
A Tabulated Summary of the FDG PET Literature

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This special supplement to *The Journal of Nuclear Medicine* is a detailed tabulation of literature on FDG PET in oncology (1993–June 2000), cardiology (1986–June 2000), and neurology (1980–June 2000). This document is a subset of the original document formally submitted to the Health Care Financing Administration (HCFA) in July 2000 to request expanded Medicare reimbursement for FDG PET. It has been improved by eliminating any errors in tabulation and further clarified as the result of comments from an independent review of the original HCFA submission. This document also differs from the original HCFA submission in that it does not include some background sections and lacks references that were identified but not used because of specific inclusion and exclusion criteria.

The goal of this document is to provide a summary of all FDG PET literature for the specified periods, with tabulated values of sensitivity, specificity, percentage in management changes, etc. This document is not intended to be a formal meta-analysis or cost-effectiveness analysis of the available literature. Instead, it is meant to provide an overview of the available literature, so that future detailed studies can use this document as a starting point.

Because of the difficulty of searching all FDG PET literature, this document inevitably does not include some research articles and abstracts that may be useful. The authors have tried to make the search as comprehensive as possible, but some literature may have been overlooked. Details of the literature search strategy are provided in Appendix A. Although a formal meta-analysis is not performed, a simple weighted averaging of data using various strategies is presented (Appendix B). This weighted aver-

aging is meant to give only a general indication of the overall accuracy values and, therefore, should be interpreted with care. A data pooling analysis is also included.

The document is organized to show, first, how the tabulated data should be interpreted. This is followed by oncologic, cardiac, and neurologic application sections that provide, for each disease process, a disease background section, a case example illustrating the clinical implementation of FDG PET, an explanation of why FDG PET helped, a key management issues section (see also Maisey et al. (285)), and a summary of evidence for FDG PET with management change data for the disease and references to the relevant full literature search (in tabulated form) for the accuracy of FDG PET in specific applications. The numbers of patient studies utilized in calculating summary management changes are displayed in Tables 20 and 24 along with management figures and are embedded (without display) within the individual spreadsheets as selected from the data lines that report management change information. In addition, a summary of results from the literature search on FDG PET in all cancers is provided, as well as a summary of FDG PET literature searched for the oncologic, cardiac, and neurologic applications. A full reference list is also provided at the end. Appendix A gives details on the way in which literature was searched and analyzed, and Appendix B reports some alternate approaches to summarizing the data.

The average FDG PET sensitivity and specificity across all oncology applications are estimated at 84% (based on 18,402 patient studies) and 88% (based on 14,264 patient studies), respectively. The average management change across all applications is estimated to be 30% (based on 5,062 patients). These data were obtained combining 419 total articles and abstracts on studies in which FDG PET was used. Various methods of analysis were applied to these data (Table 25), which revealed only a small amount of variation in the ratio values. Specifically, the sensitivity of PET ranged from 84%–87%, the specificity ranged from 88%–93%, and the accuracy ranged from 87%–90%.

At the time of submission of this work, HCFA had just announced expanded coverage for FDG PET to include imaging for various aspects of lung, colorectal, esophageal,

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and head and neck cancers and melanoma and lymphoma. In addition, coverage for seizure work-ups and myocardial viability was approved. We are confident that with continued acquisition of data from well-designed clinical studies, true broad coverage for FDG PET can soon be a reality. We hope that readers of the journal will find this to be a valuable resource in better understanding the existing diversity of literature available for FDG PET.

INTERPRETING SPREADSHEETS IN THIS DOCUMENT

This report contains spreadsheets summarizing all FDG PET literature. A spreadsheet is provided for each disease under consideration, along with summary spreadsheets (see Table 1, lung cancer, as an example). On each specific disease spreadsheet, the name of the disease appears in the upper left-hand corner. The data is broken down into applications of FDG PET for diagnosis, staging, diagnosis and staging, recurrence, monitoring response, and other applications. Some diseases include a mixture of these applications and, therefore, have multiple listings in several categories.

For each disease, the first author and year of publication of the article or abstract are listed in the far left column. The second column designates "A" for abstract or "RA" for research article. The third column lists the purpose of the study. The fourth column lists the total number of patients who were included initially in the study. The fifth column lists the total number of patient studies actually implemented and upon which results data were calculated (sometimes less than the total in the fourth column, because of patient drop out or other causes, and sometimes greater than the total in the fourth column, because multiple PET scans may have been counted). In some applications in which lesions were counted, a column is also listed for the number of lesions studied. Studies using nondedicated PET are indicated. Several additional columns show the percentages for sensitivity, specificity, predictive value, and accuracy for FDG PET and CT. The gold standard used for verifying FDG PET results is also listed in a separate column. If percent management changes were available, they are listed in the last column. Finally, beneath each table are footnotes highlighting details from specific studies to further clarify how the study populations were either composed or counted. Abbreviations used throughout the tables are listed alphabetically in Appendix C.

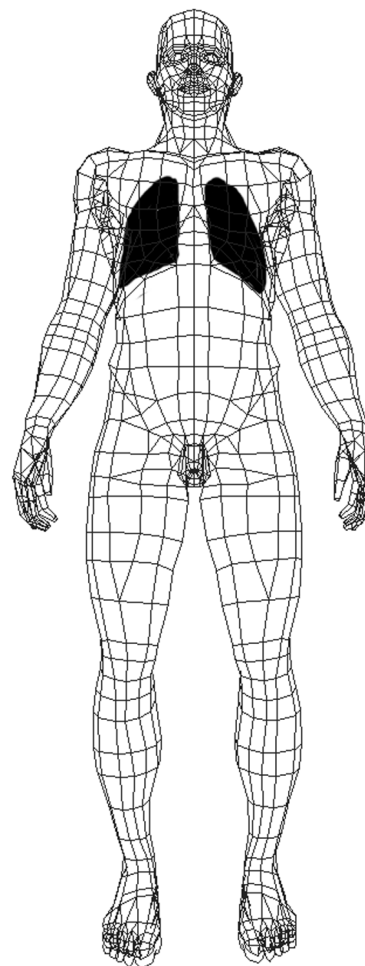
A summary for each application is provided in bold. The bolded summary totals for number of patients, number of patient studies, and number of lesions reflect the totals retrieved from the literature for all studies listing data that is inclusive of that application. Each study entry is listed with complete reported data that may include data relevant to several applications. This accounts for data repeated across applications within a given spreadsheet and is discussed in Appendix A. From within each application are selected the respective study data applicable to that application (e.g., recurrence data in instances in which both staging and recurrence data may have been listed in a given entry) and

used in the weighted average formulas generating that application's summary values, which are listed in the statistical ratio columns. These selected N values for total patient studies and total lesions do not appear in the individual spreadsheets but are embedded in the formulas and appear with the application's summary values in the overall summary sheets at the end of this report. Note that a weighted average is used, which weights studies by the number of patients, so that results obtained with more patients are given more credit. If lesion-by-lesion analysis was performed, a separate value for that analysis is also listed.

All tabular matter is presented here in the form in which it was submitted to HCFA, with the exception of various corrections to tabulation errors found in certain spreadsheets and their carryover to the overall summary sheets, and the placement of table numbers according to the style of this journal.

