients, by enabling immediate embolization.

Key features to the successful management of intraprocedural complications are active monitoring for signs of complication and immediate corrective measures. Periodic check angiograms to confirm the location and path of the microcatheter may go a long way in prevention of vascular perforations associated with microcatheterization. Similarly, periodic check angiogram play a vital role in preventing reflux of embolic materials in procedures involving embolization of the external carotid branches. As most

neurointerventions are performed under general anesthesia, signs of occurrence of an adverse event are limited. Sudden elevation and unresponsiveness of blood pressure are definitive signs of intracerebral hemorrhage and must be verified by immediate check angiogram. In the event of intracerebral hemorrhage, immediate reversal of anticoagulation and lowering of blood pressure have gone a long way in the management of three patients with intraprocedural nidal / feeder rupture.

In institutions where reuse of catheters, guidewires, and other such interventional hardware occurs, there is a high risk of intraprocedural thromboembolic events. Stripping-off of the coating on the catheters and guidewires as well as the thrombogenic nature of residual sterilizing agents on these materials are chiefly responsible for these events. This problem is compounded by the empirical anticoagulation utilized in most neurointerventional procedures. Absence of equipment to monitor the activated clotting time is responsible for fixed dose anticoagulation, which may occasionally be insufficient. Probably, in settings where reuse of hardware is rampant, meticulous care to heparinization and over-heparinization may be necessary to prevent thromboembolic complications.

Central to the management of the complications is a thorough understanding of hemodynamics of the cerebral vasculature and techniques of management of various cerebrovascular pathologies. For example, a thromboembolic complication during coil embolization of a recently ruptured aneurysm is best managed by continuation of coiling rather than the institution of thrombolysis. A neurological deficit produced by the thrombus may be more acceptable than the risk of rebleeding during thrombolysis.

Technical complications due to device malfunctions may occur in spite of best of the preparation and care. The key to successful management of such complications is to ensure the availability of all hardware that is needed in salvage techniques. Thus hardware materials such as vascular snares and loops, a wide range of microcatheters and microwires, stents, detachable and nondetachable balloons, as well as drugs such as papaverine, nitroglycerin, urokinase, antihypertensives and sedatives are an essential part of every cath lab where neurointerventional procedures are performed.

Post Procedural Factors

Four of the 16 complications (16.7 %) associated with brain AVM embolization which occurred in the post procedure period, could be directly attributed to sudden rise in the patient's blood pressure. All these complications had a poor outcome, with death in 1 patient, hemiplegia in 1 patient and minor neurological deficits in 2 patients. This points to the need of better post procedural care in patients who have undergone embolization of cerebral AVMs. Significantly, the sudden rise in BP in two of these patients was secondary to abrupt cessation of vasodilator drugs (n=1) or administration of atropine injection for transient bradycardia (n=1). Care to prevent such sudden fluctuations of blood pressure could go a long way in preventing such complications – not only in brain AVM embolization, but also in procedures such are embolization of intracranial arteriovenous fistulae, intracranial aneurysms and in carotid stenting.

ANALYSIS OF FUNCTIONAL OUTCOME AND RESCUE STRATEGIES

Analysis of hemorrhagic complications during embolization reveals interesting facts. Among 7 procedures that were associated with vascular disruption during neurointerventional procedures, 4 occurred during AVM embolization, 2 during embolization of vein of Galen malformations and 1 during coil embolization of an intracranial aneurysm. The details of these complications and outcome are outlined in Table 7.2

In the two patients with vein of Galen malformations who had fatal hemorrhages, the events occurred after transvenous coil embolization, which presumably caused a rise in venous pressure within the malformation. Thus in these two patients and in the lone patient with intraprocedural rupture of an aneurysm, the bleed occurred in a high-pressure state. This could have played a large role in the poor outcome that was seen in these patients. In contrast, bleed from a disrupted AVM is usually at a low pressure and usually has a good prognosis, as seen in 3 of the 4 patients in our series

In these 3 patients with brain AVMs, prompt intervention prevented potential neurological disasters. The mean delay between recognition of the vascular disruption

