The 1st International Conference on Esophageal Cancer
The 7th Chinese Conference on Esophageal Cancer
中国首届国际食管癌学术会议
暨第七届全国食管癌学术会议

September 16th~19th, 2005

Zhengzhou, China

2005年9月16日~2005年9月19日

中国·郑州

主办单位：

中国抗癌协会食管癌专业员会　郑州大学医学院

中国医学科学院肿瘤研究所·肿瘤医院

河南省抗癌协会　河南省肿瘤医院　肿瘤研究所

支持单位：

河南省科学技术协会　河南省国际交流发展促进会

中国首届国际食管癌学术会议
暨第七届全国食管癌学术会议
大 会 日 程

大会开幕仪式·晚宴　9月16日晚19:00

主持人:张汝刚教授、董子明教授　中州皇冠假日酒店三号楼二楼中原厅

1. 大会秘书长董子明教授宣布大会开幕仪式开始,介绍出席开幕式的各位领导和专家
2. 大会主席陆士新院士致开幕词
3. 领导同志讲话
4. 大会执行主席张汝刚教授主持开幕式晚宴

大会报告9月17日上午8:00—12:00

主持人:张汝刚教授、郑玉玲教授　中州国际快捷假日酒店14楼会议厅

报告人	报告题目
1. 孙燕院士	食管癌的内科治疗研究进展
2. Mark Krasns, M. D.	食管癌的临床分期与综合治疗研究 Clinical staging of esophageal cancer and complex treatment of seophageal cancer
3. 殷蔚伯教授	食管癌的放射治疗研究进展
4. Reinhard Gessner, Dr.	Li－cadherin 结构、功能和表达与胃肠道肿瘤发生 Li－cadherin structure, function and expression during pathogenesis of gastrointestinal carcinomas
5. 沈忠英	人乳头状瘤病毒与食管癌
6. Zhongxing, Liao. , M. D.	美国食管癌研究现状 Present state of esophagus cancer study in American
7. 乔友林教授	中国林县食管鳞癌的硒蛋氨酸及 Celecoxib 化学预防研究
8. GSW Tsao, M. D.	人食管上皮细胞永生化和转化的分子机制 Moleculau mechanism involved in immortalization and transformation of human esophageal epithelial cells

大会报告9月17日下午14:00—19:00

主持人:王瑞林教授、董子明教授　中州国际快捷假日酒店14楼会议厅

报告人	报告题目
1. 张汝刚教授	食管癌的综合治疗研究进展
2. Nishimura, M. D.	食管癌的放疗研究进展

	Progrsee in radiation therapy for esophageal cancer
3. 董子明教授	食管癌中 DNA 聚合酶 β 表达与突变
4. Zigan Dong, M. D.	食管癌中 p16 分子基础 Molecular basis of p16 for esophageal cancer
5. 杨观瑞教授	中国早期食管癌内镜诊断和治疗研究现状与展望
6. Lung, Maria, Ph. D.	食管癌候选抑癌基因功能鉴定 Functional identification of candidate tumor suppressor gene in esophageal cancer
7. Wei Cao, M. D.	美国洛杉矶地区食管癌易感基因多态性病例对照研究 Polymorphism of Susceptibility Genes in Esophageal Cancer, a Case - Control study in Los Angeles, United States
8. 王立东教授	河南省食管癌与贲门癌癌变多阶段演进的分子机制
9. Wancai Yang, M. D.	食管癌生物治疗的分子学基础 Molecular basis for esophageal cancer biotherapy
10. Gwendolyn E. P. Zahner	流行病研究的伦理学 Bioethics in Epidemiological Research