ONCOLOGIC APPLICATIONS

Lung Cancer



Disease Background. Lung cancer is among the most frequent and most lethal of cancers striking both men and women. It is the most rapidly increasing tumor in indus-

trialized countries. Most lung cancers are caused by smoking. However, smoking is a less important factor in adenocarcinoma, the lung cancer most rapidly increasing in the United States. Lung cancer accounts for 22% of all cancers in men and 8% of all cancers in women. Five-year survival is achieved by only 13% of all lung cancer patients. Basic treatment for non-small cell lung cancer (NSCLC) is surgical, with only 20% of patients presenting as operable. Patients who are not operable receive palliative chemotherapy or radiation. Small cell lung cancer patients respond well initially to chemotherapy and radiation and generally do not undergo surgery. Their long-term prognosis is poor.

Case Example. A 62-y-old patient with known NSCLC was evaluated before planned lobectomy. The patient had no symptoms (e.g., headaches). FDG PET revealed extensive metastatic disease to the brain in addition to the primary cancer in the lung.

Why Did FDG PET Help? Because the FDG PET scan showed that the patient had much more extensive disease than previously thought (Fig. 1, arrows), lobectomy was not a management option for this patient. The patient had no symptoms related to the brain metastases, but the FDG PET whole-body survey scan caught tumor spread to the brain.

Key Management Issues.

- Diagnosing the lung mass
- Staging NSCLC
- Assessing recurrence
- Monitoring response to therapy

Summary of Evidence for FDG PET in Lung Cancer. For staging: An estimated 37% change (Table 1) was noted in management effect, based on 1,565 patient studies (Table 24).

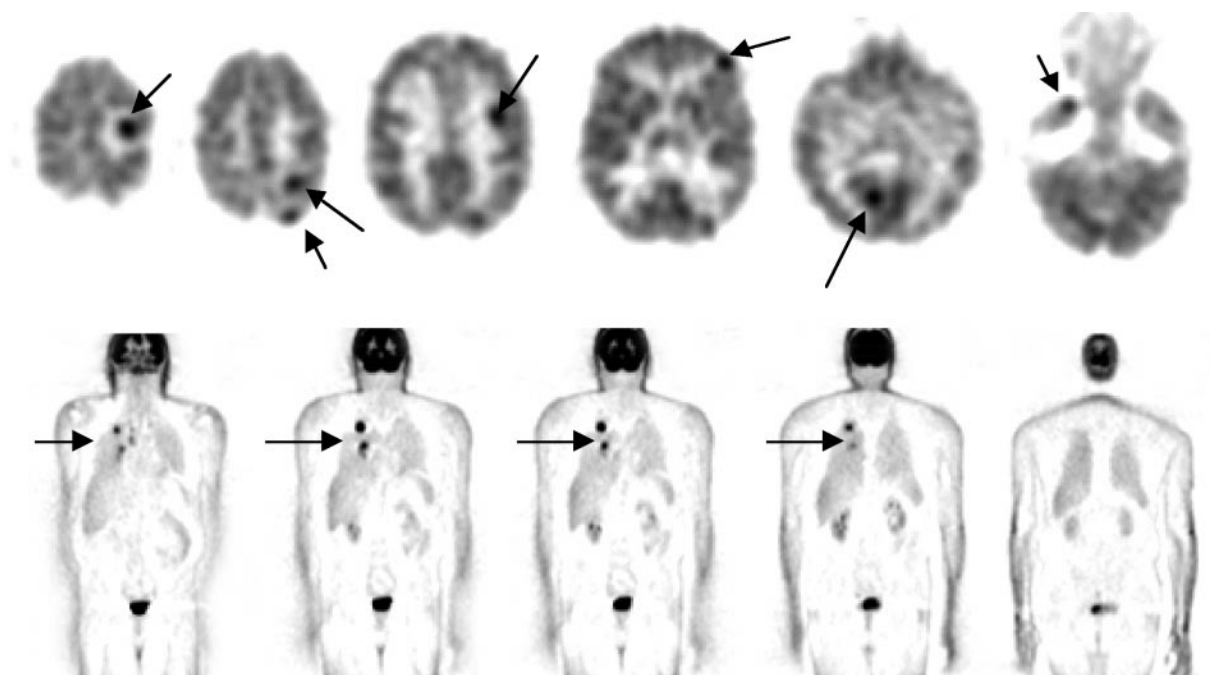


FIGURE 1. Case example, lung cancer.

TABLE 1
FDG PET in Lung Cancer: Results of Literature Search

LUNG CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT (%) Effect
							PET (%)	CT (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)		
Diagnosis																		
Menda, 2000 ¹	A	dx/lung masses	127	127			97	61									pathol	
Higashi, 2000	A	dx	63		66		83	42	87				36		76		surg/biop	
Vaylet, 1998	RA	dx/pulm nodule	17	17			93	50	93				50		88			
Orino, 1998 ²	RA	dx/eval indeterm SPN < 3cm	23	23			88	67	88				67		83		histol/biop/cytol	
Nettelbladt, 1998	RA	dx/eval tumor in lung or mediastinum mediastinal/hilar	17	17			93										histol/surg/cytol	
Shreve, 1998 ³	RA	dx/kn or susp lung nodules or masses	31	14		yes	100	50	100	80	100	50	100	80	100	71	dedPET used as ref std	
Lowe, 1998	A	eval of discrep b/w PET&other	113	25			93										multiple diagnostic tests	
Lowe, 1998	RA	eval solitary pulmonary nodules by visual analysis	89	89			98	69					89				pathol	
		using SUV data		89			92	90					91					
		for SPNs < or = 1.5 cm, visual		34			100	74					85					
		for SPNs < or = 1.5 cm, SUV data		34			80	95					88				histol/thorac	
Higashi, 1998 ⁴	RA	detect prim lung cancer < 2cm	33	33			94											
		dx/eval pulm nodule/bronchogenic ca mediastinal lymph nodes/N2	49	18	54		67	56	100	100							histopath/cytopath surg pathol CT	
Wang, 1996	A	dx lung cancer	18		19		100											
Sazon, 1996	RA	abnormal chest roentgenograms	107	107			100	52	87				100		89			
		mediastinal mets		32			100	81	100	56	100	65	100	75	100	69		
Knight, 1996 ⁶	RA	susp pulm lesion >1cm	48	45			100						100		88		pathol/clin & radiog 6 mo invasive proced	
Bury, 1996	RA	eval SPN	50	50			100	88	94				100		96		histol/clin follow-up	
Hübner, 1996	RA	differential dx chest masses & SPN SUV cutoff, 3.8	26		52		100	73										
		prim unknown lung mass		23	52		81	85									histol/clin follow-up >1yr	
Hübner, 1995 ⁷	RA	recur/proven lung ca or lymphoma	54	23			100	67										
		extrathoracic mets/susp pulm mets		13			83	80										
Duhaylonsod, 1995 ⁸	RA	detect susp prim lung cancer recurring lung cancer	100	87			87	83					92		92		biop/surg/observ>2yrs	
Duhaylonsod, 1995 ⁹	RA	dx/indeterm pulm abnorm	53	53			100										serial chest xray/CT	
Dewan, 1995	RA	dx/eval lung lesions	33		35		100	78	93				100		94		TTNA	
Dewan, 1993	RA	dx/eval indeter SPN<3 cm by xray&CT	30	30			95	80	90				89		90		histol	
Slosman, 1993	RA	detect lung ca/prior to explor thorac	36	36			94	60	94				60		89		thoracotomy	
Hoh, 1993 ¹⁰	RA	detecting prim lung carcinoma	87	6			100										histol	
Patz, 1993 ¹¹	RA	eval focal pulm abnormality on xray	51	51			89	100									biop/cytol eval sputum	
	Summary	by patients	1255	1108	278		96	67	73	68	89	91	90	58	82			
		by lesions					91	68	89	89	89	91	90	58	82			

