SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES
AND TECHNOLOGY
THIRUVANANTHAPURAM

Project Report

Name : **T. Krishnamoorthy**

Programme : **DM Neuroradiology**

Month & Year of Submission : **November 2001**

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Title of the Project:

"Assessment of hemodynamic alterations in Cerebral ArterioVenous Malformations before and after embolisation with Transcranial Color Coded Doppler sonography and feeding arterial pressure measurement"

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CERTIFICATE

I, Dr. Krishnamoorthy hereby declare that I have actually carried out the project **"Assessment of hemodynamic alterations in Cerebral ArterioVenous Malformations before and after embolisation with Transcranial Color Coded Doppler sonography and feeding arterial pressure measurement"** independently under supervision and guidance in the institution.

Thiruvananthapuram,

November, 2001

Signature .....

T. Krishnamoorthy

Forwarded.

He has carried out the above-mentioned project in the department of Radiology, SCTIMST, Thiruvananthapuram.

Signature .....

Prof. A. K. Gupta

Head of the Department of Radiology,

SCTIMST, Thiruvananthapuram.

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At the outset, I thank our director Dr. Mohandas for permitting me to conduct this study.

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T. KRISHNAMOORTHY

INTRODUCTION

Cerebral arteriovenous malformations represent a small proportion of the total incidence of stroke but typically affect otherwise healthy young adults.The main reason to treat cerebral AVMs is the prophylaxis against spontaneous intracranial hemorrhage (35).

The hemodynamics and embolisation of cerebral AVMs are of increasing interest. In most of cases embolisation treatment reduces the size of an AVM to make it treatable by surgery or radiation therapy(10).

It is theorised that during endovascular therapy of arteriovenous malformations and arteriovenous fistulas , the cerebral hemodynamics may change and result in hemorrhage and/or cerebral edema (1). This occurs probably due to a well known but unproven phenomenon called Normal Perfusion Pressure Breakthrough (NPPB) (5).

Currently during the endovascular procedures the end point of therapy is indicated by angiographic means and clinical experience. Embolisation of feeding vessels to AVM is performed in a stepwise fashion. Treatment may require upto three or four separate procedures with the hope that the risk of NPPB and intracerebral hemorrhage is thereby reduced (1).

Several authors have noted that the pressure in the feeding arteries of the AVMs is elevated considerably as obliteration is achieved (10). Transcranial Color Doppler (TCD) may also prove to be a reliable method in assessing the hemodynamic effects of therapy. It is expected that with treatment the mean velocities decrease and the pulsatility indexes increase from the pretreatment levels (44).

This study has been conducted to understand the hemodynamic response following embolisation of cerebral AVMs by Transcranial Color Coded Doppler assessment of blood flow in the basal intracranial arteries and by measurement of mean arterial pressures in the feeders of AVMs before and after embolisation.

REVIEW OF LITERATURE

Cerebral arteriovenous malformations consist of a tangle of vessels of different wall thickness and diameters with associated arteriovenous shunting. These AVMs lack a capillary bed (26). Anatomically the AVM nidus consists of primitive shunting vessels measuring 50 microns to 200 microns (3).

AVMs occur in about 0.02% to 0.05% of the population i.e. roughly one tenth the incidence of intracranial aneurysms. About 90% of AVMs are found in the supratentorial compartment (17) involving most often the middle cerebral artery territory followed by the anterior cerebral artery and the posterior cerebral artery territories.

By 40 years of age about 64% of cerebral AVMs become symptomatic (17). Intracranial hemorrhage is the most common presentation of pial AVMs (19) followed by seizures. AVMs also can cause intractable headache and neurological deficits.

The incidence of hemorrhage from an unruptured AVM is about 2% to 4% per year. Following an episode of bleeding, the risk of bleeding is 6% in the first year which decreases to 2% to 4% subsequently (18-20). The reported death rate from cerebral AVMs is 1% to 2% per year (21). Each bleeding episode from cerebral AVMs carries a 10% to 15% mortality rate(18-20).

The cumulative life time risk of bleed for young patients is substantial (25) and observation is indicated only for the elderly or for lesions that pose a high treatment risk.

The main risk factors for hemorrhage from a cerebral AVM include history of previous hemorrhage (19-21), small size of AVM (22,23), deep venous drainage (22) restricted venous outflow, intranidal aneurysms, diffuse AVM morphology (24) and a higher feeding artery pressure (22,27).

It has been demonstrated that in patients who present with AVM hemorrhage, the feeding artery pressure is significantly higher than in those

without hemorrhage (27). This may explain why small AVMs tend to have higher incidence of hemorrhage compared to larger AVMs.

LUESSENHOP and SPENCE first performed embolisation of cerebral AVMs in 1960 (13). Developed initially by KERBER in 1976,calibrated leak balloon catheters were the first catheters used to navigate intracranial vessels to embolise brain AVMs (14,15)

Even though many embolic agents are available to treat brain AVMs, tissue adhesives are the one currently in favor for embolisation of cerebral AVMs. Because of their ability to penetrate the nidus of an AVM, they offer a solution for permanent occlusion by endovascular means. On the other hand PVA particles and microcoils are most often used to reduce vascularity prior to surgical ablation of an AVM.

Endovascular embolisation of cerebral AVM offers a cure rate of 5% to 10% only (16). So it is most often performed to reduce the size of an AVM, so that it becomes amenable to treatment by surgical removal or stereotactic radiotherapy(10).

The complications associated with embolisation of cerebral AVMs include hemorrhage, stroke, normal perfusion pressure break through, catheter gluing etc.

Endovascular therapy of cerebral AVMs and AVFs can alter the cerebral hemodynamics. This may result in cerebral edema and or hemorrhage (1).

The normal cerebral vessels due to steal effect from AVM or AVF (6,28) lose their autoregulatory capability when exposed to chronically reduced perfusion pressure; following endovascular occlusion of AVM the pressure in these vessels raise to normal levels. However since these vessels have lost their autoregulatory capability , these vessels can't respond appropriately to an increase in arterial pressure and thus hemorrhage and or edema occur. This

phenomenon is called Normal Perfusion Pressure Breakthrough (NPPB). SPETZLER et al proposed this phenomenon first in 1978 (5).

Currently the large cerebral AVMS are not embolised maximally for the fear of causing NPPB (28). Therefore treatment may require upto 3 to 4 separate procedures with the hope that the risk of NPPB and intracerebral hemorrhage is thereby reduced.

In a series of 185 patients with carotid and vertebral fistulae reported by HALBACH et al., five (2.7%) patients developed neurological deficits after fistula closure that were presumed to be caused by NPPB (28).

Hemodynamic measurements of arterial feeders of AVMs have been described previously and were obtained using several methods including Transcranial Doppler, Transcranial Color Coded Doppler and intravascular pressure measurements in an attempt to establish useful criteria of safety and efficacy during embolisation procedure(12).

Monitoring of intravascular pressure is a well established technique used extensively in body angiography(9).Measurement of intracerebral vascular pressures have been obtained previously by direct intra operative puncture of vessels (2,3). These studies have shown that the arteries supplying AVMs had significantly reduced pressure that were elevated markedly after obliteration of the feeding vessels to the AVM .NORNES & GRIP have shown that the pressure in AVM feeders increase by about 60% to normal values with surgical occlusion of these vessels (2).

One study has found that the average pressure elevation in well embolised AVMs was 27mmHg (10). Similarly TCD studies have shown increased flow velocities in the main feeders with decreased pulsatility indices suggestive of reduced resistance in AVMs. Following embolisation the velocities revert back to normal values and the pulsatility indices increase.

