Carotid Stenting in Stroke Prevention : Present Status

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Abstract : *Stroke, one of the leading causes of disability and a major cause of loss of vocational ability, remains a major health problem. Large vessel disease accounts for about 30%-40% of all strokes. Atherosclerosis is the most common cause of extracranial cerebral arterial stenosis and carotid artery stenosis accounts for 20%-30% of ischaemic strokes. Studies show that carotid endarterectomy and carotid angioplasty and stenting can decrase the risk of stroke.*

INTRODUCTION

Stroke continues to remain a major health problem. In the West, it is the third most common cause of death after heart disease and all cancers. It is also one of the leading causes of disability and a major cause of loss of vocational ability. Each year, more than 600,000 men[1]. The average annual incidence of stroke in Japan is 3.94 for males and 2.52 for females per 1000 population, with cases of cerebral infarction outnumbering those of cerebral haemorrhage[2]. Fourteen per cent of people who have a stroke or transient ischaemic attach (TIA) will have another within a year. Approximately 30% of stroke survivors require assistance for the activities of daily living and about 70% have impaired vocational ability. Large vessel disease accounts for about 30%-40% of all strokes. Carotid artery (CA) stenosis, particularly involving the CA bifurcation region, alone accounts for 20%-30% of ischaemic strokes[3-5]. In this group of patients, the disease can be modified and the risk of stroke minimized by intervention.

CAROTID ENDARERECTOMY

The benefit of carotid endarterectomy (CE) for the prevention of stroke in patients with extracranial CA stenosis has been well established in randomized trials. The landmark studies of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and the Asymptomatic Carotid Atherosclerosis Study (ACAS), have proved that CE is beneficial in reducing the risk of stroke for symptomatic and asymptomatic patiens with significant CA stenosis[6-9]. For patients with recently symptomatic CA stenosis, the NASRT[6] demonstrated the efficacy of CE in preventing stroke in patients with 70%. 99% angiographic stenosis with a 2 year absolute risk reduction of 17%. In those with 50%-69% stenosis, there was a modest benefit with an absolute risk reduction of 6.5% at 5 years[10]. In the moderate category, only certain subgroups of patients experienced benefit, such as men and patients with hemispheric ischaemia. There was no demonstrable benefit of CE in patients with <50% stenosis[10]. Asymptomatic patients with extracranial CA stenosis have a lower risk of developing a stroke.

The ACAS[7] evaluated patients with > 60% stenosis. The 5-year natural history risk of ipsilateral stroke was shown to be 11% (annual rate 2.2%). CE reduced the 5- year risk of stroke to 5.1% (annual rate 1.0%). This benefit was achieved with a perioperative risk of stroke and death of 2.3%. For asymptomatic CA lesions, men lead a 66% reduction in the incidence of stroke over 5 years, whereas for women, there was a reduction of only 17%. The threshold of perioperative mortality and morbidity for achieving benefit either by surgical or interventional methods in symptomatic patients is approximately 6% and that for asymptomatic patients is 3%.

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There is controversy regarding the risk of CA surgery in day to day practice, which may not be reflected in the highly selected population of patients and surgeons represented by randomized trials. Wennberg et al[11] analyzed the mortality results for all Medicare patients (n=113,300) undergoing CE during the same period that the NASCET was being conducted (Table-1). They found that the 30-day mortality rate (1.75%) in the Medicare population was three times higher than that reported in randomized trials (0.6%). The 30 day mortality rate for Medicare patients at the trial hospitals was 1.44% compared with 0.6% reported in the NASCET study. The perioperative mortality in the non-trial hospitals was 1.77%. Given the higher mortality results in the Medicare population, whic represents the majority of patients undergoing CA surgery, the authors argue that the results of CA surgery in highly selected patients and performed by highly selected surgeons are not representative of everyday practice.

CAROTID ANGIOPLASTY AND STENTING

Early studies on carotid angioplasty and stenting (CAS) have shown promising results and CAS is emerging as a popular alternative to endarterectomy for the tratment of stenotic diseases of the CA. Endovascular stenting of the CA provides an alternative therapeutic option for patients with atherosclerotic CA stenosis, but poses a high surgical risk because of significant coexisting morbidity. It can also be offered to those with surgically inaccessible or tandem CA lesions, contralateral CA occlusion and post-endarterectomy restenosis. Non-atherosclerotic occlusive disease of the CA due to radiation therapy, fibromuscular dysplasia, spontaneous or traumatic initimal dissection of the cervical and intracranial CA can also be treated effectively using CAS.