TABLE 1 (Continued)

LUNG CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		MGMT (%) Effect	
							PET (%)	CT (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)		PET (%)
Staging																		
Hicks, 2000 ¹²	A	staging	63	63														47
Gupta, 2000	A	staging	97	77			94	98										96
Gupta, 2000	A	lymph nodes	105		295		87	91			75							82
Schiepers, 2000 ¹³	A	staging	520	184														86
Schiepers, 2000 ¹⁴	A	management	173	129														87
Seltzer, 2000 ¹⁵	A	management	536	273														44
Rakotonirina, 2000	A	preop staging/NSCLC/dedPET preop staging/NSCLC/OPET	27		84		100											44
		prim/dedPET			28	yes	73											
		prim/OPET			35	yes	96											
		mediastinal LN/dedPET			21	yes	66											
		mediastinal LN/OPET			21	yes	100											
		mets/dedPET			21	yes	52											
		mets/OPET			21	yes	92	80			85							21
Schiepers, 2000	A	dx/staging	90	67			92											
Ortuchi, 2000	A	staging mediastinal LN	36	16			100											
Crespo-Jara, 2000	A	dx of adrenal mass	19		24		89	100			100							
Mathies, 2000	A	differentiate benign/malign	35		36		95	94										
Kim, 2000 ¹⁶	A	staging lung lesions other PET	91	91		yes	100											
		nodal staging	31	31			63	74	59									
Baum, 2000 ¹⁷	A	staging nscic	63	63			76	47										
		LN mets					82	94			82							19
		mediastinal LN					100											
Wong, 2000 ¹⁸	A	staging nscic	78	78														
Albes, 1999	RA	staging/nscic/N1&2 vs N3	27	27			77	77	79	79								23
Tatsumi, 1999	RA	assessing lung lesions/DedPET lung lesions/OPET	23	23		yes	100											
		mediastinal/DedPET					96											
		mediastinal/OPET					78	79										78
		hilar/Ded PET					78	93										87
		hilar/OPET					100	84										87
Magnani, 1999	RA	mediastinal LN assessmt/nscic	28	28		yes	75	90										87
Gupta, 1999	A	staging	78	78			67	84	79	67	60	89	83	79	75			85
		LN < 1 cm			93		87	84										23
Saunders, 1999	RA	staging/primary disease	97	97			100											37
		staging/mediastinal disease			84		71	20	97	90								
Al-Sugair, 1999	A	primary/secondary mets suspicious definitive threshold definitive threshold only	314	314			67											
				314			55	97										

TABLE 1 (Continued)

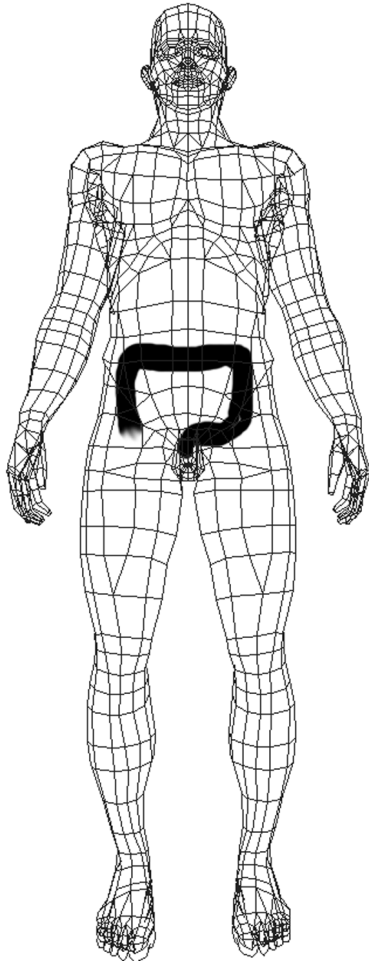
LUNG CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT (%) Effect
Staging (continued)																		
Inagaki, 1999	A	NSCLC lymph staging	24	24			22	28									surgery/pathology	
Ryu, 1999 ¹⁹	A	staging NSCLC mediastinal staging	35	35		yes	100		63								histopathological	
Marom, 1999 ²⁰	RA	after chemo mediastinal restage staging NSCLC mediastinal staging	100	100			100	100	93						83	65	pathology	23
Kutilu, 1998 ²¹	RA	pre-op staging NSCLC/concom lesions lung mets	21	100	25		92	25	93						85	58		
Nettelbladt, 1998	RA	dx/eval tumor in lung or mediastinum mediastinal/hilar	17	17			94	78	99	94	94	74	99	95	98	91	histol/clin & radiol follow-up	
Präter, 1998	RA	preop eval/pulm coin lesions	50	50	54		90	100	83	52	88		86		87		histol	
Gliem, 1998 ²²	A	eval malig in pleural disease	32	18			78										cytol/histol	
Lonneux, 1998 ²³	RA	management comparison PET/SPET overall staging/PET	42	28											86		hist/CT/bne scn	
		overall staging/SPET	28	28		yes									64			
		nodal staging/PET	28	28											62	69		
		nodal staging/SPET	28	28		yes									62			
		met staging/PET	28	28			92											
		met staging/SPET	28	28		yes	58											
Kalif, 1998 ²⁴	A	management	47	47													follow-up	85
Steiner, 1998	A	surgery mgmt/nodal & met staging	107	100											21		biopsy/CT/MRI/bne scn	21
Gupta, 1998	A	preoperative nodal staging extrathoracic mets	103	94	126		95	63	91	60					94	61	histology	35
Weber, 1998 ²⁵	RA	mediastinal staging/lung cancer	100	94													histology/radiology	14
Weber, 1998	A	mediastinal staging/lung cancer	23	17			83		82						82		histol/LN dissection	
Vansteenkiste, 1998	RA	locoreg LN staging/potent oper nscic	56		493		63	50	95	92	64	47	95	93	91	87	surg pathol	
Guhlmann, 1997	RA	staging nodes	46	46			80	50	100	75					88	59	histopathological	
Gupta, 1997 ²⁶	A	staging nodal staging	57	57			98	87			92		94		95		CT/histology	49
		nodal staging	39				96		86		90		94					
Trieu, 1997	A	staging of NSCLC	52	52			88	88	75	54					78	63	surgical pathology	38
Vansteenkiste, 1997 ²⁷	RA	mediastinal lymph staging/NSCLC pulm and lymph nodes	50	50	110		67	67	97	59					88	64	surgical pathology	10
Gupta, 1997	A	PULM <3cm PULM >3cm LN <3cm LN >3cm	57				97	83			92		94		93		histology	
		PULM <3cm	26				94		75		89		86		88			
		PULM >3cm	30				95		100		100		90		97			
		LN <3cm	42				97		89		93		93		93			
		LN >3cm	12				100		89		83		100		92			