Similar to the pressure changes found during intraoperative pressure measurements of arterial feeders, JUNGREIS et al., found changes in pressure via the endovascular route during embolisation therapy (4). They used TRACKER 18 microcatheter to monitor pressure changes.

Later experimental studies were conducted by GARY DUCKWILER et al to validate the intravascular pressure monitoring using microcatheter systems. They established the reliability of mean blood pressure measurements from the microcatheter system (1). Unlike the systolic and the diastolic pressure measurements, measurements of mean blood pressure by various microcatheter systems are quite reliable (12)

Pressure monitoring may be important in the evaluation of endovascular therapy. The data can have important therapeutic implications especially in the post procedure management of systemic blood pressure(1).

It is a long held belief that embolisation procedure for AVMs are risky because of the NPPB phenomenon (5,7). However by using microcatheters to directly measure the intravascular pressures, it is hoped that data can be generated to objectively evaluate the true nature of this phenomenon and the hemodynamics changes responsible for the complications of the therapy (1).

Several investigators have emphasized the importance of arterial feeder pressure measurements during AVM embolisation (1,4,10,29). The assessment of AVM hemodynamics remains an integral step in the planning of treatment in understanding the effects of treatment and in presenting treatment complications (12).

This study is being conducted to enhance our knowledge of hemodynamic behavior of AVMs during embolotherapy.

RELEVANCE TO THE INSTITUTE

SCTIMST is a tertiary care center. It caters to patients requiring specialized neuromedical and neurosurgical care. The Radiology department caters to patients with cerebral arteriovenous malformations, cerebral aneurysms, carotid bifurcation atherosclerotic stenosis, spinal vascular malformations etc.

Approximately around 50 to60 AVMs are dealt with by endovascular embolisation per year. The neurosurgery department caters to a large number of patients with intracranial aneurysms, vascular malformations, brain tumors and various spinal problems. About 25 to 50 AVMs are operated by neurosurgeons in an year.

OBJECTIVE

To assess the hemodynamic changes of the cerebral arteriovenous malformations for proper planning of embolisation, understanding the effects of treatment and in preventing treatment related complications.

MATERIAL & METHODS

From September 2000 to March 2001, 23 patients were included in the study. Summary of patient characteristic, AVM location and size, feeding arteries draining veins is given in Table 1.

Table-1.

Summary of patient characteristic,AVM location and size, feeding arteries, draining veins

Sl No	Age	Sex	Presentation	Location	Size	Venous drainage
1	36	F	Seizure	Left Frontal	Medium	Superficial
2	32	F	Hemorrhage	Right Temporal	Medium	Deep
3	43	M	Seizure	Right Frontal	Medium	Superficial
4	42	F	Seizure	Left Parietal	Large	Superficial
5	34	M	Seizure	Right Frontal	Large	Superficial
6	36	M	Seizure	Left Frontal	Medium	Superficial
7	52	M	Seizure	Right Frontoparietal	Medium	Superficial
8	20	M	Seizure	Left Temporal	Medium	Superficial
9	18	F	Seizure	Corpus callosum	Small	Deep
10	17	M	Hemorrhage	Left Temporo occipital	Medium	Both
11	25	M	Seizure	Left Frontal	Small	Both
12	20	M	Seizure	Left Parieto occipital	Medium	Both
13	54	M	Hemorrhage	Right parietal	Small	Superficial
14	30	M	Seizure	Right basal ganglia	Medium	Superficial
15	20	F	Headache	Right Temporo parieto occipital	Large	Both
16	46	F	Headache	Left Temporo occipital	Medium	Superficial
17	24	F	Hemorrhage	Left Parietal	Small	Deep
18	19	M	Hemorrhage	Cerebellum	Medium	Both
19	45	M	Hemorrhage	Left Temporal	Medium	Both
20	20	M	Seizure	Right Frontal	Medium	Both
21	15	F	Hemorrhage	Right Occipital	Small	Both
22	19	F	Hemorrhage	Basal ganglia	Medium	Deep
23	16	F	Seizure	Right Parieto occipital	Small	Both

The mean age of the patient was 29.7 years with a range of 15 to 54 years.

AVM was classified as small (<3 cms), medium (3 to 6 cms) or large (>6 cms) based on the measurement of maximum diameter in the CT scan. Voluntary consent was obtained from all the patients after explaining the details of the disease and the procedure. Neurology consultation was obtained in all the cases to document the clinical findings.

A day prior to embolisation, flow velocities in the basal intracranial arteries were recorded using 2 MHz Transcranial phased array sector probe through the Transtemporal and Suboccipital windows. TCCD was performed in a calm environment patients were requested to breath as normally as possible. CO_2 measurement was not assessed during TCCD.

Sample volume cursor was placed within the color flow image of the artery under insonation and correction for the angle of insonation by alignment of a cursor along the direction of blood flow was performed. No measurements were made it the angle of insonation was greater than 60 degree.

Mean velocity and pulsatility indexes were recorded. Pulsatility index was obtained using the formula Vs-Ved / Vm [Vs-peak systolic velocity; Ved-end diastolc velocity; Vm-mean velocity].

Next day embolisation of the AVM was performed under general anaesthesia in all the cases. For induction, Thiopentone 5mg/kg was used; Analgesia was obtained with fentanyl 2mg/kg; Maintenance of anaesthesia was achieved with Halothane and Nitrous oxide. The systemic mean arterial pressure was obtained in the radial artery. After placement of the guiding catheter in the cervical ICA or VA, mean arterial pressure was recorded. This was followed by recording of the mean arterial pressure at the same level with a microcatheter.

This was followed by recording of mean arterial pressure in M1, A1 or P2 segments, then in the proximal feeder of the AVM and finally in the feeder close to the nidus. Before measuring arterial pressure, contrast was injected to ensure that the tip of microcatheter was not wedging against the wall of the vessel. After measuring mean arterial pressure, embolisation was performed with glue . In one case absolute ethanol was used for embolisation . All pressure measurements were obtained pressure tranducer – Hewlett Packard.

In majority of the cases multiple embolisations were performed. Mean arterial pressure in the feeding artery stump was measured whenever possible.

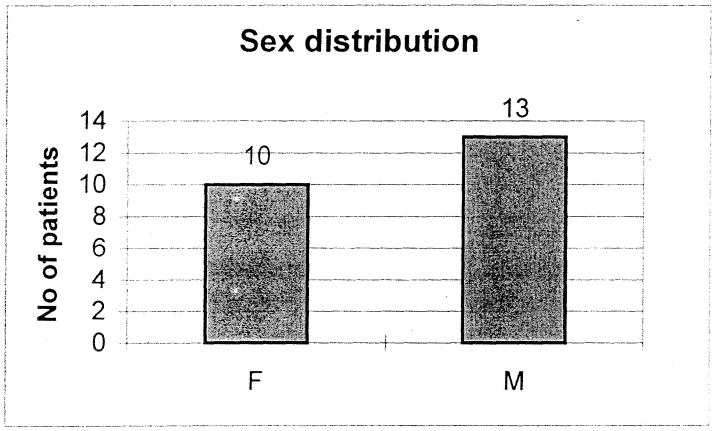
2 days after embolisation, TCCD to measure the velocities and pulsatility indexes of basal intracranial arteries was performed under conditions similar to preembolisation measurement.

Statistical analysis was performed with paired t test.

