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IASO

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INDIAN ASSOCIATION OF SURGICAL ONCOLOGY
(A section of Association of Surgeons of India)

NEWSLETTER

Past Presidents : D J Jussawala (1977), PB Desai (1979), MP Vaidya (1981), Ashok Mehta (1983), DD Patel (1984), AP Majumdar (1985), RS Rao (1986), NC Mishra (1987), NN Khanna (1988), SK Sarkar (1990), PM Trivedi (1991), KK Pandey (1992), SK Shukla (1993), JB Venkatrau (1994), Sambhu Pal (1995), CK Gupta (1996), HS Shukla (1997), SP Kharey (1998), P Subhas (1999)

Dec. '99 Vol. 13 No. 2

Hony. Secretary, IASO, Dr. Kiran C. Kothari's Report
during inauguration of
NATCOM, IASO'99
at HIMS, Jolly GRANT, Dehradun.

Respected,

Dear Colleagues,

It gives me great pleasure at HIMS amongst you after assuming the post of Honorary Secretary since last IASO sectional conference at ASICON at Ahmedabad. Since then we have worked very hard in finalising the NatCon IASO'99, this conference. All the members of the organizing committee in general, Dr. Sukamal Saha, the Organizing Chairman, Dr. Arun Gupta, the Co-organizing Chairman and Dr. Sunil Saini, the Organizing Secretary in particular have worked very hard for the success of this conference. One thing I would like to state that Dr. Saini was always in touch for finalising the program and other details of this conference.

This year we have enrolled 24 new members making the total tally to 328 members. The excellent scientific program in this conference speak further active participation of our members. We are offering ISAO Baroda travelling fellowship to young aspirant members. Those who are desirous may apply to IASO office with their Bio-data.

Prof. Ravi Kant, our editorial secretary, is working excellently for bringing our IASO news letter. The last issue of Sep.'99 has been published and I am sure you all must have received it.

We are sponsoring a CME programme at Lucknow in November this year. Dr. Sanjeev Misra is organizing "National seminar on progress in Oncology-directions from next millenium. Excellent scientific feast is being offerred in this meeting to be held from Nov. 12-13'99.

After this we have a sectional meeting on Dec. 28, 29 & 30th at Madurai during ASICON meeting. Symposium on parotid tumors, panel discussion on gastric cancer, metastasis of unknown origin is being scheduled during this meeting. An interesting session on "How I do it ?" is also being organized during this meeting. I am sure you all will be coming to Madurai.

Our next National Conference "NatCon IASO'2000" will be held at Puri. The organizing Secretary is Dr. K. Panda from Cuttack.

Coming back to this institute, I am deeply impressed by tremendous progress in six years and the organizational capabilities of this institute. I wish all the members of this institute very best of luck for the future.

As it has been done by all the organizing secretaries of previous NatCon's, I am sure Dr. Sunil Saini also will contribute generously towards IASO secretariat.

With these words, I would like to conclude my talk.

MESSAGE FROM THE PRESIDENT, IASO

As I look back at the just concluding year and on to the new millennium, I notice with satisfaction at the year that has gone by, with the wonderful and satisfying NATCON that we have had at Jollygrant and the strides that our chapter has made and still going strong! We are ready to conclude the year at Madurai where our chapter will make a very noteworthy contribution. I am thankful to all our members who gave me full co-operation and encouragement for all the activities - and to all my predecessors who guided me throughout the difficult times. I am sure our chapter will march on regardless gaining strength to strength at the new era. I am sure all of you will make earnest efforts to be at Madurai and participate at all our chapter activities. Your presence will give enough encouragement to our young surgeons who will present their work.

Wishing you all the very best of the new millennium year for you and your family.

Brig. P. Subhas,
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EDITORIAL

CONTROVERSIES IN MANAGEMENT OF BREAST DISORDERS

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TERMINOLOGY OF BENIGN BREAST LUMPS:

The benign breast disease is under a banner of ANDI-Abnormalities of Normal development and involution. Thus, the framework provided by the Hughes and colleagues is an essential factor in understanding the syndrome complex of SOLID benign breast diseases.[1]

INVESTIGATIONS:

The triage of investigations mandated is USG (Ultrasonography), Mammography, and FNA (Fine Needle Cytologty). Mammography is not advised below 30 years.[2]

On ultrasound, the cystic diseases are Bloodgood's disease (Single blue domed cyst) or Schimmelbusch's disease (Multiple Cystic disease).[3]

The sensitivity of breast FNA on palpable mass is 80 to 90 % (mean 90%). The specificity and predictive value of breast FNA is close to 100% as false positive results are exceptionally rare. The efficacy of the test ranges from 84% to 99.5%.[4-8]

PALPABLE BREAST LUMPS: RESULTS OF TRAIGE OF INVESTIGATIONS[9]

	CLINICAL	US	MAMMOGRAM	FNA
SENSITIVITY	88	85	88	95
SPECIFICITY	91	88	90	95
POSITIVE PREDICTIVE VALUE	95	92	94	99.8

In clinically palpable breast lumps, comments on axillary lymph nodes are 30% erroneous as being either false positive or false negative.[10]

IMPALABLE BREAST TUMOUR:

In impalapable mammographically detected breast lessions, Needle core biopsy is more accurate than stereotactically guided/ directed FNA. Large needle biopsy has a false negative rate of upto 20%.[11-13]

Needle localisation in bi-planer view, (needle guided into the lession) followed by biopsy may be ideal in such a situation. 11% of patient undergoing screening will require such a procedure.[11-13]

SURGERY IN BBD

More often than not nurgery is not recommended in BBD (including Fibroadenoma). Fibroadenoma tend to regress with time. More than 25% resolve within two years.[14-15] Cant et al demonstrated a probability of 0.46 for resolution after 5 years.[15] Sansbury reported 32% resolution at 2 years.[16] In Oxford, over 90% patients are treated conservatively as compared top South Africa where only 21% patients are treated conservatively. This depends upon counselling, facilities and aptitude of surgeon.[17-18]

SURGERY IN BREAST LUMP SHOULD BE DEFERRED IF:

1. Size is less than 3 cm.
2. FNA is benign on two separate events.

3. No abnormal mammographic (Mx) pattern.
4. Absence of localised soft tissue density on MX.
5. Absence of localised soft tissue density which changes on successive Mx.
6. Absence of localised soft tissue density with ill defined borders on Mx.
7. Absence of localised focus of microcalcification with stellate distortion of stroma on Mx.

NEWER SURGICAL MODALITIES FOR BBD:

Interstitial laser Hyperthermia as described by Bina Ravi, Som and Ravi Kant [21] looks like an interesting alternative, as it avoids scar.

