# IASO

## INDIAN ASSOCIATION OF SURGICAL ONCOLOGY

(A section of The Association of Surgeons of India)

# NEWS LETTER

22

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# Secretary's Report

**Prof. Ravi Kant**
Secretary, IASO
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I welcome all the members of Indian Association of Surgical oncology to the annual meet of Association of Surgeons of India - the parent body to Patna ASICON 2001.

A significant change this year has been the cutoff date of final program for ASICON is now 31.7.2001. So it is advised that your scientific contributions must reach in time.

The Panchkula meeting organised by Dr. R. K. Karwasra was a thumping success. The scientific program kept the audience spell bound with full house attendance even on the last day. The Esophageal Symposium by Dr. Kiran Kothari, the Billiary Symposium by Gen P. Subhas, the Colo-rectal Symposium by Dr. K. Gopinath the Breast Symposium by Dr. R. Badwe, the Ovarian Symposium by Dr. H. Tongaonkar, the video session, lecture by Dr. Y. Kaiwa from Japan on Intra-gastric Surgery of gastric Cancer by Laparoscopic surgery and lecture on Sentinel LN by Dr. James Rucinsky from New York were much appreciated by the audience. Equally, the effort by the team of volunteers led by Dr. R.K. Karwasra was evident every where. Every one thanked Dr. Kirn Kothari for selecting and backing the perfect venue.

Dr. Vivek Gupta's paper On "Phenotypic Lymphocytes in Breast Cancer" was acknowledged the best oral presentation but in overall assessment for Detroit program, Dr SVS Deo of AIIMS name stood first. More about the Detroit program -I donot know what actually is the status, may be Dr. Maudar will throw some more light on it.

Equally, I am foxed by the status on WFSOS. As usual, the quiz program was a superhit and as usual the winners were from GCRI, Ahmedabad.

However, we have lagged in the drive to have more members. And particularly important is a phenomenon where postgraduates from acknowledged centers of cancer care have not been attending the meetings - only the consultants are willing to come. We cannot have aninverse pyramid as it is structurally unsound.

The current advancements should appear natural to us. The concept of Virtual Biopsy, Virtual Colonoscopy, Auto-fluoresecence, Optical Coherence Tomography, High Frequency USG, and Photo-dynamic therapy must be familiar to us, as much a acknowledgement of the fact that that Surgeon is an important evidence based risk factor inn Colorectal and Breast cancer. The concept of total mesorectal Excision, and a minimal volume of turnover is the need of the day. Progress in Oral cancer appears to have stalled and hence prevention and early detection with treatment of early cancer with non-mutilating options like laser or PDT must be seriously considered.

In the last but not in the least, I thank my President Dr. K. Panda from Cuttuck, Our Vice-Presidents Dr. R.I. Dave from Ahmedabad and Dr. K. Gopinath from Bangalore, Editorial Secretary Dr. L. Sarangi, Varanasi and the entire executive specifically including all the past presidents for giving the me encouragement and advice, and taking our organisation to newer and higher elevation. Wishing you all a very happy and prosperous New Year.

**Dr. Ravi Kant**

# EDITORIAL

**Dr. L. Sarangi,**
Varanasi

## VIRTUAL ENDOSCOPY

Virtual reality imaging, a type of interactive three dimensional (3D) medical imaging, was only a vision a few years back. It is no longer regarded as a modern technological gimmick and is an evolving technology that will undoubtedly revolutionize medical education diagnostic imaging and minimally invasive therapy. Virtual reality is human - computer interface that allows the individual to interact visually with a computer generated environment[1]. The key to create endoscopic views of the organs like colon, bronchus lie in the manner in which the dataset is collected and the interaction between the operator and virtual environment. The rapid development of high technology digital imaging modalities such as spiral CT, rapid scan MRI allow three dimensional representation of a volume of tissue as an endoscopic view of the organ on a computer terminal. This type of virtual endoscopic imaging was first carried out in 1994[2] and has generated considerable interest as a non-invasive procedure with comparable results as that of fibre-optic endoscopy.

Virtual endoscopy is possible with spiral CT as well as rapid scan MRI. However most of the studies have been undertaken by spiral CT on colon. It involves cleansing the bowel of stool, inflating the colon with air or carbon dioxide, carrying out a spiral CT of the abdomen and generating a three dimensional endoscopic view of the colon for visual inspection on a computer screen[3]. The user would typically advance from rectum to caecum and then return in a retrograde manner from caecum to rectum. Any abnormality can be recorded as static image on hard copy.

The clinical potential of virtual endoscopy in the evaluation of any hollow organ is enormous. Perhaps one of the greatest potential use is in that of colon. Virtual imaging of bronchial tree is easier both practically and technically ,though there is some reservations of its applicability in stomach.

Colonoscopy— Vining et.al. [2] were the first to report the technical feasibility of colon imaging with helical CT and virtual reality. In a prospective study by Kay et.al.[3] 38 patients were subjected to spiral CT followed by fibreoptic colonoscopy, virtual colonoscopy (VC) correctly identified 10 out of 11 polyps greater than 10mm in diameter where as 5 out of 13 lesions of 5-9 mm diameter could be identified. The sensitivity and specificity of VC for lesions greater than or equal to 5mm were 66.7% and 75% respectively, and for lesions greater than 1cm were 90% and 82.1% respectively. The advantages of VC lies in its non invasive character. In addition to identifying synchronous lesions or polyps in proximal colon in a diagnosed case of colorectal cancer patient it can provide both endoscopic views of the colon and visualization of the wall and its external relationships to determine the local extent of invasion. Use of VC as a method of screening is highly debatable. VC in not suitable in identifying flat lesions[4] and may have false positive results in the presence of retained or adherent fecal matter in colon.

Although it is a technique of enormous potential , there are a number of important challenges to overcome before being made available clinically on a routine basis. The procedure needs to be made cost effective,the sensitivity & specificity should match to that of conventional colonoscopy before being accepted as a true alternative to fibreoptic colonoscopy.

Bronchoscopy— Virtual reality imaging of bronchial tree is easier. No preparation is necessary as the bronchial tree is air filled, straight , and non-collapsible. In a case controlled study on ten patients Vining et.al[2] .reported accurate demonstration of endobronchial lesions & obstruction by virtual bronchoscopy. However Chinn et.al[5]. could not visualize subsegmental bronchi by this method which could routinely be achieved by fibreoptic bronchoscopy. It has been shown by Vining that 3-mm collimation beam is critical to visualize smaller bronchi. It may be mentioned here that optimal scanning parameters have not yet been identified for each particular organ system in spiral CT-based virtual endoscopy.

Gastroscopy- Virtual gastroscopy though technically possible has not attracted much attention because of the simple reason that patient compliance is quite satisfactory for fibre-optic gastroscopy in comparison to colonoscopy. Moreover at present the method used is not capable of competing with the imaging quality provided by fibre-optic gastroscopy. The preliminary results show that virtual gastroscopy more or less can be used as an auxillary tool for the improved assessment of the extent and spatial relationships of endoluminal pathology(6).

The current technological breakthrough that is sweeping through the field of imaging has generated this exiting area of virtual endoscopy. Though VC is the leader in this field of research, the same can be extended to other hollow organ systems. Perhaps this will some day replace the upper gastrointestinal endoscopies, small-

bowel follow- through and virtual cholangioscopy & pancreatoscopy may supersede diagnostic ERCP. However virtual endoscopy is still in its infancy and clinical developments are expected before full potential is realized. But clearly virtual reality imaging is now virtually a reality (7).

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# UICC FELLOWSHIP SCHEME

**International Cancer Technology Transfer Fellowships (ICRETT) : applications accepted at any time.**

The application closing dates, in chronological order, are as follows:

**1 May - UICC Latin America COPES Fellowships (non-medical, organizational)**
**1 July - UICC Yamagiwa Yoshida Grants (research)**
**1 September - UICC Asia Pacific Cancer Society Training Grants (non-medical, organizational)**
**1 November - Trish Greene UICC Oncology Nursing Fellowships (for oncology nurses in developing and East European countries)**
**1 December - Astra Zeneca and Novartis UICC Translational Cancer Research Fellowships**
**1 December - ACS UICC International Fellowships for Beginning Investigators**

To assist in the search for a suitable host, the UICC International Directory of Cancer Organization may be helpful (http://www.uicc.org/publ/directory/) .