RESULTS

There were 13 males and 10 females included in this study (fig.1). About 6 AVMs were located in the frontal region,3 in the temporal region, 2 in the temporo-parietal region, 1 in the parieto-occipital region,1 in the temporo-parieto-occipital region, 1 in the basal ganglia,1 in the corpus callosum, 1 in the occipital region and 1 in the cerebellum. 6 AVMs were of small size (<3 cms), 14 were of medium size (3-6 cms) and 3 were of large size (>6 cms).

Figure 1 Sex distribution



Transtemporal and Suboccipital windows provided adequate visualisation of the vessels in all the cases.

In the unaffected hemisphere, basal intracranial arteries showed mean velocities and pulsatility indices which were corresponding to the values reported from the literature (48). The middle cerebral artery showed a mean velocity of 62.5 ± 10.75 cm/s and a pulsatility index of 0.83 ± 0.12. The anterior cerebral artery showed a mean velocity of 50.53 ± 7.42 cm/s and a pulsatility index of 0.78 ± 0.13. The posterior cerebral artery showed a mean velocity of 41 ± 5.93 cm/s and a pulsatility index of 0.83 ± 0.13.

In the affected hemisphere, the MCA showed a mean velocity of 124.62 ± 41.78 cm/s and a pulsatility index of 0.45±0.17 before embolisation showing a significant difference from the unaffected hemisphere (table 2). The ACA

showed a mean velocity of 97.62 ± 37.42 cm/s and a pulsatility index of 0.49 ± 0.19 (table 3). The PCA showed a mean velocity of125.37±20.73 cm/s a pulsatility index of 0.38±0.12 which was significant from the unaffected side (table 4).

Following embolisation the mean velocity in the feeding MCA decreased to 95 ± 41.27 (p value 0.002) and the pulsatility index increased to 0.61 ± 0.19 (p value 0.002) (fig. 2 and 3) (tables 5 and 6).

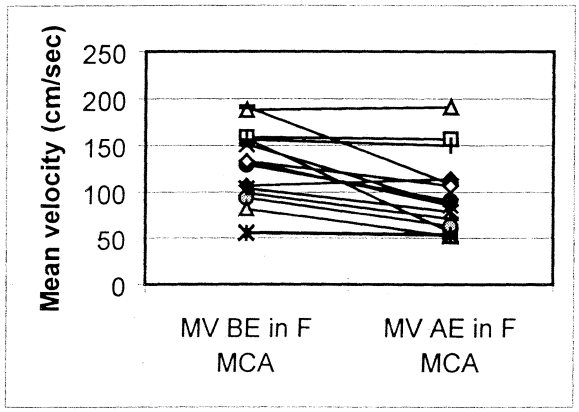


Figure 2 Chart to compare the change in the mean velocity of feeding MCAs before and after embolisation. Following embolisation, mean velocity decreases

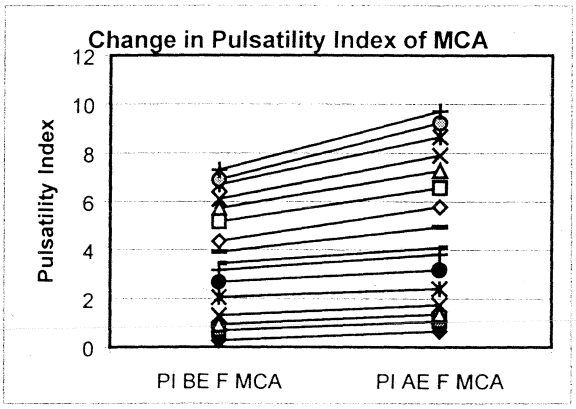


Figure 3 : Chart to compare the change in the pulsatility index of the feeding MCAs before and after embolisation. Following embolisation, the pulsatility index increases

The feeding ACA mean velocity decreased to 64.75 ± 20.26 (p value 0.003) and the pulsatility index increased to 0.67 ± 0.13 (p value 0.012) (fig. 4 and 5).

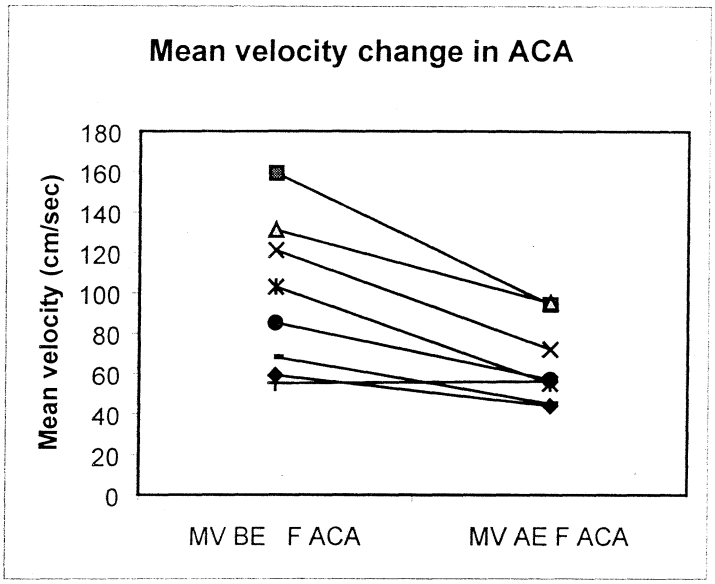


Figure 4 Chart to compare the change in the mean velocity of feeding ACAs before and after embolisation. Following embolisation, mean velocity decreases

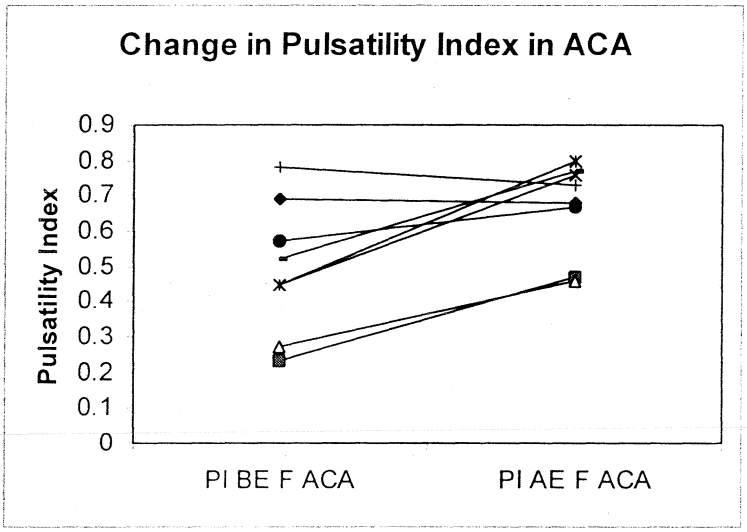


Figure 5 Chart to compare the change in the pulsatility index of the feeding ACAs before and after embolisation. Following embolisation pulsatility index increased

The feeding PCA mean velocities decreased to 82.87 ± 28.26 (p value 0.001)and the pulsatility index increased to 0.59 ± 0.1(p value 0.004) (fig. 6 and 7).