Table 7.2 : Vascular Disruption during neurointerventional procedures

	Patient Details	Underlying Vascular Pathology	Mechanism of vascular disruption	Outcome	Neurological Deficits
1	38 M	Lt Medial Parietal AVM	Feeder perforation during microcatheterization	Parietal intracerebral hemorrhage.	Death
2	23 M	Lt Post. Temporal AVM	Perforation of feeder artery during cannulation	Contained perforation	Nil
3	16 F	Rt Parietal AVM	Nidus rupture during microcatheterization	Contained rupture	Nil
4	23 M	Rt Motor Strip AVM	Feeder rupture during glue embolization	Contained rupture	Nil
5	28 M	Vein of Galen malformatn	Feeder rupture during cannulation after venous embolization	Intraventricular hemorrhage	Death
6	3 M	Vein of Galen malformatn	Feeder rupture during cannulation after venous embolization	Subarachnoid hemorrhage.	Death
7	55 M	Basilar top aneurysm	? due to balloon used in remodelling of neck of aneurysm	Subarachnoid hemorrhage	Death

and glue embolization of the vessel was 3.8 minutes in these patients. In sharp contrast, in the lone patient with brain AVM who had a bad outcome, despite immediate reversal of anticoagulation and lowering of blood pressure, the delay for glue embolization was 6.5 minutes. Thus the time taken for sealing off the disrupted vessel is a major determinant in outcome of vascular disruptions.

In these patients, prompt neurosurgical intervention and evacuation of hematoma could have played a major role in improving the neurological status. In the study of Keller and coworkers [16], patients with intervention related intracranial bleeds were managed using a special protocol that aimed to minimize the time between onset of symptoms and surgical evacuation, by avoiding the time consuming procedures that are not essential for management. They reported 100% successful recovery using their protocol, compared to 61.5% recovery in patients before use of the protocol. In our study, considerable delays occurred in shifting these patients to the CT scan suite and subsequently to the ICU and for neurosurgical consultation. By then signs of brainstem failure had set-in in most of our patients. The uncertain neurological status, presence of residual vascular malformation and anticoagulation are chief reasons that neurosurgeons cited for conservative management in our patients. Prompt decision-making and emergent surgical intervention may help salvage these patients.

The general measures that may prevent and help in management of complications during neurointerventional procedures, as well as specific measures as applicable in individual procedures, are outlined in Tables 7.3 – 7.8.

Table 7.3: General Measures for Prevention and Management of Complications of Neurointerventions

Patient factors

- Tailoring the type and extent of procedure to suit the clinical need of patient
- Correction of risk factors if any

Equipment factors

- Biplane DSA system is ideal (No time loss in critical situations)
- High resolution for high quality information that aids in decision-making
- Choice of hardware
- Optimal use of materials
- Avoiding reuse of materials

Operator factors

- Adequate knowledge of pathophysiology of disease, technique, alternative therapy, and experience in managing the procedure related complications
- Adequate practical training regarding materials and operative techniques
- Adequate knowledge of hardware

General Procedural factors

- Informed consent
- Neurosurgical standby for rescue
- Neurointerventions in general anesthesia when possible to minimize patient movement and make maximum use of roadmap and landmark facilities. However, the risk of patient movement during the procedure must be balanced with the need for physiological monitoring during the procedure.
- Evolution of protocols for performance of a procedure
    - Standardizes the procedure with reduced chance of missing vital steps
    - Ensures uniformity of technique so that results can be compared easily
- Keeping the hardware and medications for rescue at hand
- Avoiding vascular perforations during neuronavigation by keeping the microguidewire within the confines of microcatheter
- Regular check angiogram, fluoroscopy to check position of tip of microcather and wire during manipulation
- Avoiding rapid injection of fluids with 1cc syringe
- Use of ideal projection that allows unimpeded view of the anatomy
- Use of landmark and roadmap facilities.

Table 7.4: Prevention and Management of Complications of Embolization of Brain AVMs and other Arteriovenous shunt pathologies

Preprocedural Measures:

- Careful analysis of angioarchitecture to identify feeder / intranidal aneurysms for tackling before the AVM

Intraprocedural Measures:

- Meticulous anticoagulation to prevent thromboembolic events.
- Careful analysis of nidal angioarchitecture in orthogonal planes using microcatheter injections
- Use of microcatheters such as Magic (Balt Extrusion, Paris) that permit detachment of the terminal soft segment in event of microcatheter adhesion
- Avoiding rapid injection of saline / contrast into the feeder vessels
- Avoiding use of microguidewire beyond the tip of microcatheter for navigation
- Optimal positioning of the microcatheter tip for embolization
- Selection of optimal plane for visualization during embolization
- Measurement of feeder pressures and analysis of waveforms may help to identify microcatheter wedging
- Removal of any redundant loops in the microcatheter before embolization
- Use of optimal concentration of glue– based on intranidal flow patterns
- Meticulous flushing of microcatheter with dextrose
- Use of systemic hypotension to retard flow within the nidus
- Use of Berenstein Liquid coils (Boston Scientific, USA) for flow reduction
- Optimal rate of glue injection
- Pausing injection of glue if glue entry into venous components begins
- Prevention of glue reflux proximally along the catheter
- Prevention of escape of glue on to venous side
- Aspiration of the injection syringe in conjunction with abrupt guide catheter and microcatheter withdrawal at the end of the embolization.
- Avoiding embolization of a large percentage of nidus during a single sitting
- Utmost care to prevent reflux during embolization using absolute alcohol
- Reversal of additional heparin at the end of the procedure