If any of these are of interest to you, please download the application material from website at http://fellows.uicc.org/


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# EARLY COLONIC CANCER

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## INTRODUCTION

Early colorectal cancers (ECC) can be defined as malignant lesions that do not invade beyond the submucosal layer of the intestine.

The above definition is important because as per WHO classification of colorectal tumours, carcinoma is to be diagnosed only when the muscularis mucosae has been penetrated and the submucosa is infiltrated[1]. Japanese pathologists do not accept this definition[2]. Carcinomas confined only to the mucosa are not termed malignant so as to avoid overtreatment by surgical excision. However it should be realised that in this era of endoscopic surgery, intra mucosal carcinomas, which do not metastasize, should be regarded as malignant but treated endoscopically.

Most ECCs are polypoid or ulcerating following the adenoma-carcinoma sequence but non polypoid lesions are increasingly being reported, which show distinct characteristics.

The Japanese classification of ECCs is as below:

**Type 1** Protruded type
- 1p : Pedunculated type.
- 1sp: Semi pedunculated type.
- 1s : Sessile type.

**Type 2** Superficial type.
- 11a: Superficial elevated type.
- 11b: Superficial flat type.
- 11c: Superficial depressed type.

## HISTOPATHOLOGY OF EARLY COLORECTAL CANCER

In the West most, ECCs present as a malignant adenoma whereas ECC with no adenomatous component is well described in the literature. Possibly due to different approaches to histopathologic interpretation, lesions reported as superficial carcinomas limited to the mucosa by the Japanese are diagnosed as flat adenomas in the west[3]. Flat or sessile neoplasms removed by endoscopic mucosal resection (EMR) showed submucosal invasion by carcinoma in only about 1.5% cases in one Japanese series, thus suggesting that submucosal spread is uncommon.

Tumours showing deep submucosal layer invasion are associated with a more unfavourable histologic grade, lymphovascular invasion, lymph node metastasis, sessile morphology, and absence of adenomatous component within the tumour and such tumours should be considered as high risk cases for lymph node metastasis and recurrence after limited therapy,

Smaller size, better histologic differentiation, less lymph node metastasis, polypoid morphology are associated with good outcomes.

## NATURAL HISTORY OF ECCs

ECCs grow slowly with a Doubling time (DT) of 31.2 month in cases where the cancer is limited to the mucosa. However as the tumour grows down into the submucosa, the growth speed accelerates and the Doubling time shortens to 25.8 month. It is further observed that the pathologic growth pattern, non polypoid growth (NPG) v/s polypoid (PG) did not affect the tumour growth speed.

## MOLECULAR BIOLOGY OF ECCs

The adenoma-carcinoma sequence theory has generally been accepted for polypoid early colorectal cancers. However there are increasing reports of non polypoid (superficial) ECCs which show distinct characteristics histologically, genetically and clinically. Most clinicopathologic studies have shown that most non polypoid (superficial) cancers have no adenomatous lesions in the surrounding area.

There are differences in the genetic alterations in the polypoid and non polypoid (superficial) ECCs. The K-ras mutation rate is lower in non polypoid ECCs than in polypoid ECCs, but there is no significant difference in the p53 mutation rate between the two groups.

At present there is still not enough evidence to conclude whether non polypoid (superficial) ECC is derived from denovo carcinogenesis or the conventional adenoma-carcinoma sequence. The K-ras gene seems to determine the macroscopic configurations : whether polypoid or non polypoid. Further analysis, especially concerning the APC gene mutation in ECCs is essential to elucidate the carcinogenesis of non polypoid ECCs . The APC gene is considered to be responsible for the adenoma formation.

The p53 mutations are considered to be important late events in colorectal carcinogenesis. They do not seem to contribute to the formation of various macroscopic types of ECCs, whether polypoid or non polypoid (superficial).

## RADIOLOGICAL DIAGNOSIS OF ECCs

The radiologic features include the lesion's contour, central depression, converging folds and basal indentation. Superficial (non polypoid) invasive carcinoma can be di-

agnosed radiologically if a lesion measures 10 mm. and reveals moderate to severe basal indentation. Polypoid invasive carcinoma can be definitively diagnosed with much more certainty than the superficial type. The size and the radiographic sign of the basal indentation are the most important indicators for the diagnosis of invasive carcinoma.

**MANAGEMENT OF POLYPOID ECC.**

Endoscopic management of polypoid early colorectal cancer is no longer controversial. Surgery is unnecessary if the endoscopist is satisfied that the excision is complete and histology is "favorable" i.e. a resectoon margin of 2 mm. and well or moderately differentiated carcinoma. When the histology is "unfavorable" characteristics i.e. the margin between invasion and excision is1mm. or less or if the carcinoma is graded as poorly differentiated or shows signet-ring cell or mucinous characteristics. Endoscopic approaches such as saline injection polypectomy, india ink tatooing and the use of argon beam applicator as applicable in some cases.

For the challenging sessile polyps, between the choices of endoscopic polypectomy and surgical resection, less radical measures are availavle. Laparoscopic resection, with or without lymph node clearance, is one. Steroptic transanal microsurgery (TEMS) through a closed system operating proctoscope which allows for partial/ full thickness local resection supplemented by suturing/ clipping under two handed clipping can be used for rectosigmoid lesions upto 25 mm from the anal verge.[6]

**SURGICAL MANAGEMENT :**

This is indicated in the presence of "unfavorable" features such as poorly differentiated carcinoma, lymphovascular invasion or incomplete excision. Recently a new histopathological classification has been developed, sm1 is invasion to the upper one third of the sub mucosa , sm2 is invasion to the middle one third and sm3 is invasion to the lower one third. Lesions of smd1 and sm2 have low risk of local recurrence and lymph node metastasis, local excision is adequate. The sm3 and sm2 flat and depresssed types have a high risk of local recurrence and lymph nodes metastasis, further treatment is indicated[7].

*Colon and upper Rectum Lesions :-*

Pedunculated lesions with invasivse carcinoma sm1 and sm2 require colonoscopic polypectomy if no "unfavorables" factors are present. Otherwise bowel resection is indicated. Patients with sm3 pedunculated and flat or depressed sm2 & sm3 lesions should undergo bowel resection, sm1 flat or depresssed lesions can be subjected to endoscopic treatment.

*Mid rectum lesions :-*

The T1 carcinoma of mid rectum (7 to 10 cms from anal verge) can be removed by colonoscopic excision, a transacral approach and transrectal endoscopic microsurgery[9]. A redical resection is a low anterior resecton.

*Low rectum lesions :-*

T1 lesion (upto 7 cms from anal verge) can be seen easily and palpated and lesions not larger than 3 cm in diameter can be treated with fill thickness excision. Further treatment with an abdomino perineal resection is indicated for lesions with adverse factors and sm3 lesions.

**LAPAROSCOPIC SURGERY FOR ECC :**

Since the first laparoscopic colorectal surgery performed in 1991, retrospective and prospective evidence suggest that laparoscopy is as safe as conventional colorectal surgery with distinct advantages of less pain, faster recovery of respiratory parameters, and better preservation of cell mediated immune function. Laparoscopic surgery for early colorectal cancer may have a role to play in well selected group of patients[11].

**NON POLYPOID ECC-DIAGNOSIS AND MANAGEMENT :**

Non polypoid early colorectal neoplasms are grossly of three types: slightly elevated (small flat adenomas), laterally spreading and depressed. They are not easy to detect on colonoscopy and can be missed. Slight change in colour, interruption of the capilary network pattern, spontaneously bleeding spots, shape change with insufflation and deflation of air, slight deformation of the colonic wall are some of the findings at colonoscopy suggestive of such lesions. Flat adenomas are not invasive until they are large, depressed lesions can invade the submucosa even when they are extremely small.