INCIDENCE OF MALIGNANCY IN BBD:[22,23,28]

Non proliferative benign breast diseases (e.g. Adenosis, Cysts, Duct ectasis, Fibroadenoma, Mastitis, Fibrosis, Mild hyoperplasis, Mataplasia- apocrine or squamous) have no increased risk of developing malignancy.

Proliferative diseases like moderate hyperplasia, papiloma with fibroadenosis core has 1.9 relative risk of developing malignancy. Atypical hyperplasia has a 4.4 relative risk of developing malignancy.

Proliferative disorders, e.g., atypical hyperplasia associatged with family history of breast cancer has a 11.0 relative risk of developing malignancy.

Cystic disease of breast has 2.5 to 7.5 times risk of developing cancer. Incidence is nearly 8 times higher if Epitheliosis is present. The risk appears to be greater in younger women. Under the age 45, the risk is 6.8 as compared to 3.3 in women aged 40-49 years. 3.34 in 50-54 age group and 1.99 in women over 54 years of age. The risk is higher in first year after aspiration (8.07) but remains higher after even 5 years (3.08). The risk is irrespective of type and number of the cyst.[28]

SURGERY IN BBD:

Appropriate incision under direct vision or via a laparoscope[9] from an areolar incision should be used and subdermal thinning should be avoided.

LACTATIONAL ABSCESS:[24-28]

Breast abscess - lactational as well as non lactational abscess do not need general anaesthesia and disfiguring scars any more. If skin overlying breast is normal then repeated USG guided aspirations coupled with broad spectrum antiobiotic will produce satisfactory result. If overlying breast is thinned or dead then a very small incision under cover of topical anaesthesia cream or spray will give a good result. The days of disfiguring surgery (under general anaesthesia) are over. Patients whould be encouraged to continue breast feeding as this reduces engorgement and pain. Unit or individuals unable or unwilling to carry out such a treatment should hand over these patients to those who are willing to provide this improved service. The age of open surgery for this condition should have vanished.

TREATMENT OF MASTALGIA:

Patients need counselling regarding the concept of ANDI, proof of it being benign based on USG, FNA (Mammography being excluded from age less than 30 years).

The need of correct bra size is explained to the patients. It is interesting that only 19% of patients wear correct size of bra.[9,29] The patients are advised to wear the bra at night as well.

MEDICATIONS FOR MASTALGIA:

A decision is to be made as to whether the

pain is cyclical, non-cyclical or even non-breast (referral from chest wall, muscles, neck shoulder, or Tietze's syndrome can all present as breast pain). Evening Primrose Oil = Gamma Linoleic Acid (response rate 30-70%); it is more useful in women over 40 years of age and has fewer side effects.

Danazol (GLA)- Gonadotrophin release inhibitor has higher response rate but side effects are also higher. Bromocriptine - a long acting dopamine agonist has response reate similar to GLA but with higher side effects. GESTRINOME[30] has similar response rate to GLA with fewer side effects. Tamoxifen is also effective but side effects mount to 60% at 6 months. There is no place for use of diuretics or antibiotics. Misc. drugs used are Naferalin, Diosmin, Ru kuai xiao, and Phytoestrogens.[9,28,30,31]

CURRENT IMAGING MODALITIES:

The current imaging modalities are :-

1. Magnetic resonance (Gadolinium enhanced RODEO = rotating delivery of excitation off-resonance sequence);
2. Mammoscintigraphy by 99mm Tc SESTAMIBI Scanning, 99 mm Tc tetrafosmin scanning;
3. Lymphoscintigraphy and gamms probe for axilla;
4. FDG/ FES- PET (Positron Emission Tomography).

Mammography tends to underestimate tumour size, multifocality, and skips 5-15 % of cancer[33,34] USG is of limited value in detection of tumouir less than 1 cm, multifocality and intraductal disease. Mammography and USG are of limited value in assessment of response to chemotherapy and irradiated conserved breast.[33,34,35]

- **Magnetic resonance (Gadolinium enhanced RODEO - rotating delivery of excitation off-resonance sequence)**

MR IMAGES AND THEIR INTERPRETATION[36]

Images	Interpretation
No enhancement	Benign
Tiny stippled	Benign, usually stippled
Smoothly marginated	Benign
Lobulated	Benign, Fibroadenoma
Septated	Benign, Fibroadenoma
Clumped globular	Malignant, DICS
Clumped interspersed with tiny magnetic susceptibility	Malignant, Comedo DCIS
Linear ductal	Malignant, DCIS
Ring enhancing	Malignant, Invasive
Spiuculated	Malignant, Invasive

Magnetic resonance (Gadolinium enhanced RODEO=rotating delivery of excitation off- resonance sequence) or MR-CE-RO-DEO has shown sensitivity of 95%. MR is a method of choice in diagnosing muticentricity, as compared to USG and Mammography.[34] MR does not under-estimate tumor size in contrast to mammography and USG.[35] MR imaging picked up 84% multifocal disease as compared to 44% by mammography, and even less by USG.

MR imaging has a role in diagnosing axillary lymph nodes, as it enhances lymph nodes larger than five mm. MR-CE-RODEO is investigation of choice today in dense breast tissue which is significantly depicted by mammography.[36] This was proven in a series of 61 patients with breast cancer.[37]

MR-CE-RODEO is investigation of choice in diagnosing local recurrence in a conserved breast.[38] MR-CE-RODEO is investigation of choice in assessment of response to neo-adjuvant chemotherapy, thus allowing patients to be selected for breast conservative therapy. MR is accurate in the pathological determination of residual disease in 97% of cases.[39] MR is also presently investigation of choice in evaluating response to Interstitial Laser Photocoagulation of Breast Cancer.[40]

MR RODEO is more accurate than mammography and USG in local staging of breast cancer, diagnosis of local recurrence, assessment of response to neo-adjuvant chemotherapy and evaluation of silicon implants.

- **MAMMOSCINTIGRAPHY by 99mm Tc SETAMIBI SCANNING, 99mm Tc TETRAFOSMIN SCANNING**

These investigations have a sensitivity of 97% for T 2 tumors, 95% for T 1c tumors. However, for T 1a and T1b results are only 26% and 56%.[41] This has encouraged use of nuclear medicine guided stereotactic prebiopsy localisation of occult breast lesions.[42] And for preoperative and intraoperative localisation of non-palpable tumours.[43]

- **LYMPHOSCINTIGRAPHY AND GAMMA PROBE FOR AXILLA:**

Probe localisation of sentinel lymph node is becoming an integral part in the management of axilla in breast cancer patients.[44]

Mammoscintigraphy is also useful in detecting multidrug resistance in breast tumours by recognising P- glycoprotein.