Table 7.4: Prevention and Management of Complications of Embolization of Brain AVMs and other Arteriovenous shunt pathologies (Contd..)

Post Procedural Measures

- Avoidance of autonomic responses during recovery from anesthesia
- Maintenance of systemic BP at approximately 10 to 15% below baseline for 48 hours after embolization, using infusion of Sodium nitroprusside to minimize the phenomenon of normal pressure breakthrough and to reduce the risk of delayed hemorrhage. If needed, additional antihypertensive agents such as infusion Nitroglycerine, Inj.Labetalol or oral β adrenergic blockers may be added.

- Periodic neurological monitoring in the ICU, with charting of blood pressure, pulse rate, flow of vasodilator agents and input-output charting.

Management of Common Complications

- In the event of hemorrhage, immediate reversal of heparin with protamine without undue regard for systemic blood pressure. This is followed by embolization of the feeder using glue. Immediate post procedural CT scan and emergent neurosurgical evacuation of hematoma / drainage of ventricular system if required.

- In the event of microcatheter retention, antiplatelet agents / anticoagulation for 3-6 months

- In the setting of inadvertent vascular occlusion, increase of distal perfusion by blood pressure augmentation to drive adequate flow via collaterals to the area of ischemia as a temporizing measure. Adequate care should be taken to prevent rapid or high-rise of blood pressures.

Table 7.5: Prevention and Management of Complications of Aneurysm Coiling

Preprocedural Measures:

- Careful analysis of angioarchitecture with special emphasis on width of neck, sac-neck ratio, angle of projection in relation to parent artery, intraluminal thrombus and branches arising from the aneurysm sac
- Confirming the availability of additional material to deal with complications such as materials for contralateral puncture, guidecatheter, 2nd microcatheter, stents, retrieval devices, flow guided Non detachable balloons, detachable balloons and drugs: urokinase, protamine, antihypertensive agents

Intraprocedural Measures:

- Avoiding reuse of hardware
- Avoidance of wide bore guiding catheters
- Use of soft tipped guidecatheters to prevent spasm
- Optimal anticoagulation with periodic monitoring of activated clotting time
- Use of good flush line to flush the guiding catheter
- Use of softer guidewires
- Extra care during embolization of high risk aneurysms such as recently ruptured aneurysms, small aneurysms and posterior fossa aneurysms
- Extra care during use of microcatheters with hydrophilic coating
- Extra care during use of techniques such as remodeling technique or stent assisted coiling
- Removal of redundant loops in the microcatheter to prevent sudden forward movement during withdrawal of guidewire.
- Careful attention to compatibility of coil and microcatheter to prevent coil stretching and fracture
- Careful monitoring for any sudden BP changes during the procedure
- Careful attention to sizing of the coil
- Careful attention to detect any prolapse of coils
- Use of stents / remodeling technique in wide necked lesions

Table 7.5: Prevention & Management of Complications of Aneurysm Coiling (Contd ..)

Post Procedural Measures:

- Maintenance of anticoagulation and augmentation of blood pressure during the post procedural period

Management of Complications:

- If thrombus formation occurs in a proximal vessel such as a non-dominant vertebral artery, proximal occlusion of the artery can be performed.
- If thrombus formation occurs in the carotid artery, full heparinization or thrombolysis / aspiration thrombectomy with proximal balloon occlusion may be performed
- In the event of embolization of the thrombus into intracranial circulation, if the aneurysm has been occluded, full heparinization, augmentation of blood pressure and intraarterial thrombolysis can be performed. In the presence of a partly coiled aneurysm, importance must be given to securing the aneurysm.
- In the event of perforation, immediate reversal of heparinization and lowering of blood pressure are required. If the procedure is being done with the help of a balloon, inflation of the balloon may help reduce the bleed
- If the perforation is due to a microguidewire, it is left in place. The aneurysm is accessed using a second microcatheter using a contralateral arterial access and coiled completely, before slowly withdrawing the microguidewire
- If the perforation is due to a microcatheter, it is left in place. The aneurysm is accessed using a second microcatheter using a contralateral arterial access and coiled completely. A coil is passed through the microcatheter that has perforated the aneurysm, and deposited so as to lie partly outside and partly within the aneurysm sac. Then the microcatheter is removed.
- If the perforation is due to a coil, it is deployed is the same location. More coils are rapidly placed to secure the aneurysm
- If coil prolapse into main artery occurs, antiplatelet agents and anticoagulation may be required. In cases of coil migration, additionally retrieval of the coil with a snare or compressing the coil to the wall of the vessel with a stent may be attempted