*Treatment:*

(i) Small depressed lesions are treated with Endoscopic mucosal resection (EMR) technique; surgical treatment is indicated when the submucosa is massively invaded.

(ii) Laterally spreading tumours are not invasive despite their large size and therefore EMR is a good method.

(iii) Small flat adenomas need not be treated urgently as almost none is invasive.

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## The Mammadi & Alireza Soudavar Traveling Fellowships

The Mammadi Soudavar Memorial Fellowship was established in The New York Community Trust in 1981 by Fereidoon and Shamisi Soudavar as a memorial to their son, Mammade. This was designed as an opportunity for qualified physicians from across the world to study in cancer centers and ring their knowledge back to their country. In 1985, the Alireza Soudavar Memorial Fellowship Fund was similarly established by the Soudavar family to honour their other son, and to promote cancer education and research.

The program supports qualified applicants from across the world to participate in a period of observation and education in internationally renowned cancer centers. The scholars are supported with travel costs, housing cost and a monthly living stipend. The duration of the observership ranges from one to three months based on the needs and goals of the applicant. Successful applicants will be physicians who have completed residency training and who are committed to the further understanding of cancer care. The applicant must be between the ages of 28 and 40 and be able to get a visa for the country of the institution participating in the program.

Those interested in applying should request and application by country of the institution participating in the program.

Those interested in applying should request and application by contacting

David P. Saques, M.D.
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1275 York Avenue
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jaquesd@mskcc.org

## IASO - Baroda Travelling Fellowship

Rs. 5000/- only will be provided to a young surgeon who is aspirant to and has arranged attachment / observership with a Surgical Oncologist / Centre in India for 4 to 5 weeks.

An application on a plain paper enclosed with the Curriculum Vitae, place of attachment, acceptance from the centre, short objectives of the reasons for attachment and forwarding letter from the 2 members of the Indian Association of Surgical Oncology (IASO) should be sent to the office of the Secretary, IASO. The applicant must be MS in Surgery and citizen of India.

*This newsletter of IASO is going to be a regular feature and will be published twice a year. It will contain relevant professional news, events and recent topics of common interest. Members are requested to make use of the newsletter for dissemination of any valuable information.*


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# ANNOUNCEMENT

**Detroit Medical Centre, Wayne State University, USA** has instituted a visiting fellowship for four weeks at their centre for a young member of IASO, The fellow has to arrange his own passage. He will be provided free accomodation and sustenance allowance.

Those members desirous to apply for 2002-2003 may do so by sending their bio-data, research papers and publications to secretary IASO. The candidate must be below 45 years and he is required to present a research paper during **NATCON'2001** at Panchkula, as a part of selection process.

Application must reach secretary, IASO by 30th June' 2002

# RETROPERITONEAL SARCOMAS - SURGICAL MANAGEMENT

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## Introduction:

Retroperitoneal Sarcomas are rare malignant tumours accounting for only 0.1-0.2% of all malignant lesion diagnosed and for approximately 10-20% of all soft tissue sarcomas[1,2] They comprise 35-45% of all retroperitoneal tumours and majority of these tumour are malignant.[3]

## Clinical Presentation:

Retroperitoneal tumours became clinically apparent after they are sufficiently large and advanced. They have a peak incidence in fifth and sixth decade of life.

Symptoms are typically late, insidious, vague and non-specific. In 60-90% cases presenting symptoms are - abdominal pain, abdominal mass and increasing abdominal girth. One third of patients will have some distal neurologic sign or symptom from the mass effect of stretching or compressing of the lumbar or pelvic nerve plexus. Urologic symptoms are uncommon, however, non-specific symptoms such as nausea, vomiting, abdominal fullness and back pain, caused by encroachment of the tumour on adjacent viscera are not infrequent. Rarely patients may present with paraneoplastic syndrome of hypoglycaemia due to production of insulin like substances by large poorly differentiated liposarcomas. Average symptom duration is 5-6 months (range 1 week to 4 years). Abdominal mass is the most common physical finding occuring in 95% cases.

## Pathology:

Retroperitoneal sarcomas are histologically a heterogeneous group of neoplasia and are derived from the primitive masenchym. The most common histologic types have been liposarcomas, leiomyosarcomas, fibrosarcomas and malignant fibrous histiocytoma. (Table-1).

**Table-1**

**Distribution of adult retroperitoncal sarcomas**

| Histology | % |
|---|---|
| Liposarcoma | 30-60 |
| Leiomyosarcoma | 15-30 |
| Fibrosarcoma | 5-15 |
| Malignant fibrous histiocytoma (MFH) | 5-15 |
| Neurosarcoma | 5-15 |
| Undifferentiated | 5-18 |
| Embryonal rhabdomyosarcoma | <10 |
| Pleomorphic rhabdomyosarcoma | <10 |
| Alveolar soft part sarcoma | <2 |
| Synovial sarcoma | <2 |

Histologic type alone does not appear to be an independent prognostic factor, however, the pathologic grade of the tumour is one of the strongest predictors of survival and is the basis of the AJCC UICC[4] staging system. Sarcomas are designated as G1 (well differentiated), G2 (moderately differentiated) and G3 (poorly differentiated) on the basis of their cellularity, cellular pleomorphism, and mitotic activity and on extent of necrosis. Certain tumours such as angiosarcoma and synovial cell sarcoma are highly malignant, regardless of their cellular differentiation, and these should be classified as grade 3 tumours. The binary grading system employed at the Memorial Sloan Kettering Cancer Centre is outlined in Table-2.

**Table-2**

**Histological grading criteria for soft tissue sarcoma**

| Low grade | High grade |
|---|---|
| Well differentiated | Poorly differentiated |
| Hypocellular | Hypercellular |
| Increased stroma | Minimal stroma |
| Hypovascular | Hypervascular |
| Minimal necrosis | Significant necrosis |
| <10 mitoses per 10 hpfs | >10 mitoses per 10 hpf |

hpf = high powered light microscopic field.

**Table-3**

**Prognostic factors for Retroperitoneal sarcomas**

| Good | Poor |
|---|---|
| Complete resection | Incomplete resection |
| Low grade | High grade |
| Age >50 years | Age <50 years |
| Low DNA ploidy | High DNA ploidy |
| Liposarcoma | Leiomyosarcoma |

## Investigation:

Computerized tomography (CT) has assumed a major role in the diagnosis of retroperitoneal sarcoma. Conventional imaging methods- x-rays and ultrasonography still often performed first, usually provide only indirect, incomplete or imprecise information. Contrast films may also reveal displacement or distortion of viscera. Pyelography may show extrinsic distortions of the urinary tract. Ultrasonography may demonstrate - tumour extent, infiltration into adjacent organs and pressure effect on kidney. Ultrasonography is also used to monitor the response of chemotherapy, radiotherapy and recurrence

Cross - sectional imaging studies such as CT or MRI are the most definitive means of evaluating a patient and are the imaging technique of choice in evaluating a retroperitoneal soft tissue sarcoma.

**CT Scan :**

Beyond the obvious anatomical relationship is defined by CT Scan, the CT is helpful in establishing the relative degree of homogeneity of the lesion, extent and possible organ of origin, infiltration/obstructive changes in retroperitoneal organs and the presence of focal areas of necrosis within the lesion. CT Scan is also useful for evaluation and early detection of recurrence. Plane of dissection can be anticipated with the help of CT Scan.

The introduction of helical CT scanning has provided the ability to perform a detailed survey examination of the abdomen and pelvis utilizing extremely short scan times[5]. The abdominal CT scan also is valuable in differentiating sarcomas from other retroperitoneal malignancies, such as lymphomas manifesting as multinodal involvement, or urogenital tumours that tend to be more homogenous.

**MRI :**

MRI has recently been employed in the evaluation of retroperitoneal masses and is now regarded as the examination of choice for soft tissue masses[6]. However it should be noted that in the current climate of cost containment, a MRI Scan subsequent to an initial CT Scan is almost never indicated for most retroperitoneal masses. Occasionally, one may wish to discern the relationship of a medial retroperitoneal lesion to critical medial neurovascular structures such as aorta, vena cava and spinal canal, and in such cases, a MRI subsequent to an initial CT scan may be helpul. MRI enhances the contrasts between tumour and muscle, tumour and adjacent blood vessel and provides a generally superior three - dimensional definition of fascial planes. -The T1 - weighted MR image frequently provides better definition of the relationship of the mass to adjacent solid organs, such as kidney and spleen. The T2 weighted image provides improved assessment of muscle invasion. These substantial advantages may allow for improved assessment of resectability with preoperative MR imaging.