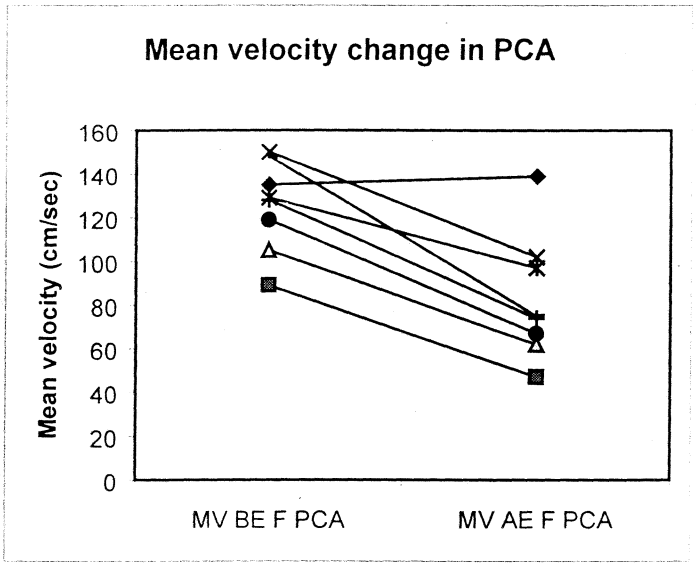


Figure 6 Chart to compare the change in the mean velocity of feeding PCAs before and after embolisation.
The embolised vessels show reduced mean velocity

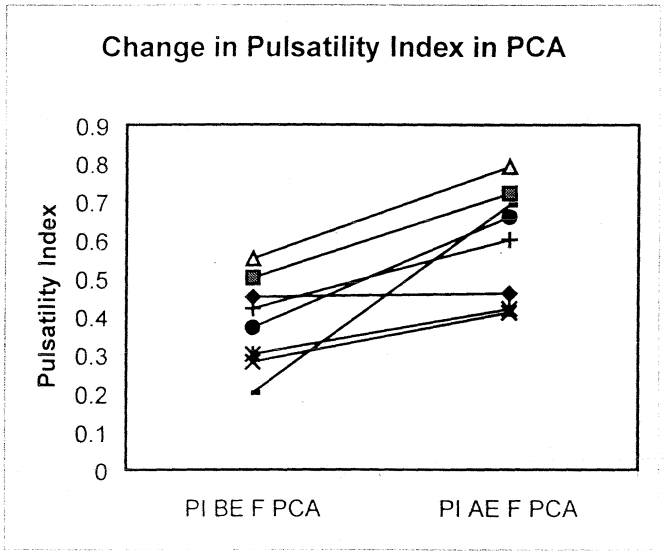


Figure 7 Chart to show the change in the pulsatility index of feeding PCAs before and after embolisation. The embolised vessels show increased pulsating index

Table-2 Comparison of the mean velocities and pulsatility indexes between feeding and nonfeeding MCAs.

	Feeding MCA	Nonfeeding MCA	P value
Mean velocity	124.62 ± 41.78 cm/s	62.5 ± 10.75 cm/s	0.001
Pulsatility index	0.45 ± 0.17	0.83 ± 0.12	0.001

Table-3 Comparison of the mean velocities and pulsatility indexes between feeding and nonfeeding ACAs.

	Feeding ACA	Nonfeeding ACA	P value
Mean velocity	97.62 ± 37.42 cm/s	50.53 ± 7.42 cm/s	0.002
Pulsatility index	0.49 ± 0.19	0.78 ± 0.13	0.001

Table-4 Comparison of the mean velocities and pulsatility indexes between feeding and nonfeeding PCAs.

	Feeding PCA	Nonfeeding PCA	P value
Mean velocity	125.37 ± 20.73 cm/s	41.5.93 ± 5.93 cm/s	0.001
Pulsatility index	0.38 ± 0.12	0.83 ± 0.13	0.001

Table-5 Comparison of the mean velocities in the feeding vessels before and after embolisation

	Mean velocity before embolisation	Mean velocity after embolisation	P value
MCA	124.62 ± 41.78 cm/s	95 ± 41.27 cm/s	0.002
ACA	97.62 ± 37.42 cm/s	64.75 ± 20.26 cm/s	0.003
PCA	125.37 ± 20.73 cm/s	82.87 ± 28.86 cm/s	0.001

Table - 6 Comparison of the pulsatility indexes in the feeding vessels before and after embolisation

	Pulsatility index before embolisation	Pulsatility index after embolisation	P value
MCA	0.45 ± 0.17	0.61 ± 0.19	0.002
ACA	0.49 ± 0.19	0.67 ± 0.13	0.012
PCA	0.38 ± 0.12	0.59 ± 0.15	0.004

Mean velocities were found to be higher in the feeders to medium and large AVMs as compared to that of small AVMs. In one of the small AVMs, the mean velocity and the pulsatility index were within normal range in the MCA and no significant difference was found with the contralateral MCA (case no.13)

No significant difference was found in the mean velocities and the pulsatility indexes of the arteries supplying the unaffected hemisphere (tables –7 and 8).

Table-7 Comparison of the mean velocities in the nonfeeding arteries before and after embolisation

	Mean velocity Before embolisation	Mean velocity After embolisation
MCA	65.95 ± 10.50 cm/s	66.26 ± 9.76 cm/s
ACA	50.53 ± 7.42 cm/s	49.62 ± 4.68 cm/s
PCA	41 ± 5.93 cm/s	42.07 ± 7.08 cm/s

Table-8 Comparison of pulsatility indexes in the non feeding arteries before and after embolisation

	Pulsatility index before emblisation	Pulsatility index after embolisation
MCA	0.80 ± 0.12	0.79 ± 0.11
ACA	0.78 ± 0.13	0.0.78 ± 0.0.13
PCA	0.83 ± 0.13	0.83 ± 0.15

No significant difference in the pressure measurement was observed between the guiding catheter mean arterial pressure measurement (80.91 ± 7.03 mmHg) and the microcatheter mean arterial measurement (82.06 ± 6.85 mmHg) either in the cervical internal carotid artery or in the vertebral artery (p value >0.05).

Table-9 Comparison of mean arterial pressure measurement with guiding catheter and microcatheter at the neck level

	Guiding catheter	Microcatheter	P value
Mean arterial pressure in ICA or Vertebral artery at the neck level	80.91 ± 7.03 mmHg	82.06 ± 6.85 mmHg	>0.05

Gradual reduction in the mean arterial pressure measurement was observed from the level of the cervical ICA or the vertebral artery to the level of the nidus. Maximum mean arterial pressure reduction was observed in the feeder near the nidus (fig 8).

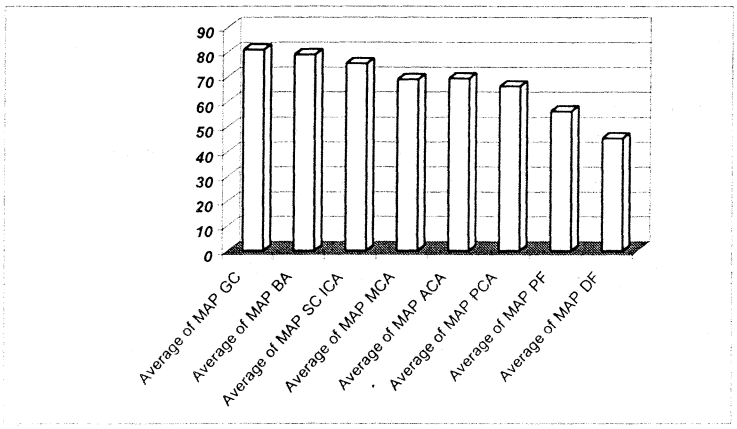


Figure 8 Gradual reduction in the mean arterial pressure from the level of the vessel in the neck to the level of the feeder closer to the nidus in cerebral AVMs.