中国抗癌协会食管癌专业委员会会议　9 月 17 日晚 20:00—22:00
主持人:张汝刚教授、平育敏教授　地点:中州皇冠假日酒店 1 号楼 14 楼会议室

1. 选举第三届委员会主任、副主任和常委
2. 研究成立国际食管癌学术组织
3. 研究制订食管癌防治研究协作规划和三届委员会学术活动计划

基础与预防分大会报告　9 月 18 日上午 8:00—12:00
主持人:沈忠英教授、张云汉教授　中州皇冠假日酒店 1 号楼 4 楼会议室

报告人	报告题目
1. 陆士新院士	食管癌病因学研究进展
2. Bruce Greenwald, M. D.	Barret's 食管、食管增生、原位癌的流行病学和分子生物学演变 Epidemiology and molecular biology development of Barret's esophagus; esophageal proliferation, carcinoma in situ
3. 杨文献教授	中国林州市食管癌高发区人群病因学预防试验研究初步报告
4. 张祥宏教授	Sterigmatocystin 污染在中国人食管癌发生中的意义
5. 张国红教授	基底膜改变在食管上皮增生和癌变过程中作用的分子机制初步研究
6. 杨治华教授	食管癌癌前相关抗原自身抗体与食管癌癌前筛查
7. 林昆教授	中国南方食管癌高、低发区人群尿总 N - 亚硝基化合物与 N - 亚硝基氢基酸的相关性
8. 张雪梅教授	COX - 2 启动子区基因变异筛选和功能分析以及和食管癌易感性的相关关系
9. 侯浚教授	磁县食管癌的防治
10. 邹小农教授	中美协作项目——林县营养干预试验
11. 陶德明研究员	3 万人群核黄素强化营养盐对食管癌的干预研究

临床分大会报告　9 月 18 日上午 8:00—12:00

主持人:平育敏教授、陈文虎教授　中州国际快捷酒店 14 楼会议厅

报告人	报告题目
1. 邵令方教授	食管癌与贲门癌外科治疗研究
2. 平育敏教授	食管癌和贲门癌 20000 例外科治疗经验
3. 张云汉教授	食管癌的病理学研究进展
4. 祝淑钗教授	胸段食管癌临床分期与病理 TNM 分期对比分析
5. 肖泽芬教授	淋巴结转移数目对食管癌胸段切除生存的影响及其放疗的价值
6. 樊青霞教授	奈达铂和替加氟治疗食管癌临床疗效分析
7. 宋太民主任	中晚期食管癌动脉插管灌注化疗疗效观察
8. 毛友生教授	食管癌和贲门癌患者术后呼吸衰竭原因分析及防治
9. 高宗人主任	食管癌术前放疗 654 例临床资料分析
10. 胡祎教授	超声内镜在食管癌术前分期的临床价值
11. 方文涛主任	选择性颈胸腹三野淋巴结清扫治疗胸段食管鳞癌

外科专业分会　9 月 18 日下午 14:00—19:00

主持人:戎铁华教授、高宗人主任　紫荆山宾馆四号楼一楼会议室

报告人	报告题目
1. 程贵余	内镜超声检查术在早期食管鳞癌术前分期中的作用
2. 胡	超声内镜和 CT 在食管癌术前 T、N 分期的诊断价值
3. 佘志廉	Tis 和 T1 食管癌的外科治疗
4. 吴明拜	食管癌、贲门癌的外科治疗—附 2019 例临床分析
5. 黄壮士	经胸小切口单手辅助器械操作微创治疗食管、贲门癌手术万法探讨
6. 师晓天	胸腔镜下食管癌的外科治疗
7. 寿化山	X 线钡餐造影、B 超检查对贲门癌切除可能性的对比研究
8. 徐海洋	根治性放疗后复发食管癌的外科治疗
9. 刘俊峰	食管癌与贲门癌术后并发症——单中心 50 年经验总结
10. 杜晓东	颈段食管癌手术喉功能保留的探讨
11. 曹景峰	食管胃层层吻合治疗食管癌的临床研究
12. 魏锦昌	原发性食管腺癌的临床诊治(附 43 例分析)
13. 郭石平	食管贲门癌切除术后并发食管胃大动脉瘘—附 18 例报告
14. 孙　伟	食管癌气管、支气管隆突浸润早期的 CT 诊断与术后病理对照研究
15. 傅建华	预防性胸导管结扎术在食管癌根治术中的价值