Table 7.6: Prevention & Management of Complications of Embolization of Head and Neck lesions and Extracerebral Arteriovenous Fistulae

Preprocedural Measures

- Setting well defined goals of embolization depending on the indication
- Identifying best route of embolization – vascular / direct puncture / venous

Intraprocedural Measures

- Careful analysis of angioarchitecture of lesion with attention to supply to vital structures and presence of any dangerous anastomoses
- Prevention of hypoxia and hypercarbia to prevent external carotid – internal carotid anastomoses.
- Use of microcatheters for superselective cannulation for embolization
- Optimal use of guidewires and guidecatheters to prevent arterial dissection
- Use of balloons for flow reduction / protection of vital territories
- Proximal embolization of nonfeeding arteries with coils to protect distal bed
- Provocative testing of the territory using Lignocaine
- Isolation of syringes used for particulate embolization from those that are used for injecting contrast / flush
- Periodic check injections to look for reflux / opening up of new collaterals
- Regular flushing of catheter system to carry the particles into a distal bed and to prevent occlusion of the catheter.
- Use of particles opacified with contrast medium for fluoroscopic visualization during embolization
- Avoidance of use of small particles (<150 μ)
- Use of progressively larger particles to occlude the bed first, followed by occlusion of the arteries supplying the lesion
- Use of small quantity of particles per syringe
- Thorough flushing of catheter system before withdrawl from the feeder

Post Procedural Measures

- Avoidance of palpation / pressure on the lesion
- Post procedural steroid medication to reduce mass effect

Table 7.7: Prevention & Management of Complications of Carotid Stenting

Preprocedural Measures:

- Careful analysis of angiographic anatomy for location, degree and extent of stenosis, presence of any plaque ulceration / soft thrombus and for presence of any loops / kinks in the native vessel
- Starting antiplatelets agents 3 days prior to the procedure.
- Temporary cardiac pacing may be required for the duration of the procedure

Intraprocedural Measures :

- Use of guidecatheters for easy access to the lesion and navigation of the stent
- Optimal anticoagulation to prevent thromboembolic events
- Use of protection devices
- Use of predilation for tight lesions
- Careful positioning of the stent device to completely cover the stenotic segment
- Second stent may be used to bail out in cases of dissection complicating the procedure / deployment of the end of the stent in an arterial loop
- In the event of thromboembolic pathology, immediate thrombolysis may be required

Post Procedural Measures:

- Careful ICU monitoring of neurological status, blood pressure, pulse rate and input – output charting after the procedure
- Prevention of hypertension that may precipitate hyperperfusion syndrome / intracerebral hemorrhage

Table 7.8: Prevention & Management of Complications associated with Percutaneous Vertebroplasty

Preprocedural Measures:

- Careful analysis of architecture of the affected vertebra, integrity of the posterior cortex and presence of any epidural component.

Intraprocedural Measures :

- Use of abundant local anesthetic agent, down to the level of periosteum
- Use of the largest caliber of needle that can be safely used
- Careful visualization in all planes to confirm the safety of the needle path
- Periodic evaluation of sensory/motor signs & symptoms during the procedure
- Optimal location of needle
- Contrast injection may help in identifying prompt venous drainage
- Rapid reconstitution of bone cement and injection
- Optimal consistency of bone cement to balance ease of injection with risk of leakage into perivertebral / epidural spaces.
- Careful fluoroscopic control during injection of bone cement to prevent retrovertebral / perivertebral / disc space leakage
- Use of a contralateral pedicular access may be safer than trying to completely fill the vertebra from with a single access, in certain cases.

8. CONCLUSIONS

CONCLUSIONS

Complications of neurointerventional procedures occurring in the periprocedural period and post procedural period are fortunately rare. However, in view of the extreme sensitivity of the neural tissues to ischemic and other insults, these may lead to irreparable damage. These events have far reaching implications for both the patient and the treating neurointerventional radiologist.