**Angiography:**

Angiography is not routinely performed prior to surgical resection of most retroperitoneal soft tissue sarcomas. It is selectively used to delineate the vascular anatomy before resection. MR angiography may be performed for preferential arterial enhancement and has the potential to replace conventional angiography for the depiction of vascular anatomy[8]

All patients with retroperitoneal sarcomas should undergo a preoperative chest radiograph. A preoperative chest CT is unnecessary unless chest x-ray is abnormal, as it is rare to see metastasis from a retroperitoneal sarcoma to the lung without intra-abdominal or liver metastases. Bilateral functioning kidneys must be ascertained because it is often necessary to perform an enbloc resection of the kidney along with the tumour.

**Biopsy:**

In general, preoperative biopsy of a presumed primary retroperitoneal mass, by either FNAC or core techniques, is not indicated as the therapeutic plan is rarely altered by preoperative attempts to ascertain a histological diagnosis.

Biopsy should be reserved for those cases in which the tumour is unresectable or a diagnosis of suspected metastasis from another primary tumour, lymphoma or germ cell tumour is being considered. Biopsy should be obtained from the most solid portion of the tumour and care should be taken to avoid spillage.

**Management :**

Management of retroperitoneal sarcomas includes Primary and Adjuvant therapy.

**Primary Therapy :**

Pack and Tabah (1954) proposed the principles of management of retroperitoneal sarcomas nearly fifty years back[9]. Principles of management include complete excision (monoblock) with adequate (negative) margin of normal tissue and dissection outside the 'pseudocapsule'.

Complete resection is usually dependent on anatomical site, multifocality and adjacent organ involvement. An adequate surgical margin can be defined as a gross and microscopic negative margin. Retroperitoneal sarcomas tend to expand and infiltrate tissue planes producing a 'pseudocapsule' that is composed of normal host tissue interfaced with fimbriae of tumour. The goal of the surgeon is to extirpate the lesion with an adequate margin without violating this 'pseudocapsule'.

As reported in several large series, complete resection rates for primary retroperitoneal sarcomas range from 38% to 94% [3,10-14] with an overall average of 53%[3]. The rate of complete resection does not appear to be related to histological type and grade or tumour size. Resectibility decreases with each local recurrence[13]. Karakousis et al[14] have reported a complete resectability rate of 95% and attributed this to proper positioning, proper incisions for adequate exposure, flexibility of mind, a

continuous adjustment of surgical plan according to operative findings, and following the path of least resistance increases the safety of dissection and the resectability of retroperitoneal sarcomas[14].

Incisions useful in the resection of retroperitoneal sarcomas are (1) the thoracoabdominal incision for upper quadrant tumors; (2) midline incision for midline sarcomas of the upper or mid abdomen, these tumors often requiring the dissection of the superior mesenteric vessels; (3) midline incision with lateral extension into the flank in the form of T, for flank sarcomas; (4) lower midline incision with bilateral transverse extensions to the pubic tubercle from the lower end of the midline incision at the pubic symphysis for a caudal midline extension of the tumor, (5) the abdominoinguinal incision for sarcomas of the iliac fossa, external iliac vessels, wall of the lesser pelvis and those extending to the pubic bone; (6) that tor internal hemipelvectomy tor retroperitoneai sarcomas involving or arising in the iliac bone and/or acetabulum.

Nephrectomy in patients with retroperitoneal sarcomas is usually indicated for technical reasons, actual invasion of the kidney is uncommon. There should be no hesitation on part of the oncological surgeon to perform a multiorgan resection where the closest margin is encompassed by such resection. However, it should be apparent that it does not make biological sense to perform a complex enbloc resection to facilitate a wider margin encompassing technically salvageable organs when the closest margin is a non-resectable structure (eg. aorta or vena cava)

Resection of adjacent retroperitoneal or intraperitoneal organs is often necessary in 50-80% patients to ensure complete excision. The most frequently resected organs are kidney (32-46%), colon (25%), adrenals (18%), spleen (10%) or pancreas (15%) [10,15,16].

Deliberate partial resection of retroperitoneal sarcomas should be avoided as there is evidence demonstrating that survival after partial resections is no different from that following simple open biopsy[11] Thus until effective adjuvant therapy is available for gross residual disease, deliberate partial resection outside the confines of a clinical trial should be reserved for the relief of bowel obstruction or the palliation of other critical manifestation of advanced disease. Surgical resection is usually not indicated in presence of malignant ascites, distant metastasis, involvement of vital structures like aorta, superior mesenteric artery and vein and poor performance status.

The morbidity and mortality rates associated with sarcomas continue to improve. Most large series report an operative mortality of 2.7% and an operative morbidity rate of 6-18%. The most common major complications associated with these extensive resections include enterocutaneous fistula, intra-abdominal abscess and intra-abdominal haemorrhage.

**Result of Surgical therapy, pallerns of failure and prognostic factors :**

Reported survival rates have gradually improved over the years because of a more aggressive surgical approach, in which every effort is made to avoid leaving residual tumor and improved postoperative care. Pack and Tabah[9] reported only 2% 5 year disease free survival rates in 1954 where-as the more recent reviews report overall 5 year survival rates of 54-74%[3,1 1-14] for patients undergoing complete resection.

Survival following complete surgical resection is related to the histological grade of the tumour, with the 5 year actuarial survival of patients with low-grade lesions being more than double than that of those with high-grade histology.[11]

Local recurrence remains a significant problem. Approximately 50-83% of complete resected patients develop local recurrence, either alone or along with systemic relapse. The median time for recurrence is significantly more for low grade sarcomas (42 months) as compared to high grade sarcomas (15 month). Locoregional recurrence account for about 75% of all recurrent retroperitoneal sarcomas. Isolated retroperitoneal recurrence are best treated with re-operation, as complete resection is possible in 40-60% of cases. The median survival of completely reresected sarcomas is 48 months as compared to 15 months for unresectable recurrent sarcomas.[11] Recurrence decreases survival despite adequate re-resection but provides additional years of survival.

Postoperative follow-up strategies are based on patterns of recurrence. All patients be seen and examined every 2-3 months, with cross-sectional imaging studies MRI performed at 6 month intervals for at least 4 year.

For patients presenting without metastatic disease, complete surgical resection and histological grade were the primary determinants of survival in several multivariate analysis[13,15,17].

**Adjuvant Therapy :**

Several retrospective studies have suggested that adjuvant external bean radiation therapy (EBRT) may improve local control after gross complete resection. In a National Cancer Institute (NCI) study, the combination of surgical resection and post-operative high-dose EBRT was

considered to be standard therapy. But due to relatively high complication rate of high dose EBRT, only some patient with advanced disease may benefit.

Intra-operative radiation therapy (IORT) offers several potential advantages over conventional EBRT. IORT allows a single large radiation dose to be delivered directly to the tumour bed intraoperatively, while simultaneously protecting normal, relatively radiosensitive tissues by operative shielding or physical exclusion from the treatment field.

No clinical trial to date has demonstrated the benefit of adjuvant therapy (radiation or chemotherapy) in treating patients with retroperitoneal sarcomas who have undergone complete resection.

Dynamic 3-D conformal irradiation for unresectable retropentoneal sarcomas is also being investigated and has shown some promise in unresectable tumors. Further studies are needed with this technique.

**Adjuvant Chemotherapy :**

Few trials exist using adjuvant chemotherapy combined with complete resection. Doxorubicin - based regimens, historically shown to have significant activity in metastatic sarcomas, have had no impact on survival in the adjuvant or neoadjuvant setting on primary sarcomas arising in the retroperitoneum.