放疗与微创治疗专业分会　9 月 18 日下午 14:00—19:00

主持人:万钧教授、王建华主任　紫荆山宾馆三号楼一楼会议室

报告人	报告题目
1. 陈俊辉	热休克蛋白在食管鳞状细胞癌中的表达及其意义
2. 祝淑钗	500 例中晚期食管癌单纯放射治疗的多因素分析

3. 高献书	食管癌放疗后 Cox 模型多因素分析
4. 周志国	食管癌每周连续 7 天常规分割加速放射治疗的Ⅱ期临床研究
5. 严福来	食管癌术中放疗疗效观察(30 例近期随访)
6. 李云英	食管癌根治术后配合化放疗的临床观察
7. 徐传良	放化同步并后程超分割放射治疗中晚期食管癌
8. 卢渊泉	$T_{1-4}N_1M_0$ 期食管癌放化同步及序贯治疗的临床分析
9. 马　群	放化结合治疗食管癌疗效观察
10. 宋祥玉	卡铂加放射治疗食管癌的前瞻性研究
11. 盖晓惠	外照射配合 252 锎中子射线腔内照射治疗食管癌的临床随机研究
12. 高献书	沙培林联合放射治疗与单纯放射治疗食管癌的疗效观察与比较
13. 王俊生	PDT 治疗早期食管癌临床研究
14. 郑颖娟	食道癌放疗后复发光动力治疗的临床研究
15. 冯笑山	食管/贲门癌光动力治疗光照时机选择的临床研究
16. 申　斌	食管支架的临床应用
17. 薛晓英	TSA 对食管癌细胞 TE13 及 TE13R120HDAC3 表达的影响及其放射增敏作用
18. 张　萍	放射抗拒性食管癌细胞系的建立及基因表达差异分析

化疗与生物治疗专业分会　9 月 18 日下午 14:00—19:00

主持人:石远凯教授、樊青霞教授　紫荆山宾馆三号楼四楼会议室

报告人	报告题目
1. 李醒亚	67 例晚期食管癌肺转移临床特点及化疗反应
2. 张蓬原	STI—571 对食管癌细胞株体外杀伤作用及机制的研究
3. 王　瑞	胰岛素对 5－氟脲嘧啶的增效作用及其机制
4. 郭宏强	化放疗未控或复发食管癌动脉内灌注化疗疗效观察
5. 任中海	CARBO + PDD + 5－Fu 方案治疗中晚期食管癌 98 例临床研究
6. 刘润森	羟基喜树碱联合氟尿嘧啶、顺铂治疗晚期食管癌的疗效观察
7. 宁　宇	流式细胞学细胞周期分析预测晚期食管癌化疗疗效
8. 马　望	食管癌患者血清中胰岛素样生长因子－1(IGF－1)检测的临床意义
9. 宋太民	经导管动脉灌注化疗治疗溃疡型和伴溃疡髓质型食管癌

标志物与诊断专业分会　9 月 18 日下午 14:00—19:00

主持人:张云汉教授、张祥宏教授　紫荆山宾馆二号楼二楼会议室

报告人	报告题目
1. 贺付成	食管鳞癌组织 NDRG1 基因的表达研究
2. 郑世营	FasL、B7－1 联合基因修饰的食管癌细胞诱导抗食管癌主动免疫
3. 张红新	血管内皮生长因子－c 在食管癌中的表达及其与淋巴结转移的关系
4. 张会娟	CD105 和 VEGF 在食管鳞癌组织中的表达及意义
5. 王新华	STAT3 在食管鳞癌细胞系中的组成性激活
6. 陈奎生	食管癌细胞中肝素酶 mRNA 的表达

病因与预防专业分会　9月18日下午14:00—19:00

主持人:林东昕教授、侯浚教授　紫荆山宾馆二号楼二楼会议室

报告人	报告题目
1.徐致祥	食管癌"氮循环"病因假说及部分验证
2.王士杰	河北省涉县2000—2004年恶性肿瘤发病与死亡情况分析
3.陈志峰	食管癌高发区重复癌及重复高级别上皮内瘤变患病分析
4.苏　敏	潮汕地区南澳岛食管癌的遗传流行病学研究
5.贺宇彤	食管癌高发区MTHFR基因C677T多态与食管癌、贲门癌遗传易感性的关系
6.宋　谦	食管癌高发区与非高发区线粒体DNA变异的比较研究
7.韩建英	改水对林州市食管癌发病率和死亡率的影响
8.韩小友	山西省食管癌患者血缘亲属、父系母系及性别食管癌患病风险比较研究
9.李媛媛	食管癌相关基因1 Arg290Gln的多态性与食管癌的易感性
10.李　沛	用cDNA微阵法整体分析原发性食管癌的基因表达谱

大会闭幕式晚宴

时间:9月18日晚　19:00—21:00

主持人:杨文献教授、常贵生研究员

大会执行主席张汝刚教授致闭幕词

紫荆山宾馆二号楼二楼餐厅

论 文 目 录

Epidemiology and molecular biology development of Barrets esophagus; esophageal proliferation, carcinoma in situ