The feeding artery mean pressure was significantly lower compared to the mean systemic arterial pressure (82.78 ± 6.64 mmHg) in the medium (38.64 ± 6.25 mmHg) and large AVMs (37.33 ± 11.02 mmHg) . The feeding artery mean pressure near the nidus in small AVMs was higher (65.33 ± 8.76 mmHg) compared to that of the medium size and large AVMs.. The mean of

ratio of systemic mean arterial pressure to the feeding mean arterial pressure in the medium size AVMs was 43.46 ± 10.24 mmHg. In the large AVMs it was 43.67 ± 5.13 mmHg. In the small AVMs it was 18.33 ± 10.94 mmHg (Fig 9). The average mean arterial pressure in the feeding arteries close to the nidus was 45.43 ± 14.06 mmHg. Following embolisation, the mean arterial pressure in the stumps of the feeders increased significantly to 66.69 ± 10.70 mmHg (p value 0.000).

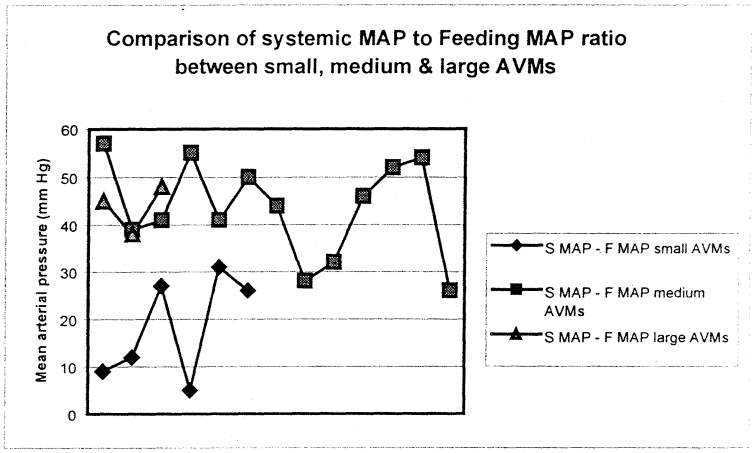


Figure 9 The systemic mean arterial pressure to the feeding mean arterial pressure ratio is lower in the small AVMs compared to the large and medium AVMs.

DISCUSSION

Cerebral arteriovenous malformations are congenital anomalies that consist of abnormal arteries and veins without an intervening capillary bed. Large autopsy series have shown the incidence of AVMs to be between 0.04% and 0.52% (30, 31). About 90% of AVMs are found in the supratentorial compartment (17).

By 40 years and age about 64% of cerebral AVMs become symptomatic (17). Intracranial hemorrhage is considered as the most common presentation of the cerebral AVMs followed by seizures (19). In addition by can cause intractable headache and focal neurological deficits.

The incidence of hemorrhage from an unruptured AVM is about 2% to 4% per year. Following an episode of bleeding, the risk of bleeding becomes 6% in the first year following hemorrhage. It decreases to 2% to 4% subsequently (18-20).

Each bleeding episode from cerebral AVMs carries a 10% to 15% mortality rate (18-20) and 30 to 50% morbidity rate (36).

The cumulative life time risk of bleeding for young patients is substantial (28) and treatment of cerebral AVMs is strongly advocated (32-34) in all patients except in the elderly in whom they may be treated conservatively.

Currently cerebral AVMs are treated by the surgical excision, endovascular embolisation, radiosurgery or a combination of endovascular embolisation plus surgery or endovascular embolisation plus stereotactic radiosurgery.

Endovascular embolisation has become an accepted adjunct or alternative therapy in the treatment of brain AVMs (36,37). Endovascular embolisation of cerebral AVMs offer a cure rate of 5% to 10% only (36,37).

Hence it is often performed to reduce the size of an AVM so that it becomes amenable to treatment by surgical removal or stereotactic radiosurgery (10).

The hemodynamics and embolisation of cerebral AVMs are of increasing interest(10).Various methods have been described to assess the hemodynamics of the cerebral AVMs. The feeding mean arterial pressure could be assessed by cannulating the feeder closer to the nidus at surgery with a 26/27G needle (2,27). The mean arterial pressure could also be assessed via the microcatheters used for embolisation (1,49).

Similarly the flow velocities and the pulsatility indices in the basal intracranial arteries would be assessed with Transcranial Doppler studies and the Transcranial Color Coded Doppler studies.

In contrast to the normal vasculature, where the arteriolar site is mainly responsible for the cerebrovascular resistance and its regulation, AVM feeders have a very low cerebrovascular resistance and loss of blood flow autoregulation (38).

An AVM is a region of very low cerebrovascular resistance producing abnormally decreased pressure values in proximal segments of feeders. Despite the low pressure level, blood flow in AVM feeders is high because of the low peripheral resistance (39).

Qualitative studies of TCD findings in patients with arteriovenous malformations have been described (40-42). Feeding arteries of AVMs typically have higher than normal mean blood velocities with turbulence and lower than normal pulsatility indexes. TCD is useful in diagnosing AVMs noninvasively and in identifying major feeding arteries (40,43).

TCD may prove to be a reliable method in assessing the hemodynamic effects of therapy. R. Aaslid et al., were the first to record flow velocity in basal cerebral arteries non invasively with Transcranial Doppler ultrasound.

G.W. Petty et al., (44) performed Transcranial Doppler ultrasonography on 15 patients with AVMs before and after embolisation or surgical resection. They found that the mean blood velocity decreased by a mean of 38.1% or 46.5 cm/s; Decreases were greater for surgically resected arteries than for embolised arteries. Pusatility index increased by a mean of 54.7% or 0.25; Again Pulsatility index increases were greater for surgically resected cases than for embolised arteries. From their data, they concluded that embolisation resulted in hemodynamic changes that are qualitatively similar to those occurring after surgical resection of AVMs (44).

Karl Frederick et al., (43) assessed blood flow velocities in basal arteries noninvasively with TCD and found significant increase in mean blood flow velocities in the feeding arteries (median 124 cm/s in the feeding arteries Vs median 65cm/s in the nonfeeding arteries)and significant decrease in the pulsatility indexes (median 0.48 in the feeding arteries Vs median 0.87 in the non feeding arteries).

Rolf R. Diehl et al (39) performed TCD in 18 patients with untreated AVMs and found a pathological increase in blood flow velocity (57.6%) and a decrease in vasomotor reactivity (72.7%).

H. Mast et al., (45) examined 114 consecutive AVM patients prospectively by TCD; they grouped AVMs into small, medium and large size based on the maximum diameter. They observed that velocities fell with decreasing AVM size, reaching the normal range in small AVMs. Pulsatility indexes rose with decreasing AVM size. They reported that sensitivity of TCD for large and medium size AVMs was high (>80%) whereas 62% of small AVMs were missed.

In our study we found that the mean velocities and the pulsatility indexes in the vessels supplying the unaffected hemisphere were corresponding to the values reported from the literature (46,48).Those vessels supplying the cerebral AVMs showed increased mean velocities and decreased pulsatility

indexes (tables 2,3 & 4). Following embolisation the mean velocity showed significant reduction and the pulsatility index showed significant increase (tables 5 & 6).These findings are consistent with the results reported the from the literature (44).

Gary Duckwiler et al., (1) with the use of Tracker and Balt microcatheter systems,measured the intravascular pressure in an experimental animal model and established the reliability of mean blood pressure measurements from the microcatheter system. In our study, we also found that the measurement of mean arterial pressure through the microcatheter was matching close with the measurement of the guiding catheter at the neck level either in the internal carotid artery or the vertebral artery. This finding establishes further the reliability of using the microcatheter system to measure the mean arterial pressure in the intracranial vessels at superselective catheterization.