There are many non-randomized CAS case series, which involve several hundred patients. At present, a larger overview of the performance of CAS can be obtained from a recent global survey of interventionists[12]. In this multicentre survey with self-reported data, 5210 vessels were stented (Tables-1 and 2). The overall rate of stroke and death was 5.07% and the rate of major stroke was 1.5%. The authors found a higher compliction rate in symptomatic patiens, with a stroke and death rate of 5.8% compared with 3.4% in asymptomatic patients. If these figures reflect the performance capability of CAS in the real world, then CAS would seem a viable alternative to CE. However, it should be stressed that for asymptomatic patients any procedure that carries a stroke risk >3% is likely to be worse than medical treatment alone. For this reason, we would at present recommend avoidance of intervention in asymptomatic patients.

Table - 1 *Results of carotid stenting : 30 day complications*

Study	Arteries (n)	Success rate(%)	All stroke death (%)	Major stoke rate(%)	Death (%)
Wholey et al[12]	5210	98.4	5.07	2.15	0.79
Yadav[13]	25	100	4.0	0	0
Hussain et al[24]	96	99	1	0	0
Bergeron et al[48]	99	NA	1	0	0
Gupta et al[49]	100	100	5	1	0
Meriole[51]	26	100	0	0	0

CAROTID ENDARTERECTOMY VERSUS CAROTID ANGIOPLASTY AND STENTING

The results of a few clinical trials comparing CE and CAS are available. These are the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS)[13] and Stent and Angioplasty Protection in Patients at High Risk of Endarterectomy (SAPPHIRE)[14].

In the CAVATAS trial, 504 patients with proximal internal carotid artery (ICA) atherosclerosis were randomly assigned to treatment with either CE or carotid angioplasty. Stenting was not mandatory and stents were used in only 26% of the patients. A vast majority of the patients (96.5%) were symptomatic and had severe stenosis (mean : 86.5% by the common carotid method). The patients had a greater prevalence of baseline coronary artery disease (36%) than the European Carotid Surgery Trial Population (24%)[13].

There was no difference at all in the occurrence of periprocedural stroke or death between the CE (9.9%) and the angioplasty (9.9%) groups. In terms of major stroke and death rate, again there was no significant different, the rates being 6.4% in the angioplasty group and 5.9% in the CE cohort. It is worth noting that the complications rates reached such high levels in both the CE (10%) and the angioplasty (9.9%) groups that it is likely no benefit would have been conferred beyond medical treatment alone.

With regard to other minor complications, some of the inherent benefits of angioplasty were borne out. Because there is no need for a neck incision, there was a lower incidence of cranial nerve palsies (none in the angioplasty group, 8.7% in the CE group) and would haematomas (1.2% in the angioplasty group, 6.7% in the CE group). With regard to cranial nerve palsies, many surgical series have shown that in the majority of instances, cranial nerve dysfunction after CE is transient. In the NASCET, for example, only 10 of 122 cranial nerve palsies (8%) were moderate in severity, which was defined as requiring a delay in discharge, readmission, or the absence of recovery[6]. The SAPPHIRE randomized trial compared CE with carotid stenting using embolic protection devices[15]. This study randomized high-risk patients to CE or carotid stenting using the PRECISE stent and the Angioguard embolic protection filter (both from Cordis, Maiami Lakes, FL). In this small study there were fewer events in the stent arm as compared with the CE arm. The composite end point of death, stroke and MI at 30 days fell from 12.6% in the CE arm to 5.8% in the stent arm (p=0.047). Each individual end-point was in favour of stenting. The benefit of stenting was seen in both symptomatic and asymptomatic patiens. The incidence of cranial nerve injury was 5.3% in the CE arm and none in the stent arm (p<0.01).