This retrospective review of neurointerventional procedures performed during the study period revealed that most complications of neurointerventional procedures could not have been predicted. They did not occur in specific cohorts of patient populations that could be identified as high risk by analysis of clinical or angioarchitectural details. However, attempts were made to identify the procedures in which occurrence of complications was associated with major deterioration of neurological status of the patient. Rates of occurrence of complications were compared with international standards and thresholds. Remedial measures instituted after the occurrence of complications were analyzed for effectiveness and changes to the protocols were suggested.

Similar periodical analyses and constant refinement of techniques and protocols, in tune with latest developments as reported in literature can go a long way in preventing periprocedural complications and in effective salvage from potentially dangerous situations.

9. REFERENCES

REFERENCES

1. CW Kerber, WO Bank, and C Manelfe
Control and placement of intracranial microcatheters
AJNR Am. J. Neuroradiol., 1980; 1: 157 – 159

2. Berenstein A.
Flow-controlled silicone fluid embolization
AJNR Am J Neuroradiol 1980; 1: 161-166

3. CM Strother
Interventional Neuroradiology
AJNR Am. J. Neuroradiol., Jan 2000; 21: 19 - 24.

4. VV Halbach, RT Higashida, CF Dowd, SL Barnwell, and GB Hieshima
Management of vascular perforations that occur during neurointerventional procedures
AJNR Am. J. Neuroradiol. 1991; 12: 319 - 327

5 Cloft HJ, Jensen ME, Kallmes DF, Dion JE
Arterial Dissections Complicating Cerebral Angiography and Cerebrovascular Interventions
AJNR Am. J. Neuroradiol. 2000; 21: 541 - 545

6 Ernest F, Forbes G, Sandok BA, et al.
Complications of cerebral angiography: prospective assessment of risk.
AJR Am J Roentgenol 1984; 142: 247 - 253

7 Dion JE, Gates PC, Fox AJ, Barnett HJM, Blorn RJ.
Clinical events following neuroangiography: a prospective study.
Stroke 1987; 18: 997 – 1004

8 Grzyska U, Freitag J, Zeumer H.
Selective cerebral intraarterial DSA: complication rate and control of risk factors. Neuroradiology 1990; 32: 296 – 299

9 Waugh JR, Sacharias N. **Arteriographic complications in the DSA era.** Radiology 1992; 182: 243 – 246

10 Heiserman JE, Dean BL, Hodak JA.
Neurologic complications of cerebral angiography.
AJNR Am J Neuroradiol 1994; 15: 1401 - 1407

11 **Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology**
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s1 – s33

12 A. Hartmann, J. Pile-Spellman, C. Stapf, R.R. Sciacca, A. Faulstich, J.P. Mohr, H.C. Schumacher, and H. Mast
Risk of Endovascular Treatment of Brain Arteriovenous Malformations
Stroke, 2002; 33: 1816 - 1820

13 The n-BCA Trial Investigators
n-Butyl Cyanoacrylate Embolization of Cerebral Arteriovenous Malformations: Results of a Prospective, Randomized, Multi-center Trial
AJNR Am. J. Neuroradiol., 2002; 23: 748 - 755

14 Debrun GM
Embolization of the nidus of brain arteriovenous malformations with n-butyl cyanoacrylate. Int Neuroradiol 1997; 3: 13-19

15 Whittle IR, Johnston IH, Besser M, Lamond TS, de Silva M.
Experience with bucrylate (isobutyl-2-cyanoacrylate) embolization of cerebral arteriovenous malformations during surgery.
Surg Neurol. 1983; 19: 442-9.

16 E. Keller, Y. Yonekawa, H. Imhof, M. Tanaka, Anton Valavanis
Intensive care management of patients with severe intracerebral haemorrhage after endovascular treatment of brain arteriovenous malformations
Neuroradiology 2002; 44: 513-521

17 Pevsner PH, Klara P, Doppman J, George E, Girton M
Ethyl alcohol: experimental agent for interventional therapy of neurovascular lesions
AJNR Am. J. Neuroradiol. 1983; 4: 388 – 390.