TABLE 1 (Continued)

LUNG CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT (%) Effect
Staging (continued)																		
Hagberg, 1997 ²⁸	RA	dx/eval pulm nodule/bronchogenic ca mediastinal lymph nodes/N2	49	18	54		93	70									histopath/cytopath surg pathol	
Steinert, 1997 ²⁹	RA	mediast LN staging/NSCLC/N2 or N3	47		112		89	57	99	94	96	76	97	87	96	85	histopath/LNsmplg@thorac pathology/thoracotomy	
Bury, 1996	RA	mediastinal lymph staging/NSCLC	50	50			90	72	86	81			100		89			
Sazon, 1996	RA	abnormal chest roentgenograms mediastinal mets	107	107			100	81	100	56	100	65	100	75	100	69		
Gupta, 1996	RA	indeterminate SPN	61	61			93		88	92							histol	
Scott, 1996 ³⁰	RA	mediast LN staging/NSCLC/N2 or N3 by LN stations	27	27			100	67	100	83			100	78	100	78	biop/surg	
Sasaki, 1996 ³¹	RA	detect mediast LN mets/NSCLC	29		75		100	60	98	93	91	60		99	89	89		
Hübner, 1995 ³²	RA	prim unknown lung mass recur/proven lung ca or lymphoma extrathor mets/susp pulm mets	54	23	71		76	65	98	87	93	61	93	89	93	82	histol	
Valk, 1995	RA	staging NSCLC/mediastinal N2 dis	99	76			83	63	94	73	88	54	92	79	91	70	CT/surg/biop	
Chin, 1995 ³³	RA	mediastinal staging/NSCLC/clin sig 1	30	30			78	56	81	86	64	63	89	87	80	77	pathol ex mediast LNs	
Wahl, 1994 ³⁴	RA	staging mediastinal LN mets/nsclc	23	23			82	64	81	44				81	52	pathol		
Lewis, 1994 ³⁵	RA	preop staging/nsclc	34	34			94		80				80	90			surg	41
Scott, 1994	RA	identify lung tumor N2 lymph nodes	62	25			66		86	40			95	84			liss based dx/CT pathol	
Rege, 1993 ³⁶	RA	known pulm nodules/bronchogenic ca	16	8			100	75									histol	
Summary		by patients	4238	4005			83	64	91	74	86	67	93	86	82	68		
		by lesions			1885		83	57	92	85	77	54	94	92	90	83		
Recurrence																		
Nunez, 1999	A	recurrence NSCLC	56	11			100		67	71			100		82		pathology/follow-up	
Bury, 1999	RA	recurrence NSCLC	126	126			100	72	92	95	92	93	100	79	96	84	pathology/follow-up	
Hübner, 1995 ³⁷	RA	prim unknown lung mass recur/proven lung ca or lymphoma	54	23			100		67								histol/clin follow-up >1y	
Duhaylongsod, 1995 ³⁸	RA	extrathor mets/susp pulm mets detect susp prim lung cancer recurring lung cancer	100	87			83	80	80				92		92		biop/surg/observ>2y	
Inoue, 1995	RA	detecting recurrence	38	16			100											
Patz, 1994	RA	recurring bronchogenic carcinoma	43	43	39		97		100				89		98		pathol/clin outcome chest xray/thorac CT	
Summary		by patients	417	337			98	72	92	95	93	93	97	79	96	84		
		by lesions			39		100		62		84		100		87			

TABLE 1 (Continued)

LUNG CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT (%) Effect	
							PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)			
Monitoring Response																			
Bury, 1999	RA	recurrence NSCLC	126	126			100	72	92	95	92	93	100	79	96	84		pathology/follow-up	
Ryu, 1999 ³⁹	A	staging NSCLC	35	35			88		63									histopathological	
		mediastinal staging		35			75		91										
Bischoff, 1996 ⁴⁰	A	after chemo mediastinal restage monitoring chemo response/scic	39	39			58		93									histol	
Summary																			
			200	270			94	72	90	95	92	93	100	79	96	84			
Other																			
Crespo-Jara, 1999	A	detect bone mets	101	101			83											BS/CT/MRI/clin follow-up	
Summary																			
			101	101			83												
¹ Other analyses were performed. ² 23 pts. 16 lung cancer pts. 1 lymphoma pt. 6 benign pts. ³ Known or susp neoplasms included in pt gp. Lung data only reported here. ⁴ 33 pts w proven lung cancer. 31TP/2FN. ⁵ 49 pts. 54 pulm nodules. 18 pts staged for LN disease. ⁶ 48 pts. Gp1=27 pts. Gp2=21 pts w prior malign. SUR=2.5. Used pts=45. Gp1 acc=.81. Gp 2 acc=.95. Overall acc=.88. ⁷ 54 pts. Includes 13 recurring pts w proven lung cancer or lymphoma. ⁸ 100 pts. Includes 16 previously resected (recur). 13 pts excluded. Used pts=87. SUR>=2.5. ⁹ Used criterion of SUR 2.5 or > for malignancy. ¹⁰ 87 pts of mixed cancer types/prim and recurrent. ¹¹ Sens and spec reported for benign lesions w SUR of 2.5 or less. ¹² 11% more aggressive trmt; 13% cur to palliative; 23% no further trmt. ¹³ 23% resect to non-resect; 15% non-resect to resect. ¹⁴ 18% resect to non-resect; 26% non-resect to resect. ¹⁵ 44% major changes/14% minor. ¹⁶ Lung lesions found to be other cns as well. ¹⁷ 52%/19% cur to palliative. ¹⁸ 23% change in stage. ¹⁹ Used SUV cutoff of 3.0. ²⁰ 12% resect to non-resect; 11% non-resect to resect. ²¹ 21 pts. 25 concomitant lesions found on CT (26-1 excluded). ²² 12 lung/2 brst/2 pleura. carcinoma/1 HD/1 unk prim.																			



Disease Background. The colon and the rectum are parts of the large intestine and are responsible for absorption of various substances not absorbed by the small intestine. In western industrialized countries, colorectal cancer is the second most common cause of death from cancer. However, 20-fold variations in international incidence rates have been noted, with the highest rates found in Connecticut in the United States. Primary treatment is surgical, leading to a 50% 5-y survival rate. Adjuvant chemotherapy (chemotherapy before removal of the tumor) is now more commonly used. Radiation is sometimes used for rectal carcinoma and less often for colon cancer. Approximately 20% of patients with recurring cancers are eligible for further resection, with half relapsing early because of previously unidentified metastatic sites. Imaging helps to determine the spread (or lack thereof) of the primary tumor in the colon or rectum.

Case Example. A patient with carcinoma of the rectum was treated with surgery and radiotherapy. One year later, results of a blood test indicated rising carcinoembryonic antigen (CEA) levels. A CT scan did not reveal the site of tumor recurrence. An FDG PET study showed a liver focus (Fig. 2, arrows), which was proven by biopsy to be recurrent rectal cancer.