SAFETY AND EFFICACY COMPARISON

A randomized clinical trial, the Carotid Revascularization Endarterectomy Versus Stent Trial (CREST)[16] has standardized definitions of outcome events and re-stenosis, and will help to determine the safety and efficacy of stenting in a controlled fashion. Though it is relatively difficult to compare CA stenting with endarterectomy without a randomized trial, some early comparisons can be made. If all causes of death within a 30 day period of CAS are included, the stroke and death rate for carotid stent placement is 6.29%. Hence, the risk of carotid stent placement is at par with the American Heart Association's (AHA) guidelines for CE : <6% for patients with TIA and <7% for patients with symptomatic stroke[17]. Safety and Efficacy of Endovascular Treatment of Carotid Artery Stenosis versus Carotid Endarterectomy was compared in a recent review[18]. Five trials involving 1269 patients were included. No significant difference in the major risks of treatment (treatment related death or any stroke) was found at 30 days and 1 year while minor complication rates favoured endovascular treatment.

RESTENOSIS AFTER REVASCULARISATION

The incidence of recurrent carotid stenosis after CAS is important in judging the benefits of CAS apart from the periprocedural complications. In a systematic review of the literature, the cumulative incidence of recurrent stenosis (for 50% stenosis) after CEA was about 10% in the first year[19]. Similarly, an early restenosis rate of 7.6% to 11.4% was found within 18 months of CEA in the ACAS[20].

Much lower restenosis rates for >50% after CAS (2.70% after 12 months, 2.60% after 24 months, and 2.40% after 36 months) have been reported in the largest multicenter survey to date[21]. In another recently published review on early recurrent stenosis after CAS[22], it was found that, the cumulative restenosis rates after 1 and 2 years were 6% and 7.5% in those studies, which used a lower restenosis threshold >50% to 70% and 4% in the first 2 years after CAS in those studies, which used a lower restenosis thereshold > 70% to 80%.

To date, the Carotid and Vertebtral Artery Transluminal Angioplasty Study (CAVATAS)[13] is the only published prospective multicenter trial that directly compared early carotid restenosis rates after endovascular versus surgical treatment. However, in CAVATAS most patients were treated with angioplasty alone without use of stent, so that these results do not reflect the risk of recurrent stenosis after CAS. Thus the available literature shows a comparable or better early restenosis rates for CAS and CEA[22].

CLINICAL AND COST COMPARISON OF REVASCULARIZATION

The cost of treatment is another factor of concern when different treatment modalities are compared. In a study, William A. Gray et al[23] compared the clinical outcome and cost of treatment in 136 endarterectomies Vs 136 carotid stent procedures. The primary clinical outcome measures were in hospital major ipsilateral stroke and death, while primary economic measures were in hospital direct variable costs and length of stay. These nonrandomized groups were similar, but the endartectomy group had more symptomatic patients (42% versus 31%; p=0.0004), and the stent group had more NASCET excluded patients (68% versus 35%; p<0.0001). In hospital major ipsilateral stroke and death occurred more frequently in the surgical group, but the difference was not significant (2.9% vesus 0%;

p=0.1). Minor ipsilateral strokes werer similar. Cost ($5409 versus $3417; p<0.0001) and length of stay (3.0 versus 1.4 days; p<0.0001) were significantly greater for the surgical group.

Cost and resource utilization with stenting appear to be substantially less than those with endarterectomy. They also found that, at 2 years, carotid stenting appeared nore only durable but also effective in stroke prevention with a zero percent 2 year ipsilateral major stroke rate. Thus there is enough evidence for equivalent clinical efficacy of CAS compared with CEA, achieved at a lower cost.

There are numerous clinical situations in which the endovascular approach of carotid stent placement could benefit patients by causing less risk, trauma and discomfort. The *ideal patients for endovascular stent placement* have classically been the following :

* Patients with an isolated, high ICA lesion near the angle of the mandible, which is inaccessible for surgery
* Patients with post-radiation CA stenosis
* Patients with post-endarterectomy recurrent CA stenosis
* Patients with contralateral CA occlusion and an incomplete circle of illis
* Patients with multiple medical problems
* Patients with tandem lesions
* Patients with non-atherosclerotic extracranial cerebrovascular diseases such as fibromuscular dysplasia, Takayasu arteritis, carotid dissection, etc.