Patricia Fogarty Mack et al., (49) recorded mean arterial pressures during superselective cerebral angiography in 96 patients with AVMs with use of a system of vascular zones and found a progressive and significant decrease in intracerebral arterial pressure in patients with AVMs that proceeded from the circle of Willis to the nidus.

They further assessed mean arterial pressure in the cortical branches of the contralateral hemisphere and found that the ratio of this normal cortical branch mean arterial pressure to systemic mean arterial pressure was 0.78 ± 0.12 where as the ratio of feeding mean arterial pressure to systemic mean arterial pressure was 0.50 ± 0.18. They mentioned several investigators have determined distal cortical arterial pressures (pial or M5 level) to be approximately 90% of systemic pressure in patients without occlusive cerebrovascular disease.

In our study , we found similar changes of gradual reduction in of mean arterial pressure from the level of cervical internal carotid artery /

vertebral artery to the level of the feeder close to the nidus of the AVM. Maximum reduction in the feeding artery mean arterial pressure was found close to the nidus (table-9).

Higher feeding artery pressure is considered as one of the risk factors predisposing to hemorrhage in AVMs. (22,27). Robert F. Spetzler et al., (27) prospectively evaluated 92 AVMs for nidus size, hematoma size and arterial feeding pressure and found that small AVMs (diameter $\leq$ 3 cms) presented with hemorrhage significantly more often than large AVMs (diameter >6cms) the incidence being 82% Vs 21%. Further they found that in the AVMs that had hemorrhaged the mean difference between mean arterial blood pressure and the feeding artery pressure was 6.5 mmHg (range 2 to 15 mmHg) and in the AVMs that did not rupture this difference was 40 mmHg (range 17 to 63 mmHg). They further observed that smaller AVMs had significantly higher feeding artery pressures than did large AVMs and they were associated with large hemorrhages.

Helge Nornes et al., (2) studied local hemodynamics of cerebral AVMs in 16 patients undergoing extirpation. They assessed blood velocities using directional Doppler Technique and calculated the blood flow. They further assessed feeding artery pressures at their entrance to the AVM and found that the pressure was well below the systemic arterial blood pressure in all cases and ranged from 40 to 77 mmHg (average 56 mm Hg). On temporary occlusion, the stump pressure instantly rose to 55 mmHg to 95 mmHg (average 76 mmHg). Further they measured the draining vein pressure before occlusion and it ranged from 8 to 23 mmHg (average 15 mmHg) and fell to zero in all patients when the AVM was occluded.

Yoshio Miyasaka et al., (46) studied the correlation between intraoperative pressure levels measured in the feeding arteries and in the draining veins and the risk of hemorrhage from AVMs. They found that the feeding artery pressure was significantly higher in AVMs with hemorrhage

(57 ± 11 mmHg) than in AVMs without hemorrhage (38 ± 4mmHg) and the draining vein pressure was significantly higher in the former (24 ± 5mmHg) than that in the later (13 ± 5mHg). Feeding artery pressure and draining vein pressure were inversely related to the number of draining veins and the size of the AVMs. Their study supported the previous reports that small AVMs and AVMs with only one draining vein are susceptible to hemorrhage.

D.Hoang Duong et al., (35) after reviewing clinical and angiographic data from 340 patients with cerebral AVMs from a prospective data base, concluded that high arterial input pressure (FMAP) and venous outflow restriction (exclusively deep venous drainage) were the most powerful risk predictors for hemorrhagic AVM presentation than the size, location of AVMs or presence of aneurysms.

Alexander M. Norbash et al., (48) measured superselective transcatheter feeding arterial pressure and mean arterial pressure before embolotherapy in 32 patients with cerebral AVMs. They found that the feeding arterial pressure and feeding arterial pressure / mean arterial pressure ratios were significantly decreased in patients with angiomatous change. Feeding arterial pressure and feeding arterial pressure / mean arterial pressure ratio progressively decreased as lesions went from peripheral, to mixed, to central venous drainage. A trend for lower feeding arterial pressure also was demonstrated with greater feeding pedicle length. They could not demonstrate a statistically significant correlation between feeding arterial pressure or feeding arterial pressure/ mean arterial pressure ratios and size of the AVMs, hemorrhage or symptoms of steal.

In our study 6 of the 23 AVMs were of small size; Out of the 23 patients , 8 patients presented with hemorrhage and of the 8 patients 3 were with small AVMs i.e., 50% of the small AVMs presented with hemorrhage.Eventhough this study consists of small number of patients it highlights the reports from the literature that small AVMs are more likely to

bleed. Our study further showed that the mean arterial pressure in the feeders of the small AVMs was higher (65.33 ± 8.76 mmHg) compared to that of large (37.33 ± 11.02 mmHg) and medium sized AVMs (38.64 ± 6.25 mmHg).This high feeding artery pressure may be one of the factors responsible for more frequent presentation of small AVMs with hemorrhage.

Charles A. Jungreis (4) et al., recorded the mean arterial pressure in the feeders to the AVMs during therapeutic embolisation of cerebral AVMs and found that the feeding arterial pressures increased gradually as the distal runoff became less during embolisation. They further reported that monitoring feeding, arterial pressures may provide better assessment of progress of embolisation than the visual appearance at angiography. It may be very useful to know immediately when a pressure change occurs so that an adequate total volume of embolus can be administered.

Takashi Handa et al., (10) measured the pressure in 47 arteries feeding AVMs with Tracker-18 microcatheter and found that on average systolic pressures increased by 22mmHg.They further noted that in AVMs with single or few feeders , embolisation was usually achieved well; in contrast giant AVMs with multiple feeders and large arteriovenous shunts were poorly embolised. They reported that large AVMs with well demarcated components may be reduced by embolisation to an appropriate size for surgery or stereotactic radiation therapy. They concluded that measurement of feeding artery pressure clarified the hemodynamics of AVMs and facilitated more successful embolisation.

In our study, we found that the mean arterial pressure in the feeders to the AVMs was low and this mean arterial pressure started increasing gradually during embolisation. In the stumps of completely embolised feeders significant increase in the mean arterial pressure was found from the preembolisation status.

CONCLUSION

Our study has shown that the mean arterial pressure in the feeders to the cerebral AVMs were low and following embolisation they showed significant increase. The Transcranial Color Coded Doppler showed high mean velocities and low pulsatility indexes in the basal intracranial arteries supplying the AVMs. Following embolisation, the mean velocities decreased and the pulsatility indexes increased significantly.

A knowledge of the hemodynamics may help us to understand the phenomenon of Normal Perfusion Pressure Breakthrough better and to avoid complications of intracerebral hemorrhage and edema following treatment. Further it may help us to assess the volume of the embolic material necessary to occlude the nidus and the end point of the procedure.