**Conclusion :**

Despite advances in our understanding of the biologic nature of retroperitoneal sarcomas and improvement in diagnosis, operative techniques, and preoperative care, these tumours continue to be associated with local recurrence and a poor prognosis. Because of their anatomical location and indolent nature, detection at an earlier stage is unlikely. Therefore, improved survival rates will only be achieved by obtaining better local control through a more aggressive surgical approach, making every effort to perform a complete resection with negative histological margins.

Close follow-up evaluation of patients undergoing surgical resection is important to allow identification of those patients who would benefit from re-resection. So far, the results of treating patients with adjuvant irradiation and/or chemotherapy after complete resection have been discouraging.

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# CELLULAR PHONES AND CANCER - An Overview

**Dr. Sanjay Panda**

Much has been said about cellular phones. It has now become a part and parcel of our fraternity. Even though there is no concrete proof, the disadvantages of cellular phones are much eugolised especially by the idle minds. Think the assurance a cellular phone gives when you party out, even after few major cases.

**Principle :**

Cellular phones operqate with radio frequencies (RF), a form of electromagnetic energy located on the electromagnetic spectrum between FM radio waves and the waves used in microwave ovens, radar and satellite stations. They do not emit ionisiog radiation which damages DNA.

Cellular phones technology works on a system of geographically separated zones called "cells". Each cell has its own "base station" that both receives and emits radio waves. When a call is placed from a cellular phone, a signal is sent from the cell phone antenna to that cell's base station antenna. The base station responds to the cellular phone signal by assigning the phone an available radio frequency channel. When the RF channel is assigned, modulated radio signals are simultaneously received and transmitted, allowing voice information to be carried between the cell phone and the base. The base station transfers the call to a switching centre, where the call can be transferred to a local teleohone carrier or another cell phone.[1]

Cellular phones are an important source of RF exposure for those who use them. The amount of RF to which a person is exposed depends on a number of factors. The number of "cells" in a geographical area depends upon the cellular phone traffic in that area. For example, large cities may have many cells per square mile, whereas a less populated, rural area may have a single cell stretching over several square miles. The farther away a cell phone antenna is from its station, the higher the power level needed to maintain the connection. Very small cells are therefore associated with much lower exposures.[2]

Each geographical cell has a different number of available channels. Cellular phones operate ideally with the least amount of interference from neighboring channels. To help achieve optimal operation, cellular phones automatically step down to the lowest power level available that still maintains a connection with the base station. On the other hand, any physical obstacle, such as buildings or trees, interfering with the connection between base station and cell phone forces the base station to increase the power sent to that phone. Therefore the amount of power sent from a base station to a particular cellular phone can vary, even within a single cell.[2]

Manufacturers are required to report the specific absorption rate (SAR) of their product to the regulating agencies. The SAR is the amount of RF energy absorbed from the phone into the local tissues. The upper limit of SAR allowed is 1.6 watts per kilogram of body weight. Exposure to RF also depends on the duration and frequency of cellular phone use, with more use implying more exposure. Finally, older cellular phones (analog models) involve higher exposure than newer, digital equipment.[3]

**EPIDEMIOLOGIC ANALYSIS :**

Widespread cellular phones use has been a new phenomenon, hence its long term effect on health cannot be scrutinised. Recently one large cohort study and three large case-control studies have compared cell phone use among brain cancer patients and individuals free of brain cancer.

Results of the cohort study, which linked data on al of the 520.095 cellular telephone users in Denmark between 1982 and 1995 to the Danish Cancer Registry are in agreement with the findings of the case-contro studies. Cellular phone use was not associated with an increased risk of developing brain tumours overall; nor was there an association with any brain tumour subtypes o with tumours in any anatomical location within the brain As in the case-control studies, there was no correlation between brain tumour risk and RF dose, as assessed by duration of cellular phone use, date since first subscription age at first subscription, or type of cellular phone used.[4]

**ANIMAL STUDIES :**

The incidence of lymphoma was increased following wholebody RF irradiation in a transgenic animal model. Apart from above blemish, RF irradiation has not been documented to cause any other kind of changes.

**PUBLIC HEALTH AND SAFETY AGENCIES :**

In October 1999, the Centre for Device and Radiological Health (CDRH) of the US FDA stated, " the available science does not allow us to conclude tha mobile phones are absolutely safe, or that they are unsafe However, the available scientific evidence does no demonstrate any adverse health effects associated wit the use of mobile phones.[6]

In the UK, the National Radiologic Protection Board Advisory Group on Non-ionising Radiation concluded in May 1999 "....there was no human evidence of a risk of cancer resulting from exposure to radiation that arise from mobile phones.[7]

**OTHER HEALTH PROBLEMS :**

The only documented adverse effect from cell phones is an increased risk of automobile crahses when the phones are used while driving.[8,9] To date, there have been no claims made that cell phones are responsible for any other health problems.[10]

**SAFETY ADVICE :**

'As times change, we change' (An old latin saying). Hence absolute safety cannot be achieved unless we have long-term follow-up with us. To derogate cell phones is equally dicey as it has not caused any tangible harm till date.

**Accodringly;** the following summary from the Food and Drug Administattion Centre for Devices and Radialogical Health Offers advice to people concerned about their risk: If there is a risk from these products - and at this point we do not know that there is - it is probably very small. But if people are concerned about avoiding even potential risks, there are simple steps they can take to do so. People who must conduct extended conversations in their cars every day could switch to a type of mobile phone that places more distance between their bodies and the source of the RF, since the exposure level drops off dramatically with distance. For example, they could switch to : a mobile phone in which the antenna is located outside the vehicle, a hand-held phone with a built-in antenna connected to a different antenna mounted on the outside of a car or built into a separate package, or a headset with a remote antenna to a mobile phone carried at the waist. Again the scientific data do not demonstrate that mobile phones are harmful. But if people are concerned about the radio frequency energy from these products, taking the simple precautions outlined above can reduce any possible risk.[6]

In addition, people who are concerned might choose digital rather than analog telephones, since the former use lower RF levels.[3]

**SUMMARY :**

Cellular telephones being a new entity , we do not yet have long-term follow-up on their possible biological effects. But the lack of ionising radiation and the low energy level emitted from cell phones and absorbed by human tissues make it unlikely that these devices cause cancer. Further more not a single well-designed epidemiologic study could find any consistent association between cell phone use and cancer.

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# BREAST CANCER - PREVENTABLE?

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The identification of risk factors by the end 2000 millenium has thrown light with respect to prevention of breast cancer. Hitherto the options available are close surveillance and prophylatic mastectomy and these have their own lacunae; hence chemotherapy is attempted basing on tamoxifen effect on breast cancer metastases and progress of disease. Jordon in 1976 first demonstrated in rats that Tamoxifen supressed the development of carcinogen induced that mammary tumors and the trialists collaborative group (1998) showed that Tamoxifen when used early breast cancer reduced the risk of development of cancer in the contralateral breast by about 50 percent. Basing on these facts, large groups conducted tamoxifen effect on risk factors in different countries independently and the conclusions were drawn and published.

Royal Marsden Hospital of England (1986), Italian tamoxifen prevention study group European Institute of Oncology (1992) and National surgical adjuvant breast and Bowel disease (NSABP) in 1992 and all published results in the year 1999.

Royal Marsden Hospital enrolled 2494 women with family history of breast cancer (20mgs/day tamoxifen for 8 years with a follow-up of 70 months). Both placebo and tamoxifen group stopped medication. Powels et al (1998) expressed inability to conclude tamoxifen effect on the risk factor and prevention of breast cancer. The incidence is equal in both placebo and tamoxifen groups.

**Italian Tamoxifen Prevention Group Study**

5804 women with prior hystrectomy were enrolled and the tamoxifen 20mg per day for five years and follow-up a period of 46 months. (Veronesi et al 1998). 41 cases (0.958 percent) developed breast cancer. The trial permitted women on Oestrogen Receptor Therapy (ERT) and 14 percent of participants were on ERT group 390 women on ERT in placebo group 8 cases had breast cancer and 1 case in 462 women on ERT in the tamoxifen group show significant reduction of breast cancer.