FDG/ FES/ PET (Position Emission Tomography):

FDG-PET has a sensitivity of 70-90% and specificity of 85-95%.[45] It has got a good predictive value to response of neo-adjuvant chemotherapy.

Radiolabeled estrogen lignad FES- PET may have a role in detecting ER, PR and Axillary and mediastinal nodes.[45]

FUTURE:

Digital technology coupled with computer assisted substraction will be an additional tool in improving the results of MR-RODEO-CE, Mammoscintigraphy, and FDG-PET. Currently, less than 1 cm tumor cannot be diagnosed my Mammoscintigraphy.

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PET - CLINICAL APPLICATIONS IN ONCOLOGY

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Positron emission tomography (PET) is an imaging technology that delivers high resolution images using biologically active compounds, substrates or drugs labelled with positron emitters. Most physiological molecules are made up of carbon, nitrogen and oxygen which enable them to be labelled with ^{11}C, ^{13}N, ^{15}O, (and ^{18}F) that are positron emitters.This provides the clinicians and researchers with an unique tool to study and quantify physiological and pathological functions of human tissues and organs. Diagnosticians have traditionally been trained to analyse informations provided by structural and anatomically based techniques. Biochemical processes are, however, altered virtually in all disease states and these alterations usually precede gross anatomical changes. With the advent of molecular biology based medicine, a transition must be made to incorporate information based on biochemical pertuberations into diagnostic informations, without waiting for structural changes. PET provides such information. PET is also very useful adjunct to anatomical imaging techniques, providing unique informations and an additional dimensions to the characterization of disease.

Application of PET initially focussed on brain and heart. Now it is being primarily used in oncological indications. This development has resulted from successful application of Flourine-18-Fluro-deoxyglucose (FDG) to a growing number of clinical indications at varying stages of diagnosis, staging and follow up. Using FDG in vivo cancer imaging is based on the observation of enhanced glycolysis in tumour cells. A high rate of aerobic glycolysis (degradation of glucose to lactic acid in the presence of oxygen) in several types of cancer cells was first described by Warburg. This phenomenon has been linked to both increase in the amount of glucoase membrane transporters and an increase in the activity of the principal enzyms controlling the glycolytic pathways. It is important to stress that FDG uptake by neoplastic tumours in vivo remains under the dependance of other physiological factors, such as, tissue oxygenation, regional blood flow and peritumoural inflammatory reactions.

DIFFERENTIAL DIAGNOSIS

Solitary pulmonary nodule:- Predictive accuracy for both benign and malignant nodule is 94% in addition to reducing the complications encountered by other methods of investigations like transthoracic needle aspiration.

Pancreatic Mass:-

Several studies from Germany and Japan have evaluated the role of FDG PET in differentiations of pancreatic adeno-carcinoma from benign chronic prancreatitis and mass forming pancreatitis. Sensivity for carcinoma has been reported to be 94% with a specificity ranging from 78% to 90%.

STAGING

Initial staging by FDG PET has been useful in lung cancer, melaoma, sarcoma and Lymphoma. It is probabaly indicated

in other tumour types like ovarian, head & neck and pancreatic carcinoma, especially when the tumour is in an advanced stage or when metastatic lesions are suspected by conventional imaging or by raised tumour markers. Indeed, in these cases, FDG PET can provide sensitive whole body screening.

DIFFERENTIATION OF SCAR AND RESIDUAL DISEASE:-

Differentiation of scar and residual or recurrent disease is a frequent indication of PET and one of the first to be documented. It is used for lung, head and neck, colorectal carcinomas. FDG has also proven useful in the evaluation of residual masses after therapy for lymphoma.

DEMONSTRATION OF SUSPECTED RECURRENCES:-

In a suspected case of recurrence (by raised marker or other clinical signs) where conventional imaging fails to detect because of small size of recurrence PET may have immense value for its high sensitivity and whole body capability. It not only can confirm but can delineate the extent of recurrent disease. The impact of PET on management, avoiding unnecessary surgery, allowing more complete surgery forms the basis of cost effectiveness.

FOLLOW - UP THERAPY

FDG PET can be helpful in evaluation of therapeutic response well before morphologral decrease of tumour mass can be demonstrated by conventional imaging. Early determination of therapeutic resistance is also important to avoid the toxicity of an ineffective therapy and to allow selection of a new therapeutic regimes.

Thus PET has a tremendous potential for diagnosis and decision making in a complex oncological problem. Extensive clinical research are bring undertaken to find out the sensitivity and specificity in various situations and define its precise role.

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NATCON-IASO'99

(L-R Dr. S. SAINI, Dr. K.C. KOTHARI, Brig. P. SUBHASH, Sri. R.P. SHASTRY, Sri. V. DHASMANA, Dr. S. SAHA)

This year annual conference of the IASO was organised on 8th, 9th and 10th October at Himalayan Institute of Medical Sciences, Jolly Grant, Dehradun, located midway between Rishikesh and Dehradun in the foothills of Himalayas. The scenic beauty of the Himalayas, climate and sprawling campus of Himalayan Institute added to the charm of scientific activities.

The conference was preceded by a pre-conference workshop on 7th October on recent advances in management of lymph node basins. Live operations to demonstrate metastatic nodes to neck, axilla and groin and an interesting session of video presentations were the main activities of workshop. A large number of delegates from India and abroad participated in the workshop.

The conference activities lasted for two and half days from 8th to 10th October, 1999. There was an overwhelming response from participants, so a number of concurrent sessions were organized to make provision for all. Health minister of Uttar Pradesh Sri Rama Pati Shashtri inaugurated the conference. Brig. P. Subhas, during his presidential addess, lauded the growth of IASO and the excellent work being done by its members. Dr. D.D.Patel released the souvenir.

More than 300 delegatesw attended the conference. There were large number of foreign delegtes,notable among them were Dr. Sukamal Saha , Surgical Oncologist from USA; Dr. Michael Stephen, Hepato-billiary Surgeon from Australia; Dr. Madan Arora, Medical Oncologist; Dr. James Ruscinci, Surgeon and Dr. Donald Weaver, Surgeon from USA;

Dr. Zeev Horowitz, Head and Neck Surgeon from Israel; Dr. L.Arthur Firth, Radiation Oncologist from Canada; Dr. John Hogg, Surgeon from Australia and Dr. A.K. Sharma from Nepal.

Scientific activities included symposiums on ovarian, uterine cervix, oro-mandibular, uro-epithelial and testicular cancers. In addition to these there were good number of invited lectures on breast cancer, sentinel node mapping, gastro-intestinal cancers, hepatic resection and head & neck cancers. Moti Bhai Oration on "Personality of a surgical oncologist "by Dr. K.K.andey and Radha Devi Oration on "Carcinoma gall bladder" by H.S.Shukla were delivered during the conference.