18 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
Cerebral Arteriovenous Malformations
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s10 – s11

19 Murayama Y, Nien YL, Duckwiler G, Gobin YP, Jahan R, Frazee J, Martin N, Vinuela F.
Guglielmi detachable coil embolization of cerebral aneurysms: 11 years' experience.
J Neurosurg. 2003; 98: 959-66

20 V Singh, DR Gress, RT Higashida, CF Dowd, VV Halbach, SC Johnston
The Learning Curve for Coil Embolization of Unruptured Intracranial Aneurysms
AJNR Am J Neuroradiol 2002; 23: 768-771

21 H Saitoh, K Hayakawa, K Nishimura, Y Okuno, T Teraura, K Yumitori, A Okumura
Rerupture of cerebral aneurysms during angiography
AJNR Am J Neuroradiol 1995 16: 539-542

22 CG McDougall, VV Halbach, CF Dowd, RT Higashida, Larsen DW, Hieshima G.
Causes and management of aneurysmal hemorrhage occurring during embolization with Guglielmi detachable coils.
J Neurosurg. 1998; 89: 87-92

23 Ricolfi F, Le Guerinel C, Blustajn J, Combes C, Brugieres P, Melon E, Gaston A.
Rupture during treatment of recently ruptured aneurysms with Guglielmi electrodetachable coils.
AJNR Am J Neuroradiol. 1998; 19: 1653-1658

24 Vinuela F, Duckwiler G, Mawad M.
Guglielmi detachable coil embolization of acute intracranial aneurysm: perioperative anatomical and clinical outcome in 403 patients.
J Neurosurg. 1997; 86: 475-482

25 Arnd Doerfler, Isabel Wanke, Thomas Egelhof, Uwe Dietrich, Siamak Asgari, Dietmar Stolke, and Michael Forsting
Aneurysmal Rupture during Embolization with Guglielmi Detachable Coils: Causes, Management, and Outcome
AJNR Am. J. Neuroradiol. 2001; 22: 1825 - 1832

26 Sluzewski M, Bosch JA, van Rooij WJ, Nijssen PC, Wijnalda D.
Rupture of intracranial aneurysms during treatment with Guglielmi detachable coils: incidence, outcome, and risk factors.
J Neurosurg. 2001; 94: 238-240.

27 HJ Cloft, DF Kallmes
Cerebral Aneurysm Perforations Complicating Therapy with Guglielmi Detachable Coils: A Meta-Analysis
AJNR Am. J. Neuroradiol. 2002; 23: 1706 - 1709

28 Pelz DM, Lownie SP, Fox AJ.
Thromboembolic events associated with the treatment of cerebral aneurysms with Guglielmi detachable coils.
AJNR Am J Neuroradiol. 1998; 19: 1541-7

29 Soeda A, Sakai N, Sakai H, Iihara K, Yamada N, Imakita S, Nagata I
Thromboembolic events associated with Guglielmi detachable coil embolization of asymptomatic cerebral aneurysms: evaluation of 66 consecutive cases with use of diffusion-weighted MR imaging.
AJNR Am J Neuroradiol. 2003; 24: 127-132

30 Derdeyn CP, Cross DT 3rd, Moran CJ, Brown GW, Pilgram TK, Diringer MN, Grubb RL Jr, Rich KM, Chicoine MR, Dacey RG Jr.
Postprocedure ischemic events after treatment of intracranial aneurysms with Guglielmi detachable coils.
J Neurosurg. 2002; 96: 837-843

31 Klotzsch C, Nahser HC, Henkes H, Kuhne D, Berlit P.
Detection of microemboli distal to cerebral aneurysms before and after therapeutic embolization.
AJNR Am J Neuroradiol. 1998; 19: 1315-1318

32 Standard SC, Chavis TD, Wakhloo AK, Ahuja A, Guterman LR, Hopkins LN.
Retrieval of a Guglielmi detachable coil after unraveling and fracture: case report and experimental results.
Neurosurgery 1994; 35: 994-998

33 VV Halbach
Comment: Retrieval of a Guglielmi detachable coil after unraveling and fracture: case report and experimental results.
Neurosurgery 1994; 35: 999

34 Pride GL Jr, Horowitz MB, Purdy PD.
Endovascular problem solving with intravascular stents.
AJNR Am J Neuroradiol. 2000; 21: 532-540

35 Phatouros CC, McConachie NS, Jaspan T.
Post-procedure migration of Guglielmi detachable coils and Mechanical detachable spirals.
Neuroradiology. 1999; 41: 324-327

36 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
Aneurysm Endovascular Therapy
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s4 – s7

37 Berenstein A, Lasjaunias P.
Embolization of Craniofacial Lesions
In: Berenstein A, Lasjaunias P, eds. Surgical Neuroangiography.
New York, NY: Springer Verlag; 1992