**SABP Breast Cancer Prevention Group**

Fisher et al (1998). The group enrolled 13,388 women and eligible women one or more risk factors (a) age 60 years or more (b) age 35-59 years (c) pathologic diagnosis of LICS (d) nalliparity or age first live birth (e) number of previous biopsies (f) pathological diagnosis of atypica hyperplasia (g) age at menarche. (20mg tamoxifen pe day for 5 years, median follow-up 69 months.

1. Risk of invasive breast cancer was reduced by 4 percent, with cumulative incidence 43.4 breas cancer cases per one thousand women in the placebo group and 22 cases per 1000 women in the tamoxfen group.
2. Risk of non-invasive cancer insitu breast cancer was reduced by 50 percent, with cumulative incidence of 15.9 breast cancer cases per 1000 women i placebo group and 7.7 per 1000 women in tamoxifen group.
3. Among ER positive tumors group 60 percent was reduced and no difference in the ER negative tumo group was observed.
4. A significant decreased risk in those with a typica hyperplasia 86% and LICS 56%.

The most important adverse distressing effect o tamoxifen group is Endometrial Proliferation or Endometria Carcinoma of Uterus and there is minimally increase from 0.91 per 1000 women per year in the placebo group and 2.30 per 1000 women per year in the tamoxifen group which is a significant incidence. Other adverse effects observed were pulmonary embolism, deep vein thrombosis stroke in women of 50 years age and development o cataract and these produced morbidity.

**Selective Oestrogen Receptor Modulators:**

The tamoxifen used widely in invasive, non-invasive breast cancer and also on risk factors and its utilituy is well established. But prolonged use produces endometria cancer of uterus which is distressing greatly though othe adverse effect produce minimal morbidity. A new selec tive Oestrogen receptor modular (SERM) was described The SERM II is raloxifene. The multiple outcomes o raloxifene evaluation (MORE) trial in Europe recruited 770 postmenopausal women aged 80 years are less (mean 66.5 years) who had osteoporosis and no family history o breast cancer or endometrial cancer. The participants were randomised and raloxifene groups(60 or 120 mg for 3 years and reviewed (Cummings S.R. et al 1999). After a median

follow-up of about 40 months, 1.04 percent developed breast cancer in placebo group and 0.25 percent in raloxifene group developed breast cancer. The breast caner risk 3.8 per 1000 women on the placebo group and 1.7 per 1000 women on raloxifene group. Raloxifene if used more than 18 months the reduction in risk increased to 77 percent. The risk of cancer in ER positicve tumor is decreased but no effect on ER negative group.

Another synthetic compound Vitamin A analogue N-4 hydroxy phenyl retinamide (4-HPR) (Fenretinide) tried (Moon R.C. et al, 1979). The preclinical experimental studies showed retinoids have anti tumor effect. Veronesi et al (1999) tried in 2972 women of 30-50 years of age of either stage I breast cancer or DICS Fenretinide (Retinamide) 200 mgs per day for 5 years and follow-up of median period of 97 months found statistically no significant differences in the occurrence of contralateral breast cancer or ipsilateral breast cancer but a possible benefit was observed in premenopausal women and ipsilateral breast cancer. Post menopausal women receiving fenretinide showed a minimal increase in breast cancer risk people. The exact role are fenretinide require further study.

Indole-3-carbinol showed chemopreventice effect in experimental models (Bradlow et al, 1995) Cruciform vegetables contain the compound which contains an enzyme P450AI acts on oetrogen, control the formation of oestrogen metobolites which acts against tumor formation.

To conclude the new generation of SERM compounds are required as a preventive agents which is a future study. The new compound must act alone or in combination for chemoprevention of breast cancer and till then we have to depend on tamoxifen alone.

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# IASO BYE-LAWS

*The bye-laws of the IASO as adopted at one of the general body meetings held in December 1997, Mumbai.*

These Bye-laws supersede all previous bye-laws of the IASO :

1. In these Bye-laws, unless there is anything repugnant in the subject or context.

(a) IASO means "Indian Association of Surgical Oncology". This will remain a section of the ASI.

(b) ASI means "Association of Surgeons of India".

(c) Memorandum and Rules and Regulations means "Memorandum of the Association and Rules and Regulations of the ASI" which came into force in 1985.

2. **Name :** The name of the Association is "Indian Association of Surgical Oncology"-A section of ASI.

3. **Address :** The office of IASO is the place from where the Secretary functions.

4. **Objects :** IASO is formed as per guidelines set in schedule II of memorandum of ASI and was approved as a section in 1977. The objects of IASO are same as stated in III of memorandum of ASI. Further to that, IASO will encourage and advance the study and practice of the science and art of surgical oncology and allied organisations concerned with cancer problems.

5. **Membership :**

(a) **Life Membership :** A life member should be a full member (Annual/Life) of the parent body-The Association of Surgeons of India.

All persons, being surgeons with sufficient interest in cancer surgery/practising Cancer Surgeons/ completed an acceptable training in cancer surgery/ pursuing research in cancer surgery or related subject, are eligible for becoming life member.

(b) **Associate Membership :** Those who are under training in cancer surgery or those who are interested in cancer surgery but belong to other specialities such as Radiology, Pathology, Biochemistry and who may not be the member of the ASI.

Subscription of membership will be as decided from time to time by the general body of the IASO. Generally all members will be inducted as members.

6. **Termination of Membership :**

(a) If a member of IASO ceases to be a member ASI he/she will cease to be a member of IASO.

(b) If a member fails to pay subscription by due date resigns he/she will cease to be a member of IAS

7. **Year :** The year of the IASO will be same of AS 1st January to 31st December.

8. **Management :**

(a) IASO will be managed by an Executive Commit consisting of following office bearers, members a ex-officio members :

i. President
ii. Vice President : 2
iii. Secretary
iv. Editor
v. Members : usually 8 members will constitute executive committee.

(b) All past Presidents will be invitees to Execut Committee meetings.

(c) Organising Secretary of NatCon IASO of the y will be a coopted member for Executive Commit of IASO for that year.

(d) Only those members and life members who ha put in minimum 5 years of membership are eligi for election to Executive Committee.

(e) Save and except President the tenure of all off bearers and members will be for two years.

(f) The President shall hold office for one year. Ser Vice President will be the President after expiry his term unless he/she has resigned, indisposed disqualified otherwise.

9. **Election :**

(a) Election of the vacant posts as notified by the Secretary of IASO will be conducted in the annual General Body Meeting of IASO to be held during the annual conference of ASI in December each year.

(b) Every eligible member shall be proposed and seconded by two full members of IASO in the meeting after the proposed member has consented for the election.

(c) If there is no contest, the President shall declare the member elected for the post. Otherwise the election shall be by show of hands or secret ballot as decided by the President.

(d) If a poll is demanded by at least 25% of members of IASO present in the meeting and President is satisfied that such demand has been carried out by majority of members present in the meeting the vote shall be taken by ballot.

10. **Power of Managing Committee :** Shall be same as that of the Governing Council of ASI.

11. **The function and responsibility of different office bearers** of IASO will be same as that of ASI. The secretary will maintain and present the audited accounts each year at the annual conference.

12. **Meeting and Conference :**

(a) IASO shall hold Annual General Body Meeting every year during the annual conference of ASI and transact the business stated in the Annual Conference of ASI and transct the business stated in bye-law 15(b). Other meeting be it of Scientific Social/Managing Committee/ General Body in nature may held as per the requirements of IASO.

(b) IASO shall endeavour to organise Mid term Conference at least once every year and appoint an organising secretary for the conference in its Annual General Body Meeting.

13. **Annual Report :** An annual report stating the activities of the year shall be prepared by the Secretary for Annual General Body Meeting, a copy of which to be sent to Headquarters of ASI.

14. **Accounts of the year :** Accounts of the year of IASO shall be prepared by Secretary and audited by an auditor appointed by General Body within six months of the closing of the year. This should be placed in the General Body Meeting and after adoption a copy to be sent to Headquarters of ASI.

15. **Annual General Body Meeting :**

(a) Annual General Body Meeting (AGM) shall be held once every year as stated in Bye-laws.