One important aspect of conference has been to bring into the focus spiritual aspect of health and cancer in particular, highlighting the teachings of Himalayan sages and Swami Rama founder of Himalayan Institute of Medical Sciences. Lecture on Spirit and Cell, Leadership and medicine in 21st century and a session on "Stress management an essential partner in cancer control" were part of this conference.

Delegates has opportunity to visit holy cities of Rishikesh & Hardwar and hill queen, Mussourie.

Dr. SUNIL SAINI
(Organising Secretary)
ASSOCIATE PROFESSOR
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INFORMATION REQUIRED

Himalayan Institute of Medical Sciences, Jollygrant, Dehradun wants to start radio-therapy services in their hospital. They want to know about any funding agencies, non-governmental as well as governmental. Any information in this regard will be appreciated. Please write to :-

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CANCER CHEMOPREVENTION - PRESENT STATUS

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CHEMOPREVENTION is the use of pharmacologic intervention for disease prevention. The intent is to intervene in the pathophysiologic pathways that lead to clinical disease, but before clinical disease occurs. Although cancer chemoprevention is relatively new, there is a long history of chemoprevention in cardiovacular diseases, where interventions to counter hypertension, hypercho-lesterolemia and thrombosis have been used for years to prevent clinical atherosclerotic disease. As in cardiovascular diseases, chemopreventive agents may be identified through epidemiologic observations, through experiences with agents that have been used to treat frank cancer and through laboratory experiments. All of these modalities have identified successful cancer chemopreventive agents and also suggested some agents that have not been clinically successful.

ANTIOXIDANT SUPPLEMENTATION AND LUNG CANCER

In the early 1980s, the hypothesis was advanced that beta-carotene, a vitamin A precursor, might have important cancer chemopreventive effect. The carotenoids, including beta-carotene, are plant pigments that are widespread in nature; they are responsible for many of the red, orange and yellow colors of plant and the animals that eat them. In plants carotenoids function to protect the cells from damage caused by excited molecular species generated by photosynthesis. These properties provided the rationale for the proposed chemo-preventive nature of beta-carotene, under the assumption that this compound would function as an antioxidant and quench excited genotoxic molecular species. A variety of other possibilities to explain anticarcinogenic effects of carotenoids were also advanced, including induction immune effects. In humans, beta-carotene is among the carotenoids found in high tissue concentrations, and epidemiologic finding suggested that high intake of beta-carotene was inversely related to the risk of several malignancies, particularly lung cancer. Vitamin E (a term that refers to eight natural compounds, including the tocopherols) is another antioxidant that has been studied with regard to lung cancer prevention. Selenium, though not in itself an antioxidant is often considered to be in this group because of its important role in glutathione peroxidase, an antioxidant enzyme system. For several reasons the study of the relationship between nutrients such as beta-carotene or vitamin E is difficult. The characterization of diet over long periods of time presents serious measurement problems, and the resulting measurement error tends to obscure associations of diet with disease. Also nutrients are not found in isolation, they are found in foods in combination with hundreds of other compounds, some of which may be biologically active.

For these reasons clinical trials are needed to clarify the effects of nutrients on risk, despite the large amount of observational data available. The increased lung cancer risks (and increased overall mortality) from beta-carotene in the Alpha Tocopherol, Beta-cartone Trial and Beta-carotene and Retinol Efficacy Trial created much scientific and public concern. The explanation for the findings is not clear, but several possibilities have been advanced. Since the

Clinical trials began, it has emerged that beta-carotene may not be as effective an antioxidant in vivo as previously thought, a pro-oxidant effect of beta- carotene at high concentration has also been reported. The difference between the observastional studies and the clinical trial findings could have been due to several factors. There may well be important differences between a lifetime of eating foods containing a complex mixture of nutrients and a few years of high dose supplementation with a single compound. Although the clinical trials involved treatment periods as along as 12 years, most of the lung cancer cases that emerged during these studies were probably the result of neoplastic progression that was already well underway at the beginning of the studies. Beta-carotene supplementation might have a preventive effect in individuals without neoplastic but a harmful effect on tumors alredy initiated. Meanwhile, the apparent protective effect of beta-carotene in the observational studies could have been due to other constituents of food rich in beta-carotene or to other characteristic of individuals who eat those foods. The protective effect of selenium on lung cancer in the Skin Cancer Prevention Trial (relative risk [RR] = 0.5, 95% confidence interval [CI], 0.2 to 0.9) was unexpected, but this provocative positive finding cannot be considered conclusive. It is not clear whether the result reflects real protective efficacy or whether the observation is the result of the play of chance. Because there were many cancer sites studied at the end of the trial, it is possible that the risk of some of these would be low in the selenium group simply by chance.

ANTIOXIDANT CHEMOPREVENTION OF PROSTATE CANCER ?

Chance finding could also underlie the lower risk of prostate cancer observed for alpha tocopherol or selenium supplementation in trials that were intended to study other cancer sites. In the Alpha Tocopherol, Beta-Carotene lung cancer study vitamin E conferred an RR of 0.71 (95% CL, 0.5 to 0.9) for prostate cancer. For clinical tumors (stages II-IV) the RR was 0.6 (95% CL. 0.5 to 0.8) and there was a similar reduction in prostate cancer death. Selenium also led to a reduction in the risk of prostate cancer incidence and mortality in the Skin Cancer Prevention Trial (RR=0.4, 95% CL, 0.2 to 0.7) (Numbers of prostate cancer deaths were too few for meaningful analysis). Because of the possibility of chance effects, neither of these agents can be considered established preventive interventions for this cancer.