Bruce D. Greenwald

Associate Professor of Medicine, GI Division University of Maryland School of Medicine

Barrett's esophagus is a condition characterized by the replacement of normal squamous esophageal epithelium by specialized intestinal metaplasia. It is believed to be caused by chronic reflux of gastric, and possibly duodenal, contents into the esophagus, resulting in reflux esophagitis and healing with the metaplastic epithelium. It is found predominantly in White men. It is found in approximately 1% of those with chronic gastroesophageal reflux disease (GERD). Risk factors associated with Barrett's esophagus include increased severity and duration of acid reflux, obesity, and the use of medications which decrease lower esophageal sphincter pressure.

The primary reason that Barrett's esophagus is of interest is because of the risk of progression to esophageal adenocarcinoma. The risk of progression is estimated to be 0.5% per year. The development of neoplasia develops through dysplasia. Standardized definitions have been developed for this progression, with changes defined as indefinite for dysplasia, low - grade dysplasia (LGD), and high - grade dysplasia (HGD). Unfortunately, pathologist agreement for these findings is sometimes poor, and specialized expertise is needed for interpretation.

Debate continues as to the cost - effectiveness of screening for dysplasia in patients with Barrett's esophagus. Some studies have demonstrated cost effectiveness. Critics, however, note that a significant number of patients diagnosed with esophageal adenocarcinoma have no symptoms of GERD.

In general, esophagectomy is recommended for patients with high - grade dysplasia in Barrett's esophagus. Several factors complicate this decision, including (1) lack of agreement between pathologists on the diagnosis of HGD; (2) variability in sampling of the Barrett's mucosa, with the potential for unsuspected adenocarcinoma in the Barrett's segment; (3) uncertainty in the rate of progression from HGD to carcinoma. Alternatives to esophagectomy in those with significant medical illnesses include photodynamic therapy, laser ablation (KTP or Nd:YAG), endoscopic mucosectomy, multipolar electrocoagulation, argon plasma coagulation, and most recently, cryotherapy.

Research is ongoing to determine predictors of development and progression of dysplasia and cancer in Barrett's esophagus. An understanding of cellular and genetic events underlying these events is developing. Current approaches include the evaluation of alterations in single genes (activation of oncogenes, suppression of tumor suppressor genes), alterations in the cellular DNA content (aneuploidy), and alterations in expression of multiple genes (genomics). Genes of particular interest include the tumor suppressor genes p53 and p16. Techniques for studying gene alterations include immunohistochemistry, loss of heterozygosity (LOH), gene sequencing for mutations, and methylation - specific polymerase chain reaction (PCR). Few clinical studies have demonstrated the usefulness of any of these alterations as useful biomarkers in Barrett's esophagus. Aneuploidy increases the risk of progression to carcinoma in patients with no dysplasia or LGD, but not HGD. LOH of 17p, the chromosome containing p53, does predict increased risk of cancer in patients with HGD. These studies are hampered by the limited number of patients with dysplasia in Barrett's esophagus and the low incidence of dysplasia or cancer in patients enrolled in surveillance programs.

Immortalization and characterization of human esophageal epithelial cells from surgically resected specimens.

Tsao SW

Dept. of Anatomy; Cancer Center; Faculty of Medicine,

University of Hong Kong, Hong Kong SAR China.