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PROFORMA

1. Name
2. Age
3. Sex
4. Hospital Number
5. Clinical Presentation — Hemorrhage / Seizure / Head Ache / Focal Deficits
6. Location of AVM
7. Size of the Nidus on CT — <3 cms / 3 – 6 cms / > 6 cms
8. Findings on DSA
   a) Feeder
      Number
      Aneurysm
   b) Nidus
      Aneurysm
   c) Draining Veins
      Number
      Superficial / Deep
      Venous Stenosis / Sac
9. Percentage of Nidus occlusion

10. Findings of TCCD

	PSV		EDV		MV		PI	
	BE	AE	BE	AE	BE	AE	BE	AE
RCICA								
RSCICA								
RM1								
RA1								
RP2								
LCICA								
LSCICA								
LM1								
LA1								
LP2								
BA								
RV4								
LV4								

11. Mean Pressure Measurements

	BE	AE
Systemic Pressure		
Guide Catheter at Cervical ICA / VA		
Microcatheter at Cervical ICA / VA		
RSCICA		
RM1		
RA1		
RPCA		
Proximal feeder		
Feeder close to the Nidus		
LM1		
LA1		
LPCA		
BA		

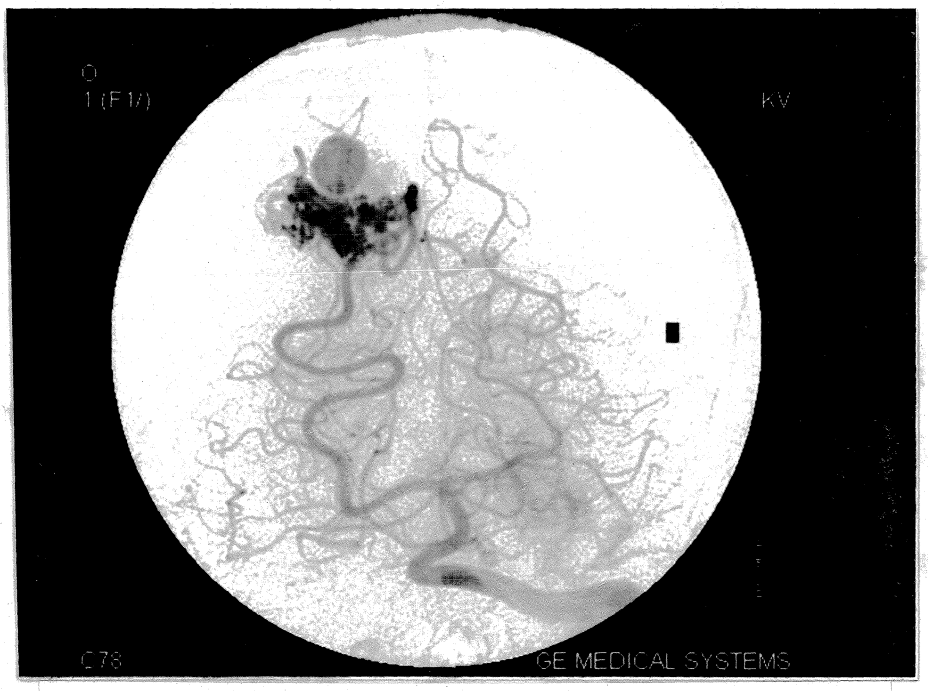

Fig-1 Right parieto-occipital AVM before embolisation.

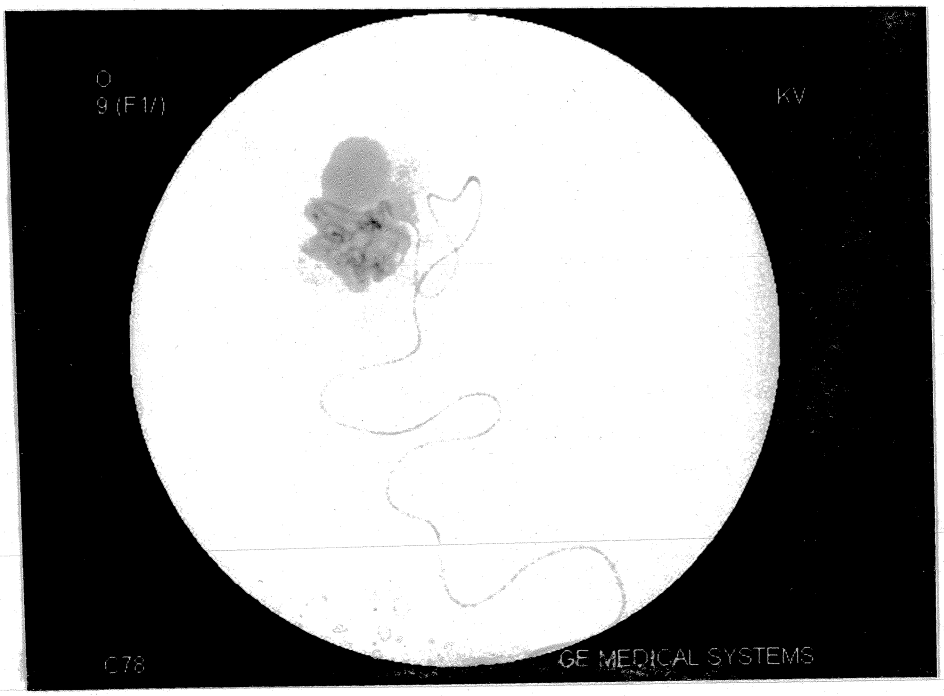

Fig-2 Superselective catheterisation. Pre-embolisation feeding mean arterial pressure close to the nidus measured 50mmHg; Systemic mean arerial pressure measured 90mmHg.

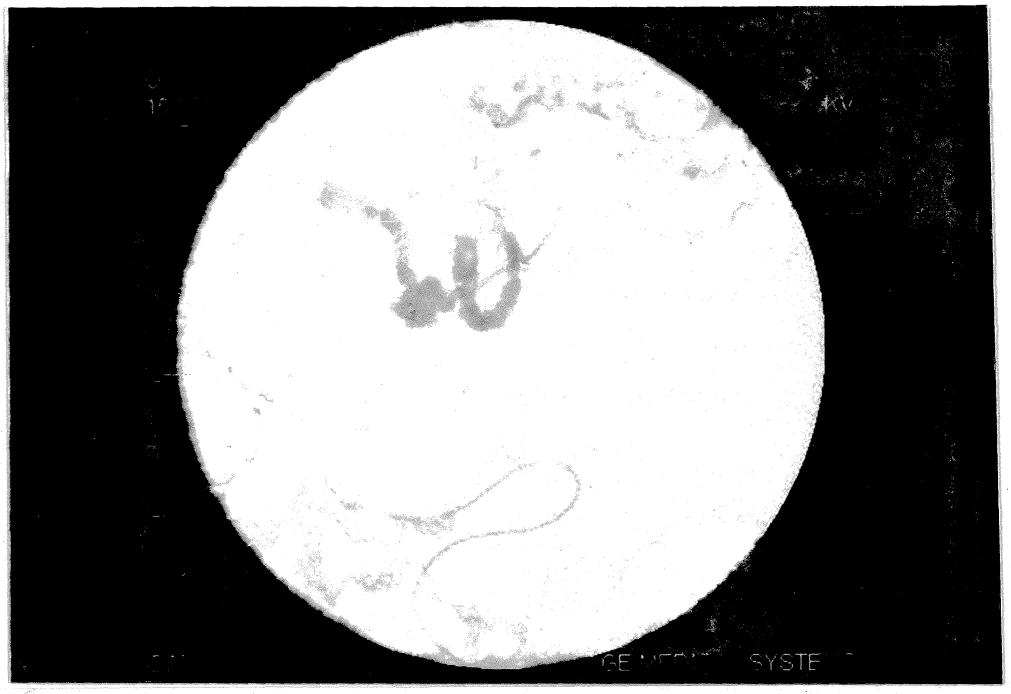

Fig-3 Superselective catheterisation. Postembolisation feeding mean arterial pressure close to the nidus measured 64 mmHg; Systemic mean arerial pressure measured 90mmHg.