Why Did FDG PET Help? The liver metastasis was identified as the likely source of this patient's rising CEA blood marker. No other source was apparent. The patient, therefore, could be managed with this information. For patients with isolated liver recurrence, surgery for removal of a part of the liver is usually a good option.

Key Management Issues.

- Evaluating suspected recurrence and restaging
- Assessing response to treatment
- Evaluating liver lesions for metastatic disease

Summary of Evidence for FDG PET in Colorectal Cancer. For staging: An estimated 36% change was noted in management effect, based on 236 patient studies (Table 2).

For recurrence: An estimated 32% change was noted in management effect, based on 915 patient studies (Table 2).

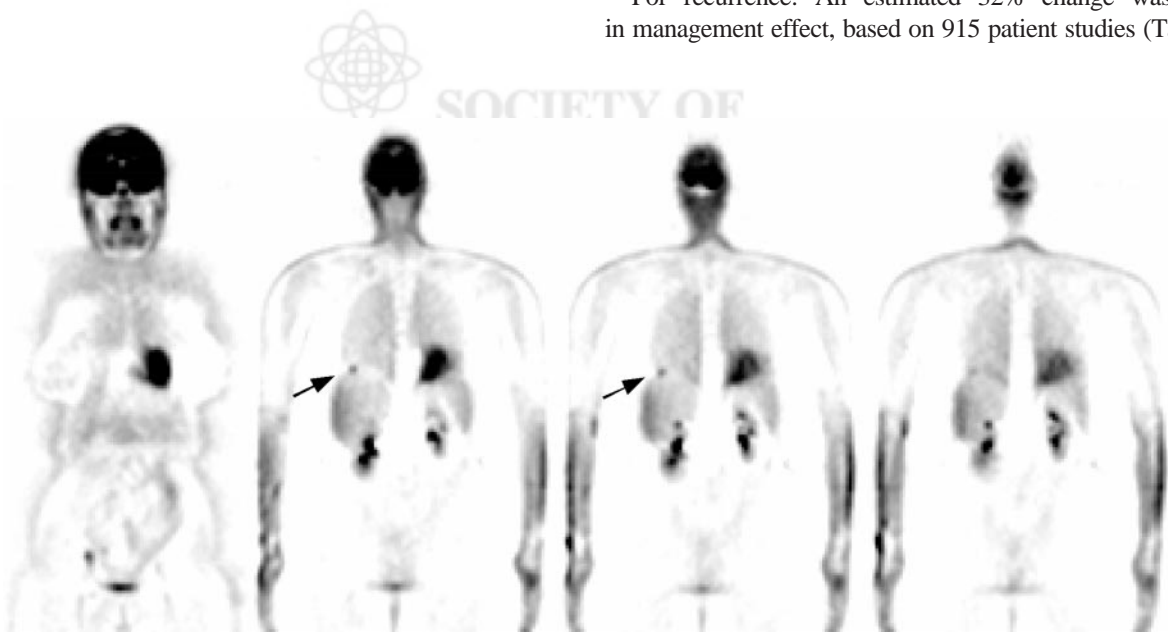


FIGURE 2. Case example, colorectal cancer.

TABLE 2
FDG PET in Colorectal Cancer: Results of Literature Search

COLORECTAL CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT	
							PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)			PET (%)
Diagnosis																			
No Articles																			
Staging																			
Amthauer, 2000	A	management	49	49													biop/surg/follow-up	42	
Oyen, 2000	A	management	48	48													histopath/follow-up	15	
Seltzer, 2000 ¹	A	management	53	53													follow-up	42	
Meta, 2000 ²	A	management	51	51													clin follow-up	40	
Baehre, 2000	A	dual head coincidence	18		24	yes	96										immunoscintigraphy		
Beets, 1994	RA	management	35	35													histo/serial radiol follow-up	40	
Summary			254	236														36	
		by lesions			24		96												
Dx/Staging																			
Abdel-Nabi, 1998 ³	RA	dx prim staging LN mets	48	44			100	43	90	100	91						CT/surg/histopath		
				14			29												
				33			88	100	97	100	50	97	86	98	81				
		staging liver mets		43															
Summary		by patients	48	134			85	34	71	92	95	50	99	86	94	81			
Recurrence																			
Whiteford, 2000 ⁴	RA	susp met or recur colorectal adenocarc overall	105	105			87	66	68	59							histopath/clin follow-up	26	
		detecting mucinous cancer		16			58												
		detecting nonmucinous cancer		93			92												
		locoregional recurrence		70			90	71											
		hepatic metastasis		101			89	71											
		extrahepatic metastases		101			94	67											
Zhuang, 2000	A	hepatic	72	72			100	76									surg/clin follow-up		
Lang, 2000	A	whole body/overall	156	156			88	80									CT/MRI	24	
		whole body/local recurrence					73	61											
		whole body/distant mets					93	92											
Baehre, 2000	A	dual head coincidence	18		24	yes	96										immunoscintigraphy		
Montravers, 2000 ⁵	A	dx/recurr	53		85	yes											post surg histol	71	
Schirrmeyer, 2000 ⁶	A	recurr/mgmt	100	100			98	91	90	72							histopath/clin follow-up	61	
Peterson, 2000 ⁷	A	resid/recurr/post local ablation to liver mets	7		9		89	44									serial CT/CEA/biopsy		
Gamez, 2000 ⁸	A	whole body	18	18			100										histol/clin follow-up		

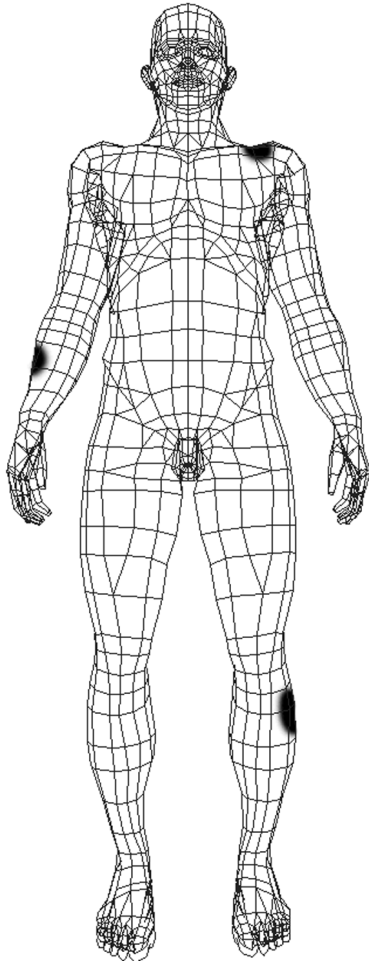
TABLE 2 (Continued)