Protocol for carotid stenting , Sir Ganga Ram Hospital

All patients qualifying for the procedure undergo a thorough neurological history and examination before the procedure. Baseline investigations include CBC, PT, aPTT, blood sugar, BUN, serum creatinine, ECG and chest X-ray. A baseline CT or MRI scan of the brain is obtained to document any pre-existing infarction and exclude other non-vascular pathologies that may resemble TIA. The patient is started on oral aspirin 325 mg/day and clopidogret 75 mg/day at least 3 days before the procedure. After the procedure, the patient is maintained on oral aspirin at the same dose lifelong and on clopidogrel at the same dose for 12 weeks.

The procedure is performed under local anaesthesia, and conscious sedation is used only if required. The right femoral approach is preferred and a 7F sheath is placed. A complete four vessel cerebral DSA is performed in a minimum of two planes to analyse the intracranial circulation and formation of the circle of Willis. A 7F guiding catheter is advanced into the common carotid artery (CCA) and the cather tip is positioned about 2.5 cm proximal to the stenosis. A baseline activated clotting time (ACT) is performed. A bolus of heparin (70-90 units/kg) is given to achieve an ACT 2-2.5 times the baseline and / or 250-300 seconds. Prophylactic atropine is not used; however, a syringe loaded with atropine is kept attacked to the i.v. line to be injected only if the patient develops bradycardia during balloon dilatation or stent placement.

The stenosis is crossed with a 0.014 inch guidewire under roadmap imaging. Predilatation is performed only if the stenosis is of a high grade. Cerebral protection devices are seldom used. A self-expanding PRECISE stent or carotid Wallstent is used. After deployment of the self-expanding stent, it is dilated with a 5 or 6 mm balloon for lesions of the ICA. Self-expanding stents have an element of rebound to external compression and can be sized to conform to both the ICA and CCA. Stents are usually oversized by 1-2 mm and if deployed within both the ICA and CCA, they are sized to the larger vessel. Many stents being developed are better suited for carotid use; for example, the PRECISE stent (Cordis) has a 5.5 F deployment system, if quite flexible and produces less friction during deployment, thus facilitating precision of placement. Heparin is not given after placement of the stent and the effect of the heparin already given is allowed to dissipate naturally. Protamine is not used to reverse the effect. At the end of the procedure, a complete angiographic evaluation is performed to ensure that there has not been any thromboembolism. The femoral sheath is removed when the ACT returns to the baseline level. The patient is kept under close observation for the day and is discharged the following day. Post procedure neurological evaluation is performed and NIH stroke scale is utilized at 24 hours, 1 month and 6 months. Fig.1 shows successful stenting of the origin of the ICA with a SMART stent (Cordis).

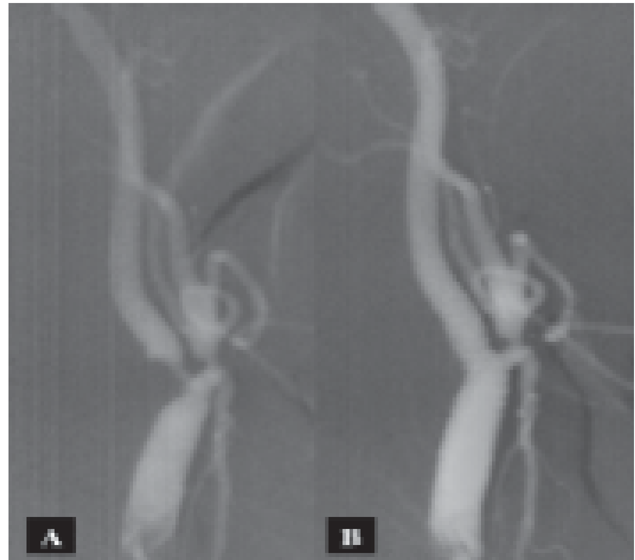

Fig. showing results of CAS in a 90% stenosed left cartotid artery before (A)and (B) Stenning

At Sir Ganga Ram Hospital, between May, 1999 and December, 2004, CAS was performed in 96 symptomatic NASCET-ineligible patients (e.g. those with multiple medical problems, atherosclerotic disease beyond the bifurcation, recurrent carotid stenosis, post-radiation stenosis, etc.)[24]. Twenty eight patients (29.1%) had complete occlusion of the contralateral ICA. The mean lesion length was 22+7.9 mm and mean percent stenosis was 85+8.0% (reference diameter 5.8+0.3mm). Self expanding SMART stents (Cordis), PRECISE monorail carotid stents (Cordis), carotid Wallstents (Boston Scientific Inc) and Sinus carotid stent (Optimed, Gmbh) were deployed (80% were deployed without predilatation). On an intention to treat basis, the overall technical success rate for carotid angioplasty was 99%. One patient had minor stroke 8 hours after the completion of the procedure, and an immediate CT scan of head performed suggested hyperperfusion injury. There were no major strokes. There was no mortality. Six patients (6.2%) had an uneventful transient focal seizure during balloon inflation, which reversed immediately following balloon deflation. All these patients had complete occlusion of the opposite ICA. Ten patients (10.4%) had transient bradycardia during balloon inflation, which did not require pacing.