(b) The following business will be transcted in the AGM :

i. Annual report.
ii. Audited accounts of the previous year.
iii. Programme and budget of the next year.
iv. Recipients of various orations for the next year.
v. The venue of Mid term Conference and appointment of Organising Secretary.
vi. Election of the office bearers and members of the Managing Committee.
vii. Any other business with the permission of the President. Topics of the symposia and their convenors, theme of CME, workshops and programme outline should be discussed in the General Body.

16. **Journal :** IASO shall publish its own Newsletter/ Journal and shall elect Editor for the same. He will be the sectional editor of the Indian Journal Surgery.

17. **Income :** Income of the IASO shall be derived from :

(a) Admission fees and subscription from members, life members and associate members.

(b) Excess of income over expenditure in Mid term Conference.

(c) Donations.

18. **Investment :** IASO shall have account with nationalised or reputed bank to be operated by persons authorised by General Body Meeting . The surplus fund after meeting statutory annual expenditure shall be invested in fixed deposits of such banks and approved securities or in any other manner to be decided in the General Body Meeting.

19. **Utilisation of Funds :**

IASO shall have account with nationalised or reputed bank and shall invest funds not required for its regu lar day to day activities in fixed deposits of such banks or approved securities as had been decided by the General Body Meeting. The accounts will be oper

ated as per provisions of memorandum of ASI. The proceeds of income from various deposits and investments shall be strictly spent for specific purpose for which such fund/funds are created.

20. **Representation :** IASO shall be represented as per Memorandum of ASI.

21. **Amendment of Bye-laws :** Any of the Bye-laws of IASO may be altered or rescinded to a new Bye-laws may be made at General Body Meeting by majority vote. The amendment shall come into force after it is circulated to all members and provide objection to such amendment of IASO is n received from ASI and 50% of valid members IASO within three months from the date circulation. A copy of such amendment is to be se to Headquarter of ASI.

22. **Schedule :** IASO secretariat shall maintain schedule comprising the various oration fellowship, research grant or any other grant f scientific works with rules and regulations for thes awards and management.

## MSKCC ONE - YEAR INTERNATIONAL SURGICAL ONCOLOGOY FELLOWSHIP PROGRAM DESCRIPTION

The Department of Surgery offers a one-year international surgical oncology fellowship to fully trained surgeons from outside the United States who desire further experience in surgical oncology. The one-year fellowship is comprised of a rotation schedule providing exposure to the Breast Service, Colorectal Service, laryngeal Service, Head & Neck Service, Thoracic Service, and 1 - 2 months of electives in the area of Chemotherapy and/ or Pathology and/or Radiation Therapy. The international fellow will be responsible for preoperative and postoperative patient care, and will perform operations under the direction of an attending surgeon. The international fellow will also gain experience in the surgical clinics, and arrange rounds, conferences, lectures, and seminars.

**ELIGIBILITY**

Candidates must be graduates of recognized foreign medical school and have completed a general surgery residency training program in their country. The candidate must have a clear interest and commitment in expanding his or her expertise in surgical oncology.

The candidate must have passed the USMLE examination and hold a valid ECFMG certificate. Selected candidates must qualify for and obtain a limited permit to practice medicine from the New York State Education Department, in accordance with its requirements.

In addition, the candidate must certify his or her intent to return to home country upon completion of training.

Number of Positions: 1

**HOW TO APPLY**

Completed applications should be returned by August 31st of the year prior to July 1 start date. Three letters of recommendation will be required; one from the medical school and two other letters form faculty members of the training program. Interviews for selected candidates will be held in October of the preceding year. The selection will be made by November 1.

Additional information about the international fellowship program should be directed to:

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1275 York Avenue
New York, NY 10021
21-639-7537
212-717-3169 (fax)

# ASICON 2001

## 26th December 2001 to 30th December 2001

### 28 / 12 / 01

| | | | |
|---|---|---|---|
| | **VENUE** | : | **J, IASO HALL** |
| | **02 - 00 PM - 03 - 45 PM** | : | **HEAD AND NECK CANCER** |
| | Conveners / ChairPersons / Moderators : | | Dr. S Pradhan and Dr. PATHAK K. A. |
| J / SYM / 28 / SYM07 / 1 | 02-00 PM - 02-15 PM | : | ROLE OF CONSERVATIVE SURGERY IN HEAD AND NECK CANCERS<br>- Dr. PRADHAN SULTAN, MUMBAI |
| J / SYM / 28 / SYM07 / 2 | 02-15 PM - 02-30 PM | : | RECENT TRENDS IN MANAGEMENT OF SALIVARY GLAND TUMORS<br>- Dr. CHATURVEDI ARUN, LUCKNOW |
| J / SYM / 28 / SYM07 / 3 | 02-30 PM - 02-45 PM | : | RECENT ADVANCES IN MANAGEMENT OF THYROID MALIGNANCIES<br>- Dr. CHANDRA SOMESH, AHMEDABAD |
| J / SYM / 28 / SYM07 / 4 | 02-45 PM - 03-00 PM | : | MANAGEMENT OF METASTATIC NECK NODES:WHERE DO WE STAND<br>- Dr. PATHAK K. A., MUMBAI |
| J / SYM / 28 / SYM07 / 5 | 03-00 PM - 03-15 PM | : | ROLE OF CHEMO-RADIATION IN HEAD AND NECK CANCER<br>- Dr. TBA |
| | **03-45 PM - 04-30 PM** | : | **SMT. RADHA DEVI ORATION** |
| | Conveners / ChairPersons / Moderators : | | Dr. Panda K<br>Dr. KANT RAVI |
| J / ORA / 28 / ORA14 / 1 | 04-00 PM - 05-00 PM | : | Stomach Cancer<br>**- Dr. Maudar K K** |

**4.30 pm to 5.30 pm on 28 December 2001 = Onco Quiz, Convenor Dr Somesh Chandra;**

| | | | |
|---|---|---|---|
| | **05-30 PM - 06-30 PM** | : | **BUSINESS MEETING** |
| | Conveners/ChairPersons/Moderators | : | Dr. PANDA K. |
| J / BM / 28 / BM02 / 1 | 05-00 PM - 06-00 PM | : | EXECUTIVE COMMITTEE MEETING OF IASO |

### 29 / 12 / 01

| | | | |
|---|---|---|---|
| | **VENUE** | : | **J, IASO HALL** |
| | **09-00 AM - 10-55 AM** | : | **INVITED LECTURE** |
| | Conveners / ChairPersons / Moderators : | | Dr. KHAREY SP<br>Dr. SANDEEP KUMAR |
| J / GL / 29 / GL08 / 1 | 09-00 AM - 09-20 AM | : | WELL DIFFERENTIATED THYROID CARCINOMA'S<br>- Dr. PARIKH DM, MUMBAI |
| J / GL / 29 / GL08 / 2 | 09-25 AM - 09-45 AM | : | VIRTUAL ENDOSCOPY<br>- Dr. KANT RAVI, NEW DELHI |
| J / GL / 29 / GL08 / 3 | 10-15 AM - 10-25 AM | : | What is new in cancer of esophagus<br>- Dr. Sanjay Sharma, Mumbai |
| J / GL / 29 / GL08 / 4 | 10-30 AM - 10-55 AM | : | CURRENT PERSPECTIVE IN CANCER<br>- Dr. Sanghvi VB, Mumbai |
| | **11-00 AM - 01-00 PM** | : | **GALL BLADDER CANCER** |
| | Conveners / ChairPersons / Moderators : | | Dr Misra, NC, Lucknow and Dr.Shukla, VK, Varanasi |
| J / SYM / 29 / SYM08 / 1 | 11-00 AM - 11-20 AM | : | GEOGRAPHICAL DISTRIBUTION AND ETIOLOGY<br>- Dr. SHUKLA V K, Varanasi |