COLORECTAL CANCER	ARTICLE TYPE (cont.)	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD. STD	MGMT (% EFFECT)
Recurrence Imdahl, 2000 ⁹	RA	detect local recurrence	71	77			92	87	87	76	96		93				histol/surg	23
		detect hepatic mets		68			100		98	96	81		100					
		detect pulmonary mets		77					91	83			93					
		detect colorectal recurrence		68			87		100	98			93					
Takeuchi, 1999 ¹⁰	RA	eval for local pelvic recurrence	23	21	25		94	100	100	100	100	100	100	100	100	100	CT/MRI/radiol/histol/biop	
Flamen, 1999	RA	staging recur colorectal adenocarcinoma/potential resect CDM inconclusive	103	60													histopath/clin follow-up 1yr	20
		whole body	115	115			95	78	79	97	69		93				histol/ser CT/clin follow-up	32
		local/pelvic			115		97		96									
		hepatic			115		95		100									
		whole body			691		93	69	99	96								
Yasuda, 1998	RA	liver mets/colorectal cancer	8		11		82	73									USCT	
Maldonado, 1998	A	recurrence	18	18													not stated	50
Flamen, 1998 ¹²	A	hepatic	103	48			96	90	100	96							histol/clin follow-up > 1yr	22
Flamen, 1998 ¹³		local/pelvic		37			81	59	100	95								
		retroperitoneal lymph node involvement		11			73	73	97	97								
		extra-abdominal		14			100											
Flanagan, 1998 ¹⁴	RA	whole body	22	22			100		71	89	100		91				pathol/LT/radiol&clin follow-up	27
Keogan, 1997	RA	rectal recurrence	18	18			92		80	92	80		89				surg/biop/follow-up	
Delbeke, 1997 ¹⁵	RA	whole body	61	61			98	83	83	98	83		97				pathol/clin&radiol follow-up	28
		hepatic			127		91	81	96	99	71		92					
		extrahepatic			96		100		40	92	100		78					
		extrahepatic			39		100		40	92	100		78					
		extrahepatic			35		74		50	92	20		71					
Ruhlmann, 1997 ¹⁶	RA	whole body	59	59			100		67	92	100						histol/clin follow-up	
Ogunbiyi, 1997 ¹⁷	RA	local/pelvis	58	47			91	52	100	80							surg/histol/clin crse/autop	43
		hepatic		58			95	74	100	85								
Keogan, 1997	RA	local/pelvic	18	18			92		80	92							surg/biop/clin follow-up	
Vitola, 1996 ¹⁸	RA	whole body	24	24			95	80	80	95	80		92				surg/biop/clin follow-up	25
		hepatic			55		90	100	100	100	80		93					
		hepatic			33		86		58	78	70		76					
Lai, 1996 ¹⁹	RA	hepatic	34	34			93	100	57	14							surg/biop/serial CT	29
Schiepers, 1995 ²⁰	RA	hepatic	76	83			94		100	100			98				surg/biop/clin follow-up	
		local/pelvic		80			94		97				95					
		local/pelvic		83			94		97				95					
		local/pelvic		74			80		50				75				pathol/CT	
Bohdlewicz, 1995 ²¹	RA	detecting recurrence	12	12			80		50	89	33		75				histol/ser radiol follow-up	40
Beets, 1994 ²²	RA	management	35	35														
Summary		by patients	1387	2244	1460		94	79	87	73	93	88	94	80				
		by lesions					93	71	96	89	98	89	74	41	87	67		

TABLE 2 (Continued)

COLORECTAL CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS. PET (%)	SENS. CT (%)	SPEC. PET (%)	SPEC. CT (%)	PPV. PET (%)	PPV. CT (%)	NPV. PET (%)	NPV. CT (%)	ACC. PET (%)	ACC. CT (%)	GOLD. STD	MGMT EFFECT	
Monitoring Response																			
Bender, 1999 ²³	RA	detect non-resect liver mets rsponse to single hi-dose chemo	10		11		100								100		follow-up 6 mos		
Findlay, 1996 ²⁴	RA	color liver mets/rsponse to 1st mo chemo	20		23		100		90								compar w tumor dimens CT		
Other	Summary	by patients by lesions	30		45		100		90						100				
No articles																			
<p>1 Major mgmt chgs occurred in 42% pts w cancer other than lung (44%). 2 40% overall (43% clinstge/37% upstge/6% downstge/49% m/mgmt/22% min/mgmt/27% no chge/79% m/ajupstg/16% minupstg/5% noneupstg/67% m/sj/dwn/33% min/dwn/18% avd/maj/surg or rad/22.2% per/surg or rad. 3 CT spec liver mets reported and listed as .97. It cats to be 32/35=.91. 4 109 PET scans/105 pls/101 CT scans. 5 2x2 does not add to N=85 sites. 6 61% influence on therapy/14% addit into fm PET over conv/47% PET clarid results conv. 7 PET may identify residual disease or early recurrence after ablative therapy including those pls w neg CT scans. 8 FDG-PET has superior accuracy than MET-PET&S. Sens implied as 100% in abst. Rpts # of pls having ea lesion type but does not explicitly say tot # of lesions. 9 71 pls/77 PET investigations/68 CT scans. Chge-in-mgmt in 16/71 pls=22.5%. 9 71 pls/77 PET investigations/68 CT scans. 9 71 pls/77 PET investigations/21 CT scans. 10 Sens based on 25 lesions from 23 colorectal cancer pls. An additional 6 prim lesions from rectal cancer pls were also included in study. Acc value based on DAR=2.8. 11 25/78 preop pls upstaged away from surg. 12 This is % of correct chge in mgmt. 5 corr dwnstged+11 corr upstgd+5 corr for relapse+2 corr for excluding disease. Were also 2 incorr oversgd+5 incorr understgd. 13 The total pls used for the 4 subgs=110 where tot pls in study =103. Some subgs overlap in pls(e.g. pt having pelvic and retroper simulit). 14 4of15 guided to curative surg; 11 guided away from surg. 15 6 directed to surg/11 avoid unnecess surg. PET:54TP/1FP/1FN/5TN. 15 PET:95TP/1FP/9FN/22TN. 15 CT:66TP/6FP/15FN/9TN. 15 PET:34TP/3FP/2TN. 15 CT:23TP/2FP/8FN/2TN. 16 At reports PET wd have had a definitive impact on pt mgmt in at least 6 pls. 6/59=10% theor chge in mgmt. 17 Tot used pls exceeds tot pls in study as diff #'s apply to diff subgs. 18 Altered surgical plans in 6/24 pls. PET:18TP/1FP/1FN/4TN. 19 PET:35TP/4FN/16TN. 19 CT:18TP/5FP/3FN/7TN. 19 10/34 pls were influenced in clinical mgmt. Seems that 2FP reported in art are actually 2FN (for hepatic lesions by PET - 25/27 correct). 20 76 pls/93 studies. Values calculated based on 83 studies for both pelvic and liver results. Liver PET:33TP/2FN/46TN. 20 Pelvic PET:45TP/1FP/3FN/34TN. 21 12 pls. 10 pls w colorectal carcinoma. 2 pls w ovarian carcinoma. 22 Chge in mgmt=14/35=.40 23 10 pls. 11 mets. 6 responders w signif FDG decr. 5 non-responders(2 s.t. w sl FDG decr/3 progress w enhanced FDG.) 24 Values obtained using a 15% decrease in pre-irfirt T.L ratio by 4-5 wks in compar w tumor rsponse.</p>																			

Melanoma



Disease Background. With an increasing mortality rate second only to that of lung cancer, malignant melanoma is the most rapidly increasing cancer in white populations, with incidence

increasing at $>5\%/y$ since 1973. The most common cancer striking young women between ages 25 and 29, melanoma accounts for 18% of all cancers in young adults 15–39 y old. Melanoma risk factors include pre-existing skin lesions and lighter hair color, with red-haired and blond individuals having 3 and 2 times greater risk than average, respectively. An overall increase in risk appears related to strong solar ultraviolet radiation. Approximately 20% of patients who present with nodal metastases with no distant metastases are cured by surgery. For isolated metastases to the brain and lung, surgery can improve survival. Thus FDG PET's role in identifying truly isolated metastases is central to the making of rational decisions about radical surgical removal of metastases.