Follow-up consted of serial duplex ultrasonography and clinical assessment in 76 patients at a median of 16 months. 2.6% of the patients (2 out of 76) had TIA involving the same arterial territory in the 16 month median follow up period[24].

CEREBRAL PROTECTION DEVICES

There has been concern that balloon angioplasty in the proximal ICA would send emboli intracranially to the brain. Although emboli do occur from carotid angioplasty, early case series suggested that emboli do not invariably cause clinical stroke and in fact, the occurrence of post procedure stroke was roughly comparable with the stroke rate seen after CE. However, there has been a recent surge in the development of cerebral protection devices that trap the embolic particles during the procedure[25]. Various devices have been used for thromboembolic protection during stenting[26-28]. Henry et al[26] report the results of CA stenting with cerebral protection using Percusurge, a guide wire device, in 48 high-risk patients with 53 ICA stenoses. Their 30-day periprocedural risk has been minor stroke in 1 patient (2.08%) and death in 1 (2.08%).

The drawback of occlusive devices is that they cause prolonged occlusion of the ICA till the procedure is complete. The average time calculated from the beginning of the set-up to complete removal was 46 minutes[27]. Patients with an incomplete circle of Willis or inadequate collateral flow may not tolerate prolonged occlusion. Moreover, prolonged occlusion carries a risk of vasospasm, circumscribed dilatation at the site where the protective balloon is inflated and perioperative CA occlusion[27,28].

Recently, Kastrup et al[29] systematically reviewed single-center CAS studies and concluded that protection devices appear to reduce thromboembolic complications during CAS. This review included a variety of single-center studies from 1990 to 2002. The combined stroke and death rate within 30 days was 1.8% in CAS with protection and 5.5% in CAS without protection.

In another study, Cremonesi et al[30] studied in hospital and 30 day adverse events in 442 patients treated percuntaneously for CA disease with embolic protection devices. The percutaneous procedure was successful in 440 patients (99.5%). There was no periprocedural death with any embolic protection device. The in-hospital stroke and death rate and 30 day ipsilateral stroke and death rate was 1.1%. The overall complication rate was 3.4%. Major adverse events included 1 major stroke (0.2%), 4 intracranial hemorrhages (0.9%), ICA wall fissuring (0.2%) and 1 diffuse cardio embolism (0.2%). Minor adverse events included 4 minor strokes (0.9%) and 4 TIAs (0.9%). There were 4 complications (0.9%) related to the use of cerebral protection devices : 1 case of abrupt closure of the ICA because of spiral dissection (0.2%), 1 case of trapped guide wire (0.2%) and 2 cases of intimal dissection (0.5%). Transient loss of consciousness, tremors and fasciculations were present in 6 of the 40 patients (15%) in whom occlusive protection devices were used.

Neither Cremonesi et al not Kastrup et al repored on the failure rate on application of protection systems, which can be upto 20% according to literature[31]. The observation of EVA-3S Trial commitee[32], favoring protected stenting has been rejected[33] as the higher incidence of strokes in the first 30 days in patients treated withough protection could not be related to the non-use of a protection device.

IS A CEREBRAL PROTECTION DEVICE USE FOR EMBOLIC CAPTURE NEEDED?

The appearance of debris during CAS and CE is a common event[34-36] the first glance, it seems reasonable to apply protection systems to catch the particles by means of occlusive balloon systems or filtration baskets if the ICA. The beneficial use of such devices seems to be supported by a growing number of publications, mostly from the field of cardiology, which report declining rates of neurological complication. Despite he lack of further controlled studies, the use of protection devices has even become obligatory in the CREST (United States)[16] and EVA3S (France) trial testing for the equivalence of CAS and CE[36]. Paradoxically, some neurologists and neuroradiologists continue to successfully perform CAS without protection devices and hesitate to apply these as they demand an increase in both catheter time and technical complexity.