Esophageal carcinoma is a common disease in China. Establishment and characterization of immortalized and non – malignant esophageal epithelial cell lines from from local Chinese subjects will provide valuable experimental tools for investigation of esophageal carcinogenesis in this geographical region. Immortalization is an early and pre – requisite event involved in carcinogenesis. Molecular events involved in immortalization may represent early steps involved in the transformation of normal cells into cancer cells. Our laboratory has been involved in immortalization of human esophageal epithelial cells. We have been using the explantation method to generate primary epithelial culture from non – malignant esophageal epithelium resected from esophageal cancer patients. This simple culture method has reliably generate monolayer of esophageal epithelium with high purity. The esophageal epithelial cells could be propagated in culture to generate homogeneous population for experimentation. They could also be stored in liquid nitrogen and revived for future use. Similar to other human epithelial cells, the esophageal epithelial cells will eventually undergo senescence after limited number of passage. Introduction of genetic elements including telomerase, E6/E7 viral genes of HPV16 and Id1 could extend their life span and faciliate their immortalization. We have generated a panel of immortalized esophageal cell lines generated by single or combination of different genetic elements. The combination of human telomerase (hTert) and HPV16E6/E7 was higly efficient in immortalization of human esophageal epithelial cells (100%). Immortalization of esophagel epithelial cells by telomerase alone could also be achieved but at a lower success rate (about 20%). Introduction of Id1 alone could extend the life span of esophageal epithelial cells but insufficient to immortalize the esophageal epithelial cells on its own. Expression of Id1 also accelerated progression of immortalized esophageal cells into cell cycle. Esophageal epithelial cells immortalized by various genetic combinations reveal distinct gene expression properties. Interestingly, in multiple cell line immortalized by telomerase and E6E7 of HPV16, we consistently observed upregulation of Aurora A, which is an important kinase involved in mitotic checkpoint. The overespression of Aurora A in the immortalized esophageal epithelial cells is related to the amplification of the gene locus of Aurora A at chromosome 20q. Aurora A overexpression is known to induce mitotic checkpoint defects resulting in genomic instability and may account for some of the chromosomal aberration observed in our immortalized esophageal cell lines. In addition, we observed that COX – 2 expression was elevated at the early stage of immortalization of esophageal epithelial cells. Inhibition of COX – 2 by siRNA silencing induced apoptosis of the immortalized esophageal epithelial cells suggesting that COX – 2 expression plays a functional role in facilitating the immortalization of esophageal epithelial cells. Both Aurora A and COX – 2 are frequently overexpressed in esophageal cancer cells. The observation that they are observed in immortalized esophageal epithelial cells suggests that they are early events involved in esophageal carcinogenesis.

Functional identification of candidate tumor suppressor genes in esophageal cancer

Maria Li Lung

Department of Biology, Hong Kong University of Science & Technology

Objectives: Although esophageal cancer (EC) is ranked amongst the top causes of cancer deaths worldwide and has the highest incidence amongst the Chinese, its molecular genetic basis is still not clearly elucidated. High allelic loss has been detected on many regions of multiple chromosomes in EC tumors. Because of the difficulty of deciphering these complex and extensive molecular alterations, a functional approach is required to identify and better define key critical regions associated with tumor suppression. Using a microcell – mediated chromosome transfer (MMCT) approach in which an intact or truncated human chromosome is transferred specifically into a recipient cancer cell line, we have investigated the ability of several chromosomes to functionally complement EC tumor formation in a nude mouse model. The objectives of these studies are to identify critical regions associated with tumorigenicity and to discover the key candidate tumor suppressor genes (TSGs) involved in EC tumorigenesis.

Methods: We have utilized the SLMT1 EC cell line, which was established from a Hong Kong EC patient (Tang et al, 2001), for these studies. This cell line is 100% tumorigenic in nude mice. It was subcloned and tested for its uniformity in tumorigenic potential. A selected subclone was utilized as the recipient EC cell line for MMCT studies. The microcell hybrid (MCH) cell lines established were analyzed by microsatellite typing and fluorescence in situ hybridization (FISH) techniques to verify the successful transfer of the chromosome of interest. These hybrid cell lines were subsequently injected into nude mice and their tumorigenic potential was monitored weekly. Tumor segregants that arose in the mice after a long latency period were excised and tumor segregant cell lines were established for further molecular and cytogenetic analyses. Comparative analysis of the MCH cell lines and their tumor segregant derivatives was used to narrow down the critical regions associated with tumor suppression. Candidate TSGs were identified using genome resource databases and by expression profiling of panels of tumorigenic recipient and tumor segregant cell lines as compared to tumor – suppressive MCH cell lines. We utilized a 19k oligonucleotide array provided by our collaborator (E Liu) at the Genome Institute of Singapore. Results and Conclusions: Transfer of a 9p21.3 microdeleted human chromosome 9 into the recipient SLMT1 cell line resulted in tumor suppression, indicating that TSGs other than p16, which maps to the microdeleted region, are involved in EC tumor formation. The critical region associated with tumor suppression was mapped to a 2.4 Mb region at 9q33 – q34 (Yang et al, 2005). A high range of allelic loss was detected in this critical region in primary EC tumor tissues.