Case Example. A 63-y-old patient had a melanoma removed from the skin overlying the right scapula (shoulder region). A second metastasis was excised at the nape of the neck. An FDG PET scan was ordered to stage the patient's cancer. Increased FDG metabolism was seen after surgery at the shoulder site (Fig. 3, far right, top arrow). In addition, multiple metastases were seen within the anterior mediastinum, left lung, left adrenal, left axilla, and para-aortic nodes.

Why Did FDG PET Help? The FDG PET scan showed that the melanoma had spread to various tissues and that chemotherapy would be the only option.

Key Management Issues.

- Determining the stage of thick melanoma lesions at presentation
- Assessing nodal spread from lesions of intermediate thickness
- Confirming the recurrence of disease
- Monitoring response to treatment
- Restaging before surgical removal of isolated metastases

Summary of Evidence for FDG PET in Melanoma. For staging: An estimated 26% change was noted in management effect, based on 283 patients (Table 3).

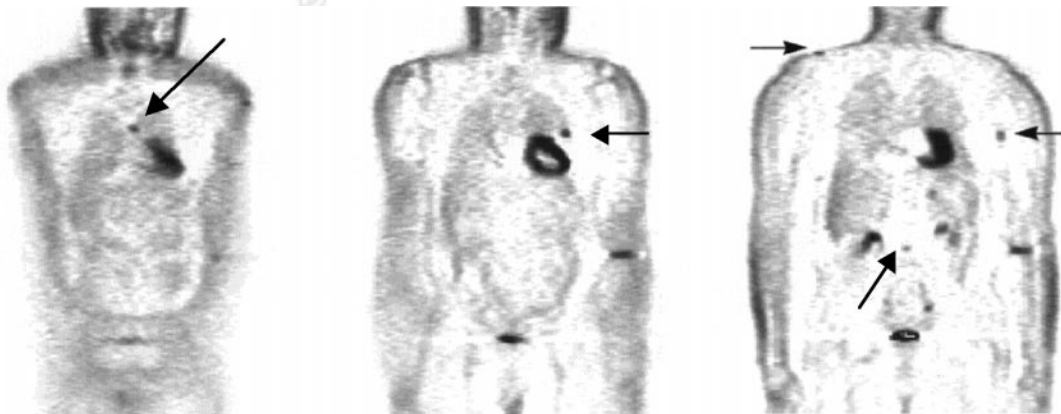


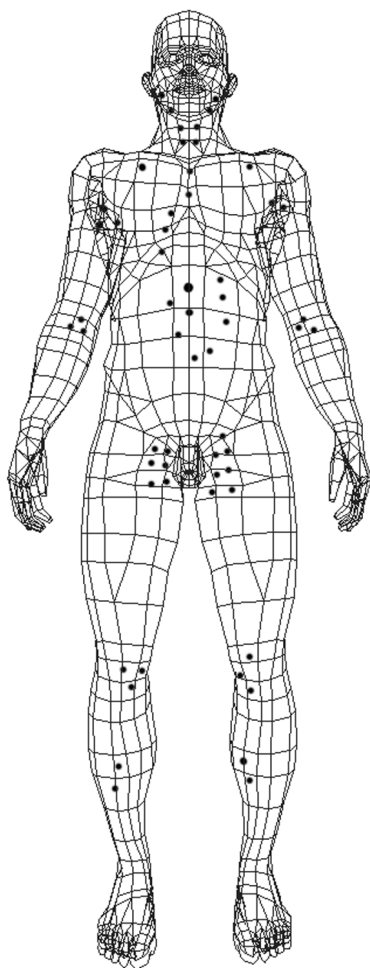
FIGURE 3. Case example, melanoma.

TABLE 3
FDG PET in Melanoma: Results of Literature Search

MELANOMA	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMTI% EFFECT
Diagnosis No Articles																	
Staging																	
Acland, 2000 ¹	RA	detect subclin mel melan/overall stage I	54	62		78	87									histol	
		stage II		35		50	87										
		stage III		9		33	100										
Jadvar, 2000 ²	RA	management/newly dx & recur	38	17		93	50										
Eightved, 2000	RA	staging malig melan/stages II & III	38	38												CT/clin follow-up 10 to 36 mos	8
		all foci		38		97	56			88		83		87		clin ex/CT/US/radiog/liver fn/histol/clin follow-up	
		all foci		7			62										
		intra-abdominal foci				100	100										
		pulmonary/intrathoracic foci				100	33										
Paquet, 2000 ³	RA	staging metastatic melanoma	24	28												clin/pathol	34
Wong, 2000	A	management	47	47												follow-up	34
Seltzer, 2000 ⁴	A	management	47	47												follow-up	34
Bohuslavizki, 2000	A	prim staging/therapy monitoring	82	51	189	72	70			93		32				histol	
Mruck, 1999 ⁵	RA	post-surg follow-up/hi-risk melan	50	51		100	92		82							CT/MRI/LN sonog/bne scint/histol/6 mo follow-up	
Wagner, 1999	RA	occult regional lymph node	70	70		17	96			50		82				SNB histol/clin follow-up 6 mos	
Nguyen, 1999	RA	staging	45	51		81	80									biop/clin crse	33
Laningham, 1999	A	staging prim & recur	25	25		96										histol/other corral data	
Chisin, 1999	A	staging	21	21		91	90									biop/PCS/US/CT/MRI	
Steinert, 1998 ⁶	RA	known met or newly dx	55	24	83	89	29			93		20		84		histol/other corral data	
Macfarlane, 1998	RA	staging	24	24		85	92									histopath	
Hsueh, 1998	RA	at 6 months follow-up	87	87		72	92			78		89				clin crse	
		at 12 months follow-up	87	87		61	94										
Holder, 1998	RA	metastatic melanoma scan	100	100		94	83									biop/cytol	
		by lesions	100	8		100											
Rinne, 1998	RA	whole-body lesions	59	59		100	94									follow-up	
		whole-body patients	52	52		100	94										
		brain	15	15		100	100										
		neck LN	25	25		100	100										
		lung	37	37		70	100										
		mediastinum/hilus	20	20		71	100										
		liver	20	20		100	100										
		abdomen	33	33		100	94										
		abdominal LN	19	19		100	100										
		peripheral LN	49	49		97	100										
		bones	17	17		100	100										

TABLE 3 (Continued)