RETINOID CHEMOPREVENTION OF HEAD AND NECK CANCER

Retinoids are natural and synthetic analogs of Vitamin A, many of which act through specific receptors to modulate differentiation, the drugs can prevent - or in some case reverse - neoplastic phenotypes. Based on the premise, 13-cisretinoic acid(isotretinoin) was tested in patients with oral leukoplakia. Short -term (3 months) use of relatively high doses (1 to 2 mg/kg/d) was found to decrease the size of the lesions, reverse dysplasia, and inhibit progression to carcinoma. After this treatment, lower dose (0.5 mg/kg/d) maintenance therapy suppressed progression in contrast to beta-carotene, which was ineffective. Unfortunately the preventive effect is not sustained after cessation of treatment. Other retinoids also seem to be effective in preventing progression of leukoplakia to oral cancer (Vitamin A 200,000 IU/wk, 4-hydroxy-carbophenyl retinamide 40 mg/d or 4N-hydroxyphenyl retinamide 200 mg/d), at least over the short term. In trials of patients treated with surgery and/ or radiation therapy for head and neck cancer isotretinoin has also been effective in reducing the risk of second primary cancers (mostly smoking related cancer of the aerodigestive tract). After 12 months of adjuvant of treatment (1.5

mg/kg/d) suppression of second primaries lasted for approximately 3 years. Similar findings have been reported for adjuvant treatment of stage I lung cancer with retinol palmitate (300,000 IU daily for 1 year). However, etretinate, a synthetic retinoid, was not effective in preventing second primary cancer is one large trial among patients with oral cancer. A dose - dependent toxicity has been a feature of isotretinoin treatment, mucocutaneous effects, hepatotoxicity, and elevation in serum triglycerides. Unfortunately in some circumstances higher doses may be important for efficacy of this drug. In nonmelanoma skin cancer for example, 10 mg is ineffective in preventing recurrence, but 2 mg/kg/d reversibly reduced the number of cancers by more than 60% in xeroderma pigmentosum and 0.4 mg/kg/d may be effective for basal cell nevus syndrome. For patients with xeroderma pigmentosum or head and neck cancer with a very risk of cancers, some toxicity may be tolerable in view of the benefit. For lower-risk individuals, however (eg. in primary prevention) such toxicity is unacceptable, particularly because effective prevention may require prolonged aministration of the drug.

ANTIOXIDANT SUPPLEMENTATION AND COLORECTAL NEOPLASIA

Because of the size and complexity of trials that are adequate for studying colorectal cancer itself as an end point in prevention studies much prevention research on colorectal carcinogenesis has focused on adenomas, which are precursors to most colorectal cancers. Patients with sporadic adenomas are routinely followed with endoscopy, and patients with familial adenomatous polyposis (FAP) receive even more intense surveillane. Assuming that effects on adenomas reflect those on cancer, these end points provide a convenient end point for study of the prevention of colorectal cancer itself.

Antioxidants have been studied in many trials, beta-carotene most intensively. It is clear that beta-carotene is ineffective when given for up to 4 years in middle-aged subjects who have already had at least one adenoma. Vitamin E also seems to be ineffective. Several of the antioxidant lung cancer trials were large enough to accrue sufficient numbers of colorectal cancer cases for meaningful analysis; the findings confirm that several years of beta-carotene or vitamin E will not alter colorectal cancer risk. For vitamin C, an early study suggested some benefit but subsequent studies failed to confirm this. Thus as in lung cancer, beta-carotene and Vitamin E have not demonstrated beneficial effects for large bowel neoplasia. However the interpretation of these trials is also not straightforward. The end points in the adenoma trials were small adenomas (typically < 0.5 mm), the earliest visible signs of neoplasia in the bowel. Thus these investigations studied relatively early phases of carcinogenesis, and if beta-carotene affected later developments, the effect would be overlooked. Nontheless, more prolonged supplementation might be required and this caveat applies particularly to the large trials that studied clinical cancer.

The Skin Cancer Preventation Trials suggested that selenium might inhibit colorectal neoplasia, but as for the lung cancer and prostate cancer findings, it is not clear whether this is a chance finding or a reflection of genuine efficacy.

CHEMOPREVENTION OF COLORECTAL NEOPLASIA: OTHER PROSPECTS

The suggestion in the early 1980s that calcium intake might lower the risk of colorectal neoplasia generated considerable investigation. Although some epidemiologic studies have supported this hypothesis, these investigations have been mixed, perhaps reflecting the difficulties of dietary epidemiology. A recent clinical trial

has confirmed a modest beneficial effect of calcium carbonate supplementation on adenoma occurrence. Over a 4 year treatment period, the risk of an individual having a recurrent adenoma was decreased by approximately 15% and the number of adenoms reduced by approximately 25%. Findings that nonsteroidal anti-inflammatory drugs (NSAIDs) decreased colorectal neoplasia in animals also motivated numerous human epidemiologic studies. With few exceptions, these have pointed to a chemoprotective effect, suggesting that risk can be reduced by approximately 50% among individuals who take aspirin or other NSAIDs regularly. Indeed, clinical trials in FAP patients have uniformly found that sulindac can lead to polyp regression and prevention of new polyps. In patients with sporadic adenomas there have been some hints of efficacy, but the effect seems to be less striking than in FAP. Thus anti-inflammatory agents have considerable promise for chemoprevention of colorectal neoplasia. Tempering this enthusiasm, however is the difficulty of extrapolating findings from FAP to the population of patients with sporadic adenomas : to the extent that the biology of FAP differs from the sporadic situation, there could well be difference in efficacy. In addition, it is clear from the observational studies (of sporadic colorectal cancer and adenomas) as well as the clinical trials (of FAP) that long term continued use of the NSAIDs is necessary for prevention. In individuals without inherited colorectal cancer syndromes, 15 or more years will probably be required before there is a reduction in the risk of clinical colorectal cancer.

BREAST CANCER

The efficacy of tamoxifen as adjuvant treatment of breast cancer is well established. With 5 years of treatment, the drug confers approximately a 50% reduction in the risk of recurrence in women with early breast cancer. The benefit is concentrated in patients who have estrogen receptor (ER)-positive tumors, although there may be a small reduction in risk among women with ER-negative disease as well. However both younger and older women benefit. More relevant to cancer prevention is the effect of the drug to preventing contralateral breast cancer : again, approximately a 50% reduction with 5 year of treatment. In this case, women with both ER - positive and ER - negative primary cancers have about the same relative benefit, and the reduction in risk was similar in younger and older age group.

Building on these data, the Breast Cancer Prevention Trials studied 20 mg/d of tamoxifen in more than 13,000 women without breast cancer but deemed to be at higher than average risk. The trials was stopped after a median follow-up of approximately 4-5 years; tamoxifen reduced the risk of breast cancer by almost 50%. Only ER-positive tumors were affected; there was no reduction in risk for ER- negative breast cancer. The benefit entailed a cost : increase in the risk of endometrial cancer, venous thromoboembolism, and possibly stroke. Preliminary results from two European trials have not confirmed the finding but the small size (and consequent variability) of these studies and difference in the patient population leave the .Breast Cancer Prevention Trials as the dominant result.

In conclusion chemoprevention research to date has provided many surprises and illustrated many of the difficulties that may accompany studies in the future. The adverse finding of the beta-carotene trials has forced a re-evaluation of the assumptions underlying a whole body of research. Negative trials introduce their own questions : whether the dose tested was too low and whether the administration of the agent was too late or too limited in duration. Positive findings for the main

hypothesis of a trial are relatively straightfoward, but in secondary analyses, chance findings can be difficult to interpret.