38 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
External Carotid Artery Embolization
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s12 – s13

39 Halbach VV, Higashida RT, Hieshima GB, Norman D
Normal perfusion pressure breakthrough occurring during treatment of carotid and vertebral fistulas
AJNR Am. J. Neuroradiol. 1987; 8: 751 - 756

40 Wholey MH, Wholey MH, Tan WA, Toursarkissian B, Bailey S, Eles G, Jarmolowski C.
Management of neurological complications of carotid artery stenting.
J Endovasc Ther. 2001; 8: 341-353

41 Henderson RD, Phan TG, Piepgras DG, Wijdicks EF.
Mechanisms of intracerebral hemorrhage after carotid endarterectomy.
J Neurosurg. 2001; 95: 964-969

42 Vitek JJ, Roubin GS, Al-Mubarek N, New G, Iyer SS.
Carotid artery stenting: technical considerations.
AJNR Am J Neuroradiol. 2000; 21: 1736-1743.

43 Deramond H, Depriester C, Galibert P, Le Gars D.
Percutaneous vertebroplasty with polymethylmethacrylate. Technique, indications, and results.
Radiol Clin North Am. 1998; 36: 533-546

44 Padovani B, Kasriel O, Brunner P, Peretti-Viton P.
Pulmonary embolism caused by acrylic cement: a rare complication of percutaneous vertebroplasty.
AJNR Am J Neuroradiol 1999; 20: 375-377

45 Phillips FM, Wetzel FT, Lieberman T, Campbell-Mupp M.
An in vivo comparison of the potential for extra vertebral cement leak after vertebroplasty and kyphoplasty Spine 2002; 19: 2173-2178

46 McGraw JK, Cardella J, Barr JD, Mathis JM, Sanchez O, Schwartzberg MS, Swan TL, Sacks D; SIR Standards of Practice Committee.
Society of Interventional Radiology quality improvement guidelines for percutaneous vertebroplasty. J Vasc Interv Radiol. 2003; 14: 827-831

47 Radanovic B, Simunic S, Stojanovic J
Therapeutic Embolization of Aneurysmal Bone Cyst
Cardiovasc Intervent Radiol 1990; 12: 313 - 316

48 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
Embolization of Spinal Arteriovenous Fistulae, Spinal Arteriovenous Malformations, and Tumors of the Spinal Axis
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s28 – s30

49 Forbes G, Nichols DA, Jack CR Jr, Ilstrup DM, Kispert DB, Piepgras DG, Wiebers DO, Earnest F 4th, Axley PL
Complications of spinal cord arteriography: prospective assessment of risk for diagnostic procedures.
Radiology 1988; 169: 479-484

50 Rodesch G, Hurth M, Alvarez H, David P, Tadie M, Lasjaunias P
Embolization of spinal cord arteriovenous shunts: morphological and clinical follow-up and results--review of 69 consecutive cases.
Neurosurgery 2003; 53: 40-49

51 Katzan I, Furlan A. Thrombolytic therapy. In: Fisher M, Bogousslavsky J, eds.
Current Review of Cerebrovascular Disease, 3rd ed.
Boston, Mass: Butterworth Heinemann; 1999: 185–193

52 Higashida RT, Furlan AJ
Trial Design and Reporting Standards for Intra-Arterial Cerebral Thrombolysis for Acute Ischemic Stroke
Stroke 2003; 34: e109

53 Kidwell CS, Saver JL, Carneado J, Sayre J, Starkman S, Duckwiler G et al
Predictors of Hemorrhagic Transformation in Patients Receiving Intra-Arterial Thrombolysis
Stroke 2002;33:717

54 von Kummer R, Hacke W.
Safety and efficacy of intravenous tissue plasminogen activator and heparin in acute middle cerebral artery stroke.
Stroke 1992; 23: 646–652

55 The NINDS t-PA Stroke Study Group.
Intracerebral hemorrhage after intravenous t-pa therapy for ischemic stroke Stroke 1997; 28: 2109–2118

56 Gore JM, Sloan M, Price TR, Randall AM, Bovill E, Collen D, et al.
Intracerebral hemorrhage, cerebral infarction, and subdural hematoma after acute myocardial infarction and thrombolytic therapy in the thrombolysis in myocardial infarction study: Thrombolysis in Myocardial Infarction, Phase II, pilot and clinical trial.
Circulation. 1991; 83: 448–459