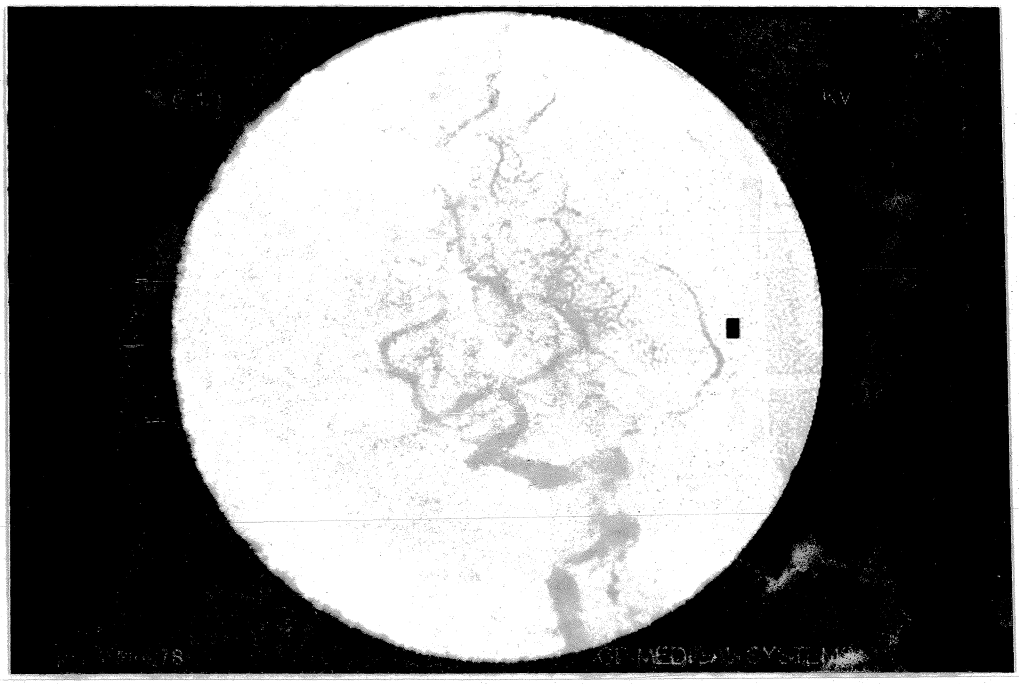

Fig-4 Postembolisation DSA; Arterial phase image shows obliteration of the nidus.

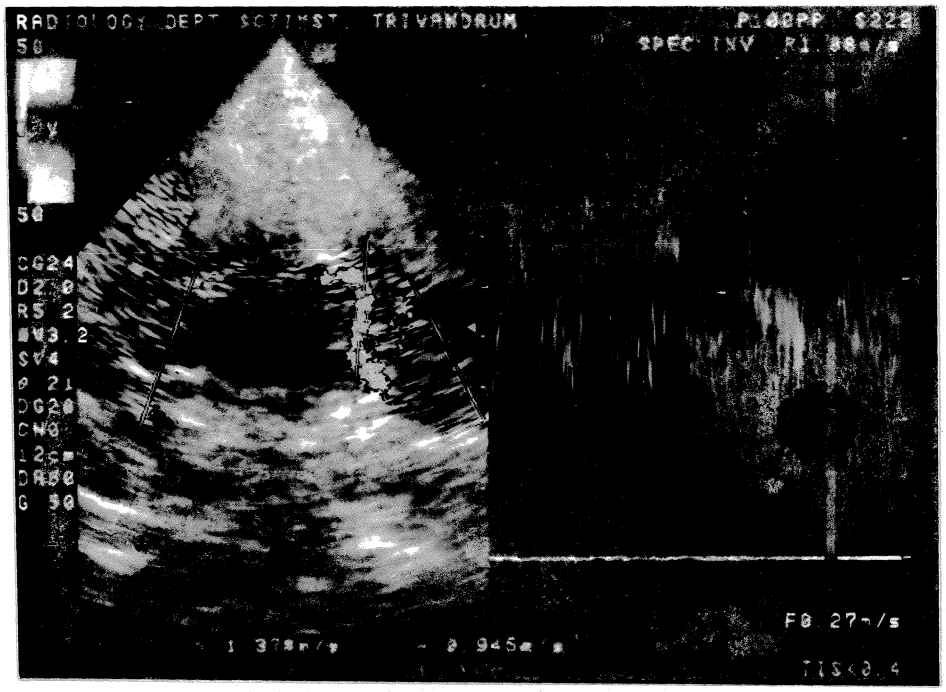

Fig-5 Preembolisation TCCD image. Sampling at P3 segment of right PCA shows Peaksystolic velocity of 137.8 cm/sec , Enddiastolic velocity of 94.5 cm/sec, Mean velocity of 109 cm/sec and Pulsatility index of 0.39 .

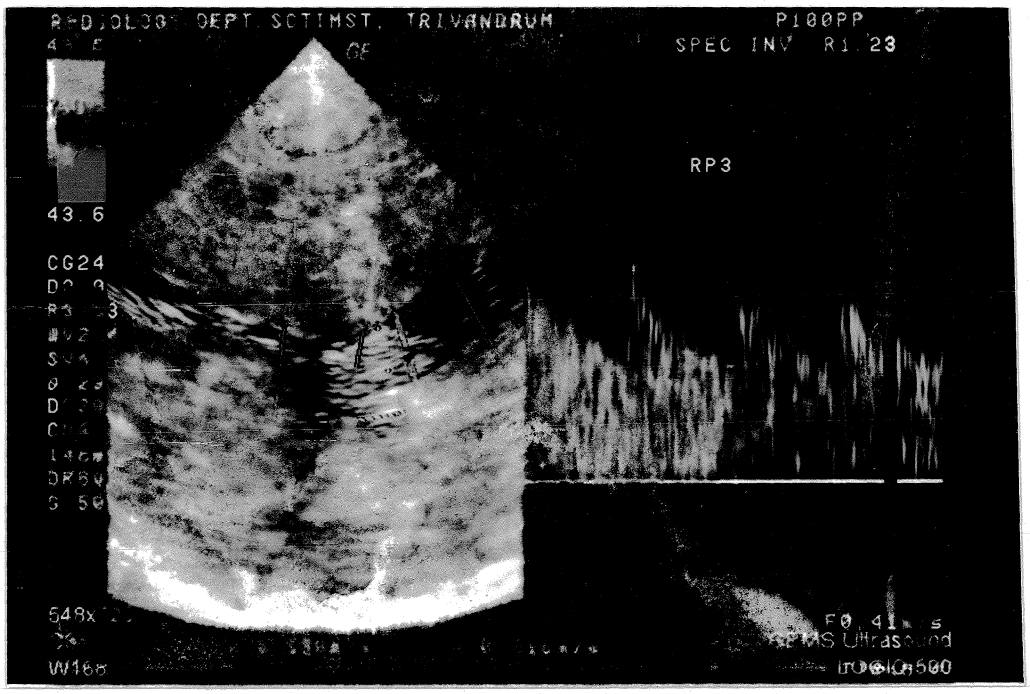

Fig-6 Post embolisation TCCD image. Sampling at P3 segment of right PCA shows Peaksystolic velocity of 58 cm/sec , End-diastolic velocity of 31.5 cm/sec , Mean velocity of 41 cm/sec and Pulsatility index of 0.63