MELANOMA	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	SENS		SPEC		PPV		NPV		ACC		GOLD. STD	MGMT/EFFECT
						CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)		
Staging	(cont.)																
Wagner, 1997	RA	regional lymph node eval	14		14	100	100									histol	
Damian, 1996	RA	staging	100	100	415	93										Xray/CT/MRI/Bne Scint	22
Blessing, 1995	RA	dx lymph nodes	83		83	74		93								histol/US	
Böni, 1995	RA	staging	39		39	91		67								Conv/biop	
Steinert, 1995	RA	dx metastatic melanoma	53		53	92		77								histol/other imag	
Gritters, 1993	RA	superficial lymph node intra-abdominal LN & visceral	13	13		100		100								biop/physical exam	
					15	100											
	Summary	by patients by lesions	1642	1327	899	83 87	88 87	91 68	75 93	70 93	85 28	91 84	80				26
Dx/Staging	No Articles																
Recurrence	No Articles																
Monitoring Response	No articles																
Other	No articles																
<p>¹ Overall 54 pts/62 scans=Used #. 34 pts/35 scans stg I. 9 pts/9 scans stg II. 16 pts/17 scans stg III. 1pt/1scn stg IV. ² 3/39=8% had chge in surg mgmt from PET w no effect to WW strat or decis to init immunotherapy. ³ 24 pts/28 assessments. ⁴ 34% major/21% minor. ⁵ 50 pts/51 studies. ⁶ Used pts=24. Values based on 83 lesions. 68TP/5FP/8FN/2TN.</p>																	



Disease Background. Both Hodgkin's disease (HD) and non-Hodgkin's lymphoma (NHL) are common malignancies that are increasing in frequency. The underlying problem in lymphoma arises from the individual's white blood cells, cells involved in helping to fight infections. Significant differences exist between HD and NHL, and these differences factor into both diagnosis and treatment. HD begins as a unifocal disease located in a single group of malignant lymph nodes and spreads via adjacent associated

lymph node groups. Limited disease is treated appropriately with radiation therapy, resulting in complete cures for a high percentage of patients. Even after recurrence, treatment still may result in permanent cure. Patients with advanced disease have a poorer prognosis and usually require chemotherapy in addition to other treatments. NHL is a multifocal disseminated disease, usually requiring combined chemotherapy, sometimes radiotherapy, and, in some instances, high-dose chemotherapy with bone marrow transplantation. In most patients the disease is ultimately fatal. However, long remission and cure can be induced effectively in high-grade tumors that would be rapidly fatal if untreated. Low-grade NHL, which has a better prognosis untreated, does not respond as well to chemotherapy and consequently can result in a worse prognosis after treatment.

Case Example. A 27-y-old man with lymphoma underwent an FDG PET study before chemotherapy in July 1999 (Fig. 4, top row). At that time, evidence of cancer was found in the right shoulder and thoracic spine (arrows). The first follow-up FDG PET scan (Fig. 4, middle row) showed nonspecific bone marrow response to chemotherapy (a common finding). The second follow-up scan (Fig. 4, bottom row) demonstrated complete remission, with the right shoulder and thoracic spine regions no longer showing increased FDG metabolism.

Why Did FDG PET Help? FDG PET showed that the chemotherapy was working and that the cancer cells were being destroyed. This helped doctors know that further treatment was not needed at that time and gave the patient a sense of relief that his condition was improving.

Key Management Issues.

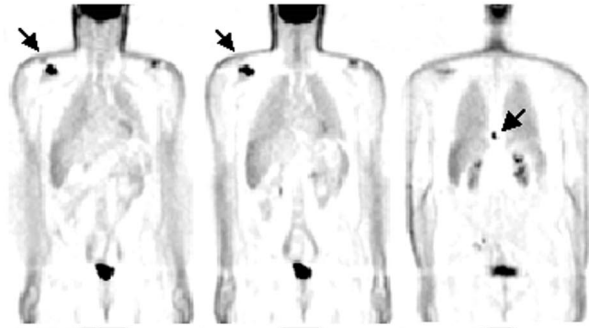
- Staging the disease before treatment
- Monitoring response to treatment
- Detecting recurrence
- Making a differential diagnosis

Summary of Evidence for FDG PET in Lymphoma. For staging: An estimated 21% change was noted in management effect, based on 407 patient studies (Table 4).

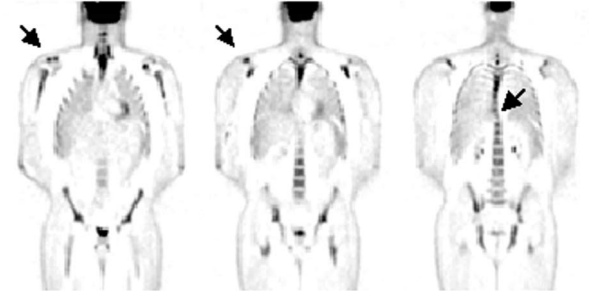
For diagnosis/staging: An estimated 5% change was noted in management effect, based on 62 patient studies (Table 4).

For recurrence: An estimated 10% change was noted in management effect, based on 158 patient studies (Table 4).

PET I
7/9/99



PET II 10/19/99
(following chemotherapy)



PET III
2/18/00

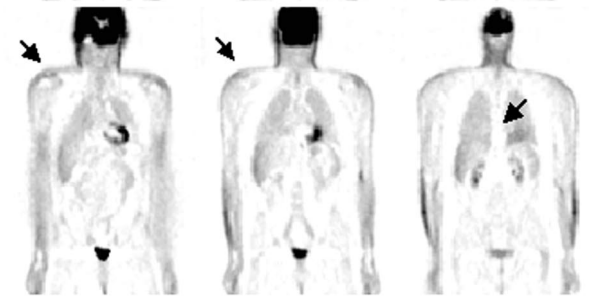


FIGURE 4. Case example, lymphoma.



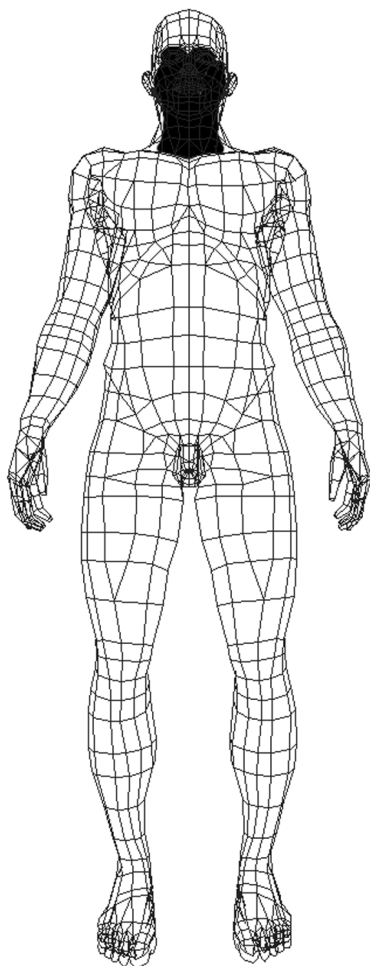
TABLE 4
FDG PET in Lymphoma: Results of Literature Search

LYMPHOMA	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC CI (%)	ACC PET (%)	ACC CT (%)	GOLD STD	_MGMT(%) EFFECT
							PET (%)	CT (%)	SENS (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)					
Diagnosis																			
Hoffman, 1993