Nonetheless, cancer chemoprevention is a reality. Tamoxifen can be considered to be an established preventive drug for breast cancer, and isotretinoin seems to be effective is in preventing second primary cancers in patients with cancers of the head and neck. However understanding these effective agents is not straightforward. Although on average both drugs provide a net benefit, it is likely that for some low-risk patients, their adverse effects will counter-balance their benefits. On the other hand, a very nontoxic agent, calcium carbonate, has been shown to have chemopreventive efficacy against large bowel carcinogenesia in one clinical trials but its modest effect may not lead to change in follow-up recommendations. Balancing efficacy against toxicity may well be a recurring difficulty for follow-up of successful chemoprevention research. This balance is made more difficult because of the apparent need for continuous use of chemopreventive interventions; to date, no agent has been found to confer a "permanent" protective effect.

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ONCOINFOSCAN

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IMMUNOTHERAPY - A RAY OF HOPE IN PANCREATIC CANCER

Inspite of poor results in pancreatic cancer, the search for more accurate methods of diagnosis and better methods of treatment should be actively pursued, a recent review on pancreatic, cancer states. In the light of this observation, further study of immunotherapy may be appropriate.

Immunotherapy is currently at the fore, as we can now , actively or passively, stimulate the immune system of patients with pancreatic cancer, creating an immunotherapeutic regimen which may be partially or completely effective in curing the disease. Why this optimism ? Recently, developments in genetic engineering techniques have led to breakthroughs in identifying tumour antigens, the description of numerous cytokines (approximately 25, including 1L 1-18, tumour necrosis factor-(TNF) 0/0, interferons (INF) α,β,γ and others) and more recently, chemokines, of which there are more than 30, including their receptors. Possible peptides targets in immunotherapy for pancreatic cancer are as shown in the table.

ANTIGEN	Appro. % incidence
KAL 1	89
MUC 1	80
Her 2/neu	50
PAP	20
NT-3	72
Cfos	75
p53	67
CAI9-9	76
TPA	85
TUM2-PK	71
CEA	39

MUC I is found in virtually all pancreatic cancers. The current plethora of agents will take many years to sort out, but better response will be generated by Granulocyte macrophage colony stimulating factor (GM-CSF) before blood harvest, IL-4 and GM-CSF for dendritic cells possibly with IL-7, and followed by IL-12.

A BREATHALYSER FOR LUNG CANCER

Many volatile organic compounds (VOC), principally alkanes and benzene derivatives, have been identified in breath from patients with lung cancer. Breath samples from 108 patients with abnormal chest radiograph who were scheduled for bronchoscopy were collected with a portable apparatus, then assayed by gas chromatography and mass spectroscopy. Lung cancer was confirmed histologically in 60 patients. A combination of 22 VOCs, predominantly alkanes, alkane derivatives, and benzene derivatives, discriminated between patients with and without lung cancer, regardless of stage (all $p<0.0003$). For stage 1 lung cancer a combination of 22 VOCs had 100% sensitivity and 81.3% specificity. Cross validation of the combination correctly predicted the diagnosis in 71.7% patients with lung cancer and 66.7% of those without lung disease.

Testing for breath VOC profiles might complement other innovative methods currently being investigated. either as markers of early cancer or, perhaps more importantly, as markers of pre neoplastic bronchial epithelial changes. There is hope that in future a breathalyser will be used for more than screening for ethanol intoxication.

BIOMARKERS IN LUNG CANCER

The expression of several putative surrogate biomarkers in sputum cytology has been studied. Preliminary results suggest that the most accurate marker for prediction of an antigen detected by monoclonal antibody 703 D4. The accuracy (True positive + True negative/ Total) of this biomarker was 88% in 62 archived dysplastic (but not diagnostic) specimens collected 2 years in advance of clinical lung cancer. The antigen target was subsequently identified as heterogenous nuclear ribonucleoprotein (hn RNP) A2/B1. hnRNP is an RNA binding protein that is required for maturation of mRNA precursors. The predictive value of hnRNPA2/B1 overexpression has been prospectively assessed in two high risk populations: 595 patients with stage 1 resected lung cancer, for whom the annual risk of second primary lung cancer is 1-5%: and 6285 Chinese tin miners with extensive exposure to tobacco smoke, radon, and arsenic, among whom the annual incidence of lung cancer is 1%. In these two populations, hnRNP A2/B1 overexpression predicted lung cancer in 67% and 69%. a 35-fold and 76-fold improvement in positive predictive value over background cancer risks of 2.2% and 0.9% respectively.

MOLECULAR BIOLOGY IN GALL BLADDER CANCER

Recently, genetic, immunohistochemical and molecular biology techniques have been applied to explore the pathogenesis of cancer of gallbladder. The p53 tumour suppressor gene, located on the short arm of chromosome 17, has roles in cell division and apoptosis. The data suggests that corelation exists between p53 abnormalities and the development of gall bladder cancer. In one study 8 out of 11 gallbladder cancers expressed p53 immunopositivity. p53 Protein expression was related to higher grades of malignancy. K-ras mutations have also been investigated and more recent evidence suggests that K-ras mutation may also be an important factor in the early stage of carcinogenesis when associated with an anomalous junction of the pancreatico-biliary tract. Malats et al found that K-ras 12 mutations were an independent prognostic indicator in patients with extra hepatic biliary system cancer, including gallbladder cancers and also postulated that mutations at codons 13 and 61 might have oncogenic potential. Biliary phospholipase A2 is also associated with such cancers.

TAMOXIFEN --- THE WONDER DRUG ?

Is there anything that Tamoxifen cannot do ? An overview published in Lancet, 1998, suggests that the scope and effectiveness of the drug as an adjuvant have been underestimated. Now Bernard Fisher and colleagues in 1999, report that Tomoxinfen reduces, by about 40%, the risk of cancer recurrence in women treated for ductal carcinoma in situ (DCIS). In this randomised trial, NSABP-24, about, 1800 women with DCIS were treated with lumpectomy, radiotherapy, and either tamoxifen or placebo. Primary end points were same (ipsilateral) or opposite breast (contralateral) invasive or in situ tumours. After a median follow-up of 5 years, the incidence of breast tumours was 13.4% among the controls and 8.2% in treatment group (p=0.0009), a relative reduction of nearly 40%. Recurrence of DCIS was also reduced (a relative reduction of 30%). Although most recurrences were ipsilateral, contralateral tumour recurrences, both invasive and in situ, also developed and were reduced by about 40%.