57 Simoons ML, Maggioni AP, Knatterud G, Leimberger JD, de Jaegere P, van Domburg R, et al.
Individual risk assessment for intracranial haemorrhage during thrombolytic therapy.
Lancet. 1993; 342: 1523–1528

58 Anderson JL, Karagounis L, Allen A, Bradford MJ, Menlove RL, Pryor TA.
Older age and elevated blood pressure are risk factors for intracerebral hemorrhage after thrombolysis.
Am J Cardiol. 1991; 68: 166–170

59 Gebel JM, Sila CA, Sloan MA, Granger CB, Mahaffey, KW, Weisenberger J, et al.
Thrombolysis-related intracranial hemorrhage: a radiographic analysis of 244 cases from the GUSTO-1 Trial with clinical correlation: global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries.
Stroke. 1998; 29: 563–569

60 Kase C, Furlan A, Wechsler L, Higashida R, Rowley H, Hart R, et al.
Symptomatic intracerebral hemorrhage after intra-arterial thrombolysis with pro-urokinase in acute ischemic stroke: the PROACT II Trial.
Neurology. 2001; 57: 1603–1610

61 Mathis JM, Barr JD, Jungreis CA, Yonas H, Sekhar LN, Vincent D, Pentheny SL, Horton JA.
Temporary balloon test occlusion of the internal carotid artery: experience in 500 cases.
AJNR Am J Neuroradiol. 1995; 16: 749-754.

62 Simonson TM, Ryals TJ, Yuh WTC, Farrar GP, Rezai K, Hoffman HT.
MR imaging and HMPAO scintigraphy in conjunction with balloon test occlusion: value in predicting sequelae after permanent carotid occlusion.
AJR Am J Roentgenol 1992;159:1063-1068

63 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
Carotid Artery Balloon Test Occlusion
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s8 – s9

64 Wasay M, Bakshi R, Kojan S, Bobustuc G, Dubey N, Unwin DH
Nonrandomized Comparison of Local Urokinase Thrombolysis Versus Systemic Heparin Anticoagulation for Superior Sagittal Sinus Thrombosis
Stroke. 2001;32:2310

65 Horowitz M, Purdy P, Unwin H, Carstens G, Greenlee R, Hise J et al.
Treatment of dural sinus thrombosis using selective catheterization and urokinase.
Ann Neurol. 1995; 38: 58–67

66 Frey JL, Muro GJ, McDougall CG, Dean BL, Jahnke HK.
Cerebral venous thrombosis: combined intrathrombus rtPA and intravenous heparin.
Stroke. 1999; 30: 489–494

67 Lylyk P, José E. Cohen JE, Ceratto R, Ferrario A, Miranda C
Angioplasty and Stent Placement in Intracranial Atherosclerotic Stenoses and Dissections
AJNR Am. J. Neuroradiol. 2002; 23: 430 - 436

68 Nakatsuka H, Ueda T, Ohta S, Sakaki S
Successful percutaneous transluminal angioplasty for basilar artery stenosis: technical case report.
Neurosurgery 1996; 39: 161 - 164

69 Standards of Practice: The American Society of Interventional and Therapeutic Neuroradiology
Mechanical and Pharmocologic Treatment of Vasospasm
AJNR Am. J. Neuroradiol. (Supplements) 2001; 22: s26 – s27

70 Hendrix LE, Dion JE, Jensen ME, Phillips CD, Newman SA
Papaverine-induced mydriasis
AJNR Am J Neuroradiol 1994; 15: 716 - 718

71 Barr JD, Mathis JM, Horton JA
Severe transient brain stem depression during intraarterial papaverine infusion for cerebral vasospasm
AJNR Am J Neuroradiol 1994; 15: 719 - 723

72 Mathis JM, DeNardo A, Jensen ME, Scott J, Dion JE
Transient neurologic events associated with intraarterial papaverine infusion for subarachnoid hemorrhage-induced vasospasm.
AJNR Am J Neuroradiol 1994; 15: 1671 - 1674

73 Joint Writing Group of the Technology Assessment Committee American Society of Interventional and Therapeutic Neuroradiology; Joint Section on Cerebrovascular Neurosurgery a Section of the American Association of Neurological Surgeons and Congress of Neurological Surgeons; Section of Stroke and the Section of Interventional Neurology of the American Academy of Neurology
Reporting Terminology for Brain Arteriovenous Malformation. Clinical and Radiographic Features for Use in Clinical Trials
Stroke 2001; 32: 1430 - 1442

74 Cognard C, Gobin YP, Pierot L. **Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage.** Radiology 1995; 194: 671 – 680.