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|---|---|---|---|
| J / SYM/29 / SYM08 / 2 | 11-20 AM - 11-40 AM | : | CLINICAL FEATURE, DIAGNOSIS AND STAGING OF CARCINOMA OF THE GALLBLADDER<br>- Dr. SARANGI L., Varanasi |
| J / SYM / 29 / SYM08 / 3 | 11-40 AM - 12-00 PM | : | PALLIATIVE SURGERY IN CARCINOMA OF THE GALL BLADDER<br>- Dr. KHANDELWAL C, Patna |
| J / SYM / 29 / SYM08 / 4 | 12-00 PM - 12-20 PM | : | MULTI MODAL APPROACH TO ADVANCED CARCINOMA OF THE GALLBLADDER<br>- Dr. JAGANNATH P., Mumbai |
| J / SYM / 29 / SYM08 / 5 | 12-20 PM - 12-40 PM | : | RADICAL SURGERY IN CARCINOMA OF THE GALLBLADDER<br>- Dr. CHAUDHURY A, New Delhi |
| | **02-00 PM - 04-00 PM** | : | **SYMPOSIUM ON ORAL CANCER** |
| | Conveners / ChairPersons / Moderators : | | Dr VIJAY KUMAR, BANGALORE |
| J / SYM / 29 / SYM 13 / 1 | 02-00 PM-02-20 PM | : | MOLECULAR BIOLOGY-WHERE ARE WE GOING?<br>- Dr. KIRAN KOTHARI, AHMEDABAD |
| J / SYM / 29 / SYM 13/2 | 02-20 PM- 02-40 PM | : | RADIOLOGICAL IMAGING-WHAT IS OPTIMAL ?<br>Dr. LALA MURAD E., MUMBAI |
| J / SYM / 29 / SYM 13 / 3 | 02-40 PM - 03-00 PM | : | SCREENING-HOW MUCH IS EFECTIVE ?<br>Dr. MISRA SANJEEV, LUCKNOW |
| J / SYM / 29 / SYM 13/4 | 03-00 PM - 03-20 PM | : | TOBACCO BAN-WHERE DO WE STAND ?<br>- Dr. RAO SUBRAMANYESHWAR. HYDERABAD |
| J / SYM / 29 / SYM 13 / 5 | 03-20 PM - 03-40 PM | : | MANAGEMENT OF NECK-WHAT IS IDEAL ?<br>Dr. MAZUMDAR, KOLKATA |
| J / SYM / 29 / SYM 13 / 6 | 03-40 PM - 04-00 PM | : | MICROVASCULAR FREE FLAP - IS IT VIABLE OPTION IN INDIA ?<br>Dr. GOPINATH K S, BANGALORE |
| | **04-00 PM - 05-00 PM** | : | **BUSINESS MEETING GBM of IASO** |
| | Conveners / ChairPersons / Moderators : | | Dr. PANDA K. |
| J / BM / 29 / BM03 / 1 | 04-00 PM - 05-00 PM | : | GENERAL BODY MEETING OF IASO |

## 30 / 12 / 01

| | | | |
|---|---|---|---|
| | **VENUE** | : | **J, IASO HALL** |
| | **09-00 AM - 10-30 AM** | : | **FREE PAPER SESSION** |
| | Conveners / ChairPersons / Moderators : | | Dr. PANDA K. CUTTUCK<br>- Dr. KARWASRA, R K, Rohtak |
| J/FP / 30 / FP05 / 1 | 09-00 AM - 09-10 AM | : | OUR EXPERIENCE WITH ADRENOCORTICAL TUMOURS<br>- Dr. VERMA RAVINDER KUMAR, Dr.VIVEK AGARWAL |
| J / FP / 30 / FP05 / 2 | 09-10 AM - 09-20 AM | : | STUDY OF COMPARISION OF VARIOUS PROCEDURES IN MALIGNANT OBSTRUCTIVE JAUNDICE<br>- Dr. DAVE R.I., Dr. R. A. TANKSHALI,<br>- Dr. H. K. SHUKLA, Dr. DARSHAN BHANSHALI,<br>- Dr. SHAILESH PATEL, Ahmedabad |
| J / FP / 30 / FP05 / 3 | 09-20 AM - 09-30 AM | : | SEROLOGICAL ASSAY OF CA 242,CA 125,CA 15-3 AND CA-19-9 IN MALIGNANT DISORDER OF GASTROINTESTINALTRACT.<br>- Dr. GURUBACHAN, Dr. V. S. SHUKLA |
| J / FP / 30 / FP05 / 4 | 09-30 AM - 09-40 AM | : | MORBIDITY AFTER MASTECTOMY<br>- Dr. VENKAT G, Dr. R. KHANNA,<br>Dr. A. K. KHANNA, Varanasi |
| J / FP / 30 / FP05 / 5 | 09-40 AM - 09-50 AM | : | CANCER OF GINGIVO-BUCCAL COMPLEX : LESSONS FROM PATTERNS OF SPREAD<br>- Dr. PATHAK K. A., Dr. V. KHANNA, Dr. V. KABRA,<br>- Dr. V. D. SANGHVI, Mumbai |

| | | | |
|---|---|---|---|
| J / FP / 30 / FP05 / 6 | 09-50 AM - 10-00 AM | : | TUMOR NECROSIS FACTOR SERUM LEVELS IN ORAL CANCER PATIENTS AND THEIR RELATIONSHIPS WITHNUTRITIONAL PARAMETERS<br>- Dr. KHANNA RAHUL, Dr. H. D. KHANNA, Dr. S. KHANNA, |
| | | | - Dr. A. K. KHANNA, Dr. H. S. SHUKLA, Varanasi |
| J / FP / 30 / FP05 / 7 | 10-00 AM - 10-10 AM | : | AGNOR IN PANCREATIC ADENOCARCINOMA<br>- Dr. YADAV SANJEEV KUMAR, Dr. G. NATH, Dr. KUMAR M, Dr. DIXIT V. K., Dr. KHANNA A. K., Varanasi |
| J / FP / 30 / FP05 / 8 | 10-10 AM - 10-20 AM | : | LEIMYOSARCOMA OF THE SEMINAL VESICLE<br>- Dr. AGRAWAL VIVEK, Dr. SUNIL KUMAR, Dr. ARUN GUPTA, Dr. D. SHARMA |
| | 10-30 AM - 12-30 PM | : | VIDEO SESSION |
| | Conveners / ChairPersons / Moderators : | | Dr. KOTHARI KIRAN, Ahmedabad and Dr Sanjay Sharma, Mumbai |

## 29th September
## GBM Agenda
## Time 4 pm at Patna ASICON

- Minutes of the last meeting.
- Accounts presentation
- Report on Panchkula by Dr. R K Karwasra
- Plan for the next meeting in Ooty.
- Plan for the next meeting-letter of Dr DD Patel.
- WFSOS-what is next
- European J of Surg Oncology-publication in India.
- Detroit fellowship-any news
- Election-should we have in Natcon?
- Election of the Vice president and the executive members.
- Any other matter with the pemission of the chair
- Installation of the the New Executive

**"This issue of the News Letter has been brought out by Dr.Sanjeev Misra from Lucknow. I must thank him for his sincere efforts to shoulder the responsibility to publish this issue on a short notice and arrange the necessary finance as well."**

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# INDIAN ASSOCIATION OF SURGICAL ONCOLOGY

(A Section of ASI)

## MEMBERSHIP APPLICATION FORM


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Affix Passport Size Photograph here

Dear Sir,

I wish to be a member of Indian Association of Surgical Oncology as life member and I am enclosing a draft/cheque/money order of Rs. 1000.00 (Rs. 40/- to be included if a cheque is drawn) towards subscription and enrolment on being elected.

Name in Full (in Block) :

Date of Birth :

Qualifications :

ASI Regd. No. & State where registered :

Address for correspondence (in Block Letters) :

Position engaged in Teaching/Research/Practice etc. :

Practice of Oncology or related speciality (%age)

Sub-speciality if any :

Sponsored by (1) _____
Name of Member ASI ASI Number Signature

Sponsored by (2) _____
Name of Member ASI ASI Number Signature

Date of Signature

* Cheque/Draft for Rs. 1000/- Life membership should be drawn in favour of "Indian Association of Surgical Oncology", payable at New Delhi.

## Addition/Correction form for the IASO Members Directory

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