A reasonable view of the data presented should be that the benefits of tamoxifen far outweigh the risks. The question arises - Should all women with

mammographically detected DCIS have tamoxifen ? Probably - no. Should most ? Probably - Yes.

FAMILY HISTORY AND CANCER MORTALITY

What is the association between family history of cancer and cancer mortality in women ? A case controlled study nested within a large cohort, the American Cancer Society Cancer Prevention Study-1 was conducted to test associations between a family history and cancer mortality in women. By using logistic regression, the authors analysed family history, as reported by 429483 women enrolled in 1959, relative to subsequent mortality through 1972 from cancer within and across multiple sites. The association between family history and cancer mortality were generally stronger within cancer sites than across cancer sites. Within site associations were found for breast cancer (odds ration OR=1.91), colorectal cancer (OR=1.6), stomach cancer (OR=1.9), and lung cancer (OR=1.7).

Across site associations were observed for a family history of (1) Breast cancer as a risk factor for ovarian cancer mortality (OR=1.6), (2) Stomach cancer as a risk factor for ovarian cancer mortality (OR=1.5), and (3) Uterine cancer as a risk factor for pancreatic cancer mortality (OR= 1.6). A general pattern of positive association was observed between a family history of cancer at several sites and subsequent death from pancreatic cancer. These findings suggest that inherited cancer - susceptibility genes increase the risk of cancer at many sites and are not specific to cancer risk within a single site.

SEROLOGICAL TESTS FOR PANCREATIC CANCER

The role of serological tests for screening of pancreatic cancer has not been established. However Yiannakon et al (1997) have prospectively assessed a new type of combined lectin/antibody enzyme - linked mucin assay, (CAM 17.1), in 250 patients whose differential diagnosis included pancreatic cancer. The control group comprised of 75 patients who did not have symptoms of pancreatic cancer and had alternative diagnosis. The patients were followed up for at least 8 months.

Of the 250 patients, 36 had pancreatic cancer, as defined by histological and imaging criteria, and 8 of these patients had a resectable tumour. Sensitivity and specificity of the CAM 17.1 assay were 86% and 91% respectively, in all patients, 85% and 81% in those who presented with jaundice, and 89% and 94% in patients who did not have jaundice. The sensitivity of the assay compared well with that of ultrasound scanning (59%) and CT (83%) in these patients. Use of the CAM17.1 assay in combination with ultrasonography identified 94% of patients with pancreatic tumours and all of those with resectable tumours.

RADIATION ENTERITIS

More and more patients are now receiving radiotherapy and chemotherapy for various intra-abdominal malignancies. The hazards of these are troublesome post-radiation enteritis. Columnar epithelial cells of the intestine, are extremely susceptible to radiation as they are rapidly proliferating cells. It is postulated that the damage can be minimised by reversibly preventing cell cycle progression before radiotherapy. Transforming growth factor (TGF) is such an agent. TGF, in experimental animals can effectively protect enterocytes against agents like vinblastin, vincristine, taxol, methotrexate, 5FU, etc. However, TGF is not effective against cisplatin and adriamycin which act

throughout the cell cycle. Keratinocyte growth factor (KGF), akin to the fibroblast growth factor has also been shown to prevent radiation injury if given before radiotherapy. Following radiation enhanced production of prostaglandin E2 has been shown to promote crypt cell survival. Vitamin A is also a potential protector against radiation injury to the bowel. Enteral glutamine, a non essential amino acid, has mucosal barrier action and hence acts as a radioprotector.

CANCER STOMACH

Stomach partioning gastrojejunostomy has been shown to give better results than a standard gastrojejunostomy. Ability to tolerate food and survival (13.4 v/s 5.8 months) are better in the former case. In unresectable disease, hyperthermic chemoperfusion has shown good results (median survival 4 years). Using bone marrow immunohistochemistry and tumour immunohistochemistry, Heiss et al have shown that low levels of plasmino gen activator-inhibitor (PAI-1) urokinase type plasminogen activator (UPA) and cathepsin (UPA activator) are associated with better disease-free survival. Aggressive tumours, on the other hand, E-cadherin-catenin, CD44, cathepsin B, plasma matrix metalloproteinase, platelet-derived growth factor, epidermal growth factor, cyclin DI and cyclin E. Tumours with these markers have a higher pathological stage with higher nodal status.

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PROGRAM-IASO Section
ASICON'99 - Madurai

Tuesday, December 28, 1999 ---- Day 1

12.00 Noon to 1.45 PM **SYMPOSIUM : PAROTID TUMORS**

Convener : Dr. A. K. Dewan, New Delhi

Surgical Anatomy & Pathology	12 mins	Dr. A. K. Dewan, New Delhi
Approach to Parotid	12 mins	Dr. V. K. Mallik, New Delhi
How I Do it? Superficial Parotidectomy	15 mins	Dr. Somesh Chandra & Dr. Kiran Kothari, Ahmedabad
Management of Advanced Parotid Tumors - (including deep lobe tumor)	15 mins	Dr. Samir Mehta, Mumbai
Complications of Parotid Surgery	15 mins	Dr. Sandeep Mehta, New Delhi
Parotid and Radiation	12 mins	Dr. Jayaraman, Madurai
Conclusions	9 mins	Dr. A. K. Dewan, New Delhi
Discussion and Audience Participation	**15 minutes**	

1.45 PM to 2.30 PM **LUNCH**

2.30pm to 3.30 PM **FREE PAPERS :SESSION - 1**

Chair Persons: Dr. K. Panda, Cuttack
Dr. Garima Mehta, Udaipur

1. Evaluation of Near Total Thyroidectomy for Differentiated Thyroid carcinoma
   Lt. Col. V.P. Singh ; T.A.Majeed, B. Fanthome, Ramesh Kumar, Mumbai.
2. An analysis of metastasis of unknown origin presented as neck node
   Dr. A. Suresh Venkatachalam, Coimbatore
3. Prognostic factor in head and neck sarcoma, results of univariate and multivariate analyses
   Dr. Elizabeth Mathew, Trivendrum
4. Radical neck dissection for secondaries neck - What should be the timing?
   Vinod Malik, R. K. Karwasra, J. C. Dhall, Rohtak
5. Radical cholecystectomy for carcinoma of gall bladder
   Dr. Kundan Kumar, J.Ramkumar, H.S. Shukla, Varanasi
6. Cancer trends in the coal fields - Epidemiological study
   Dr. Ajay Vidhyarthi , R.R. Sinha, Ranchi.