In centres in which experience with unprotected CAS has been gathered, skepticism about the the assumed self-evident improvement in implementation of protection devices is based not only on the low neurological complication rate without them but also on the technical complications associated with their use[37] such as dissection of the ICA (0.7%) or trapped guide wire needing surgical intervention (0.2%)[30]. Haemodynamic intolerance in occlusive balloon systems (5-15% of patients)[31,35] and congested nets are other typical problems encountered with the use of protection devices. Such experiences have apparently been poorly reported until now[38]. Furthermore, predilatation, which is often necessary in protected stenting and the removal of the protection devices may result in embolization[38]. From a neurological perspective, concentrating on the 10 studies appearing since 2002[35,38-40], a strong recommendation for the use of protection devices can not be made.

Carotid artery senting without protection devices appears to be safe. Of late, in a series of 245 consecutive patients[41] (260 hemispheres) underwent carotid artery stenting without protection devices, the technical success rate was 98.8%. Neurological complications (inclusive of transient ischemic attacks) were observed in 5.4%. The rate of major complications (death, major stroke and myocardial infarction) was 1.6% among the symptomatic and 1.5% among the asymptomatic cases. The rate of minor strokes was 3.2% in the symptomatic and 1.5% in the asymptomatic group. Neurological and other complications (death, major stroke and myocardial infarction) occurred only in comparable frequency as with protection devices. Majority of the neurological complications (64.3%) occurred post procedurally which could not have been prevented with protection devices.

The recent dramatic technical progress (e.g. less traumatic, self expandable stent devices, more friction-resistant introducer catheters and better guide wire systems leading to marked improvement of CAS), the associated learning curve of active interventionalists and the improved periprocedural antiplatelet and anticoagulation regimens may have been important factors in preventing cerebral embolism in the recent protected CAS studies[33].

The use of cerebral protection devices remains a controversial issue. The real purpose of distal protection is to prevent embolization sufficient to cause stroke. Reports in the literature reveal that the use of protection devices does not completely prevent embolization. Moreover, the use of these devices may inherently add to the risk of the procedure. However, cerebral protection with the filter device is technically feasible in most cases. A much larger investigation will be required to determine whether stroke is less likely with distal protection than without it. Could stroke develop even if these devices successfully trap all macroscopic material? Hypothetically, yes. However, a shower of small pariculate matter could cause microvascular obstruction. Of the various devices available, is there one that is superior? These critical questions still need to be answered by a head to head comparison in the form of a randomized clinical trial. We believe that protection devices not only complicate the entire procedure of CAS but also impose a significant cost concern. We propose to undertake a double blind randomized trial to clarify this controversy.

GUIDELINES

In patients with severe symptomatic CA stenosis, the evidence in favour of intervention (CE/CAS) is strong. The merits and demerits of both CE and CAS should be discussed with the patients and their families and the final choice should be left to them. For patients in whom surgery carries a high risk, i.e. NASCET excluded patients, we consider CAS, especially if medical therapy has failed. Intervention is not indicated for patients with mild symptomatic CA senosis (<50%). Some patients with 50%-69% symptomatic stenosis will benefit from surgery or stenting, but the decision should be individualized on the basis of several factors, including the presence or absence of vascular risk factors for stroke and technical expertise. For patients with asymptomatic 60% CA stenosis, the decisions are difficult. Clinicians should consider intervention (CE/CAS) for patients without apparent contraindications at a centre where they know that the surgeon/interventionist performing the procedure has a perioperative risk of stroke or death rate of <3%. Preoperative cardiac or haemodynamic evaluation may help in risk stratification. After the risks and benefits are explained to the patient, the ultimate decision depends on whether the patient is willing to accept the early risk and discomfort of surgery in the hope of long term benefit. In all cases, risk-factor control should be emphasized. We often recommend angiotensin-converting enzyme inhibitors to patients with hypertension and prescribe statins even for patients with normal to borderline cholesterol levels. Antiplatelet therapy (we prefer a combination of ASA and slow-release dipyridamole) is appropriate for all patients who do not have a contraindication and changes in lifestyle (smoking cessation, weight control, exercise and avoidance of excessive alcohol consumption) should be routinely encouraged.