The DEC1 gene, which maps to this critical region, was analyzed in more detail. It was previously isolated and cloned by Nakamura's group (Nishiwaki et al, 2000). DEC1 expression is reduced or absent in EC cell lines. DEC1 transfection into two EC cell lines did not significantly reduce colony formation in vitro. However, injection of stable DEC1 transfectants into nude mice showed a statistically significant reduction in tumorigenic potential compared to the vector – alone controls, indicating DEC1 is a potential candidate TSG involved in EC. Expression profiling of the stable DEC1 transfectants versus the vector –

alone transfectants identified several candidate genes, which were differentially expressed. Results of these studies will be presented.

Another gene of interest mapping to the 9q34 critical region for EC is ENG, which encodes a glycoprotein that is predominantly expressed on vascular endothelial and hematopoietic cells (Fernandez – Ruiz et al, 1993) and may be involved in adhesion and invasion. RT – PCR and qRT – PCR analysis of this gene in a panel of EC cell lines shows its frequent down – regulation. Transfection studies indicate its in vitro growth suppressive effects. Thus, it is an interesting candidate EC gene, which is now under study in my laboratory.

Chromosome 14 transfer into the recipient SLMT1 cell line induced significant tumor suppression in nude mice. Microsatellite typing and BAC FISH of MCH and tumor segregant cell lines indicated there were at least two critical regions associated with tumorigenicity of 680 Mb mapping to 14q32.13 and 2.2 Mb at 14q32.33 (Ko et al, 2005). Several candidate genes mapping to these critical regions and others identified after microarray analysis of tumorigenic versus non – tumorigenic cell lines were identified. Their differential expression levels in EC cell lines and tumor tissues were verified by RT – PCR. Results of these studies will be presented.

Pretreatment surgical staging, trimodality therapy and molecular markers as predictors for survival in esophageal cancer

Mark Krasna[1] Bruce Greenwald[1] Xiaolong Jiao[2] Yousheng Mao[3]

1. University of Maryland Medical System

2. Hubei Cancer Hospital Wuhan

3. Chicams – Chinese Cancer Institute

OBJECTIVE: This report describes the role of pretreatment surgical staging, trimodality therapy and p53 protein expression in esophageal cancer and the correlation with response and survival after chemoradiation.

METHODS: Pretreatment thoracoscopic/laparoscopic LN staging (Ts/Ls LN) was performed before treatment. Post resection and post chemoradiation esophagectomy specimens were analyzed for p53 expression. The slides were stained by automatic P53 immunohistochemical staining technique.

RESULTS:

P53 protein was expressed in 84.0 % (21/25) of EGD biopsies. 71.4% (10/14) of the patients with LN metastasis by H/E staining in Ts/Ls LN biopsy were p53 (+). 14.2% (3/21) of the patients with H/E (–) Ts/Ls LN were p53 (+).

18 patients post chemoradiation were both H/E (+) and p53 (+) in the EGD specimen. After chemoradiation, 11 of these remained H/E (+) and P53 (+), (38.8%) of these patients had a pathological complete response(pCR). The median survival of this group is 15 months and 3yr survival is 10.0%. Of 4 patients with pretreatment H/E (+) and p53 (–) EGD, 1 patients was still HE (+); all of these pts were still p53 (–) after chemoradiation. 3(75%) of these patients had a pCR. The median survival is 30 months and 3yr survival is 100%.

In 13 patients with p53 (+) in Ts/Ls LN, only 23.1% (3/13) had pCR after chemoradiation. The median survival in this group is 16 months with 3yr survival of 14.3%. 18 patients were p53 (–) in Ts/Ls LN, (50.0%) of these patients had pCR after chemoradiation. The median cause specific survival of those pts is 31.5 months and 3yr survival is 50%.

CONCLUSIONS: p53 had a high rate of expression in EGD biopsy. Chemoradiation does not seem to change the p53 expression in the residual tumor. P53 expression in pretreament EGD and LN biopsy may be a useful predictor for effect of chemoradiation and survival. p53 in HE – Ts/Ls LN biopsies should be further investigated as an indicator of occult metastases.

INTRODUCTION:

Esophageal cancer is the fastest growing malignancy in the United States with 10,000 to 11,000 deaths per year. The results of single modality treatment using surgery, chemotherapy or radiotherapy alone have been poor because of a high rate of local recurrence and distant metastasis. This is probably due to the prevalence of advanced esophageal cancer at the time of diagnosis. Even though a small number of esophageal cancer patients survive longer than 5 years after initial surgical treatment, over 60% of patients die of