3.30 PM to 5.00 PM **INVITED LECTURES**

Chair Persons: Brig. P. Subhas, New Delhi
Dr. H. S. Shukla, Varanasi

1. **Dr. Tjakra Wibawa Manuaba, Indonesia** : Locally advanced Breast cancer
2. **Dr. Itamar Kott, Israel** :Acute toxicity and efficacy of Chemo-irradiation for carcinoma of rectum
3. **Dr. Fausto Badellino, Italy** : The state of the art of Radioimmunoguided Surgery

5.00 PM to 5.30PM **SMT. K.K. RADHADEVI ORATION - Speaker : Dr. S.P. Kharey, Mumbai.**

Chairpersons: Dr. K. K. Maudar, Ranchi
Dr. L. Sarangi, Varanasi

5.30 PM to 6.30 PM **IASO EXECUTIVE COMMITTEE MEETING**

PROGRAM-IASO Section
ASICON'99 - Madurai

Wednesday, December 29, 1999 ---- Day 2

9.30 AM to 10.15 AM — *ONCOQUIZ*
Quiz Master: Dr. Somesh Chandra, Ahmedabad

10.15 AM to 11.00 AM — *Short Guest Lectures*

11.00 AM to 11.30 AM — *Coffee Break*

11.30 PM to 1.30 PM — *PANEL DISCUSSION: "GASTRIC CANCER"*
Moderator: Dr. R. I. Dave, Ahmedabad

Introduction and Epidemiology	15 mins	Dr. R. I. Dave, Ahmedabad
Staging and Genetics	12 mins	Dr. S. K. Shukla, Indore
Role of endoscopy in diagnosis and management	15 mins	Dr. Kiran C. Kothari, Ahmedabad
Surgery for Gastric Cancer	15 mins	Dr. Hemant Raj, Chennai
Rationale for lymphadenectomy	15 mins	Dr. Dhananjay Sharma, Jabalpur
Role of Chemotherapy	12 mins	Dr. T. Raja, Madurai
Conclusion	6 mins	Dr. R. I. Dave
Discussion and Audience Participation	**30 mins**	

1.30 PM to 2.30 PM — *LUNCH*

2.30 PM to 4.35 PM — *"HOW I DO IT?" VIDEO SESSION*

Chair Persons: **Dr. N. N. Khanna, Varanasi**
Dr. T. Gunasagaran, Chennai

1) Total 3 Stage Radical Esophagectomy	:	**Dr. Sanjay Sharma, Mumbai**
2) Low Anterior Resection (Stapled)	:	**Dr. Kiran C. Kothari, Ahmedabad.**
3) Total Thyroidectomy	:	**Dr. R. K. Karwasra, Rohtak**
4) Total Cystectomy with Ileal Reservoir	:	**Dr. Tongaonkar, Mumbai**
5) Mandibular Reconstruction	:	**Dr. Omrish Coshic, New Delhi**
6) Abdominoperineal Resection	:	**Dr. K. S. Gopinath**

4.35 PM to 5.30 PM : **IASO GENERAL BODY MEETING**

PROGRAM-IASO Section
ASICON'99 - Madurai

Thrusday, December 30, 1999 ---- Day 3

9.30 AM to 11.00 AM : PANNEL DISSCUSSION : MUO-NECK

Moderators: Dr. Kiran Kothari, Ahmedabad & Dr. Nootan Shukla, New Delhi

Introduction		
Approach to patient	10 minutes	Dr. Kiran Kothari, Ahmedabad
Role of Immunohistochemistry in diagnosis	10 minutes	Dr. Paul Sebastian, Trivendrum
Role of Imaging (for Primary)	10 minutes	Dr. Manish Bhatria, Ahmedabad
Role of Surgery	10 minutes	Dr. K. Ramendrun, Trivendrum
Role of Radiotherapy	10 minutes	Dr. Manoj Pande, Trivendrum
Results of Treatment	10 minutes	Dr. Jayaraman, Madurai
Conclusions	10 minutes	Dr. A. Suresh Venkatachalam, Coimbtore
	5 minutes	Dr. Kiran Kothari, Ahmedabad
Audience Participation and Discussion	**15 minutes**	

11.00 AM to 12.00 Noon : FREE PAPERS - SESSION-2

Chair Persons: **Dr. Mohd. Iqbal Ahmed, Thiruvananthapuram**
Dr. K. S. Gopinath, Bangalore

1. Sarcomas of the breast (other than cystosarcoma phyllpides)
   Dr. Manoj Pandey, Trivendrum.
2. Impact of Newer Investigations on detection and surgery of early Breast cancer
   Brig. K.K.Maudar, Dr. M. Durai Swamy, Comdr. S.K.Mohanty, Ranchi.
3. Tumor Angiogenesis K167 and AgNOR in early Breast cancer
   Dr. Himanshu Choudhary, M. Kumar, A.K.Khanna, Varanasi
4. Treatment Options for Carcinoma Esophagus - How to decide?
   J. C. Dhall, R. K. Karwasra, A. R. Bansal, Rohtak
5. Sphicter conservation for anoractal cancers - What are the surgical options?
   A. R. Bansal, R. K. Kawasra, J. C. Dhall, Rohtak
6. Leimyosarcoma of the gastrointestinal tract: A clinicopathological review
   Naresh K Soni, Ajay Kumar, Jayesh Prajapati, Jalaj Baxi, Jignesh Goswami, Ketan Trivedi, J J Patel, D D Patel

Departure

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

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SEMINAR ON PROGRESS IN ONCOLOGY

"A National Seminar on Progress in Oncology - Directions for the next Millennium" was held at K.G. Medical College, Lucknow on 12 & 13th Nov'99, sponsored by Surgical Oncology Department of K.G. Medical College Lucknow & IASO. The seminar was highly appreciated and meaningful.

Visitors

International faculty

1. Prof. I. Taylor. England
2. Prof. T. Takahashi. Japan
3. Prof. D. Khayat. France
4. Prof. K. Kawabata. Japan
5. Prof. Chitti Moorthy. USA
6. Prof. A.K. Sharma. Nepal.

National Faculty

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22. Prof. K.K. Pandey
23. Prof. G. Kilara
24. Prof. S.K. Shukla
25. Prof. D.C. Doval
26. Col. V.P. Singh

Topics Discussed

- Thyroid Cancer
- Benign Breast Disease
- Head and Neck Cancer
- Newer Agents in Anticancer Treatment
- Gastric Cancer
- Soft Tissue Sarcoma
- Newer Direction in Radiation and Organ Preservation
- Gene Therapy

"Public symposium" on Cancer Awareness.

An open house with public participation, Question & Answer Session also was a successful venture.

INDIAN ASSOCIATION OF SURGICAL ONCOLOGY

(A Section of ASI)

MEMBERSHIP APPLICATION FORM

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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.

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Name of Member ASI ASI Number Signature

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

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