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IMSA News

IMSA CHAPTER ACTIVITIES - April to June 2006

Tamil Nadu Chapter

09-04-2006 : Dr. Arjun Rajagopalan, "***Finding, Filtering, Evaluating and Adopting Medical Evidence in Clinical Practice***"

14-05.2006 : Dr. Abraham Thomas, "***Face Transplantation***"

11.06.2006 : Dr. Noordeen, "***Elimination of Leprosy***"

Delhi Chapter

01.04.2006 : "***Clinical Meeting***" in collaboration with Delhi Rheumatology Association at AIIMS, New Delhi

17.06.2006 : "***Scientific Programme***" organised in collaboration with Delhi Rheumatology Association at Seminar room, Indian Spinal Injuries Centre, Vasant Kunj, New Delhi.

Rural CME

T.N. Chapter *Venue : Ranipet (Vellore)*

18.06.2006 : Dr. B. Sumathi, "***Recent Advances in Management of Diarrhoeal Disorders in Pediatrics***"

: Dr. M. Rajkumar, "***Diabetic Foot Syndrome***"

HONOR

Dr. Tarun Gupta, Addl. Secretary General, IMSA has been installed the Hony. Secretary of the Rotary Club of Delhi Chanakyapuri, R.I. Dist. 3010 for the year 2006 -2007.

ANNOUNCEMENT

World Congress on Clinical & Preventive Cardiology (WCCPC 2006)

Organised By

Cardiological Society of India, Delhi Branch (CSI Delhi Branch),

Asian Pacific Society of Cardiology (APSC),

Healthy Heart Education and Research Foundation (HHERF),

International Medical Sciences Academy (IMSA)

In association with

J.W. Global Hospital & Research Centre (J.GHRC),

Defence Research & Development Organisation, Govt. of India (DRDO)

Indian Academy of Echocardiography(IAE) &

World Academy of Spiritual Science (WASS)

On September 22 to 24, 2006

At The Academy for a Better World Gyan Sarovar, **Mount Abu, Rajasthan,** India

SPECIAL HIGHLIGHTS : Recent advances in clinical cardiology, Preventive cardiology, hypertension, congestive heart failure, dyslipidemia, coronary artery disease, medical angioplasty, coronary imaging, cardiodiabetes, metabolic syndrome, echocardiography, electrophysiology and cardiac arrhythmia, interventional cardiology, cardiovascular surgery and cardiac anaesthesia. ***REGISTRATION*** : ***"Registration is free"*** for all Registered Delegates on "First cum First Serve Basis" upto 800 only upto July 15, 2006.;fee of Rs. 2500 /- will be charged from 16 th July,2006 ***For Registration Contact:*** **Dr. H.K. Chopra,** Organising Chairman, IMSA World HQ, 2nd Floor National Medical Library, Ansari Nagar, New Delhi 110029 E-mail: drhkchopra@yahoo.com, drhchopra@gmail.com Fax: 011-26444242,M: +91-9811090204 Ph: 011-26444242, 25658826, 26656990 **Dr. Satish Kr. Gupta,** Organising Secretary, J.W. Global Hospital & Research Centre , Shantivan, 307510 (Abu Road, Rajasthan) E-mail: wccpc2006@bkindia.com , smbmhealheart@gmail.com, Fax : 02974 - 228116, M: +91-9829479888,+91-9414154041, Ph: 02974-228577, 228101...106, 228340

IMSACOM 2006 ANNOUNCEMENT

Organising Secretary IMSACOM 2006 at Lahore has desired that all delegates/ participants who have already sent their passport papers to Dr. Shaheena Asif , should apply for **Visa** to the Pakistan High Commission in India urgent. ***For Details see Page 74.***

Dr. Pinnamanani Narasimha Rao International Award

Recommendations for ***"Dr. Pinnamanani Narasimha Rao International Award"*** *are invited from Fellows and Members as also the Board of Trustees of IMSA. The recommendations should be sent alongwith biodata, colour passport size photograph and abstract of the paper, to IMSA Headquarter, New Delhi by* **31st August 2006**. *The Selected awardes will be required to deliver an oration at* **IMSACOM 2006** *at Lahore.*

Secretary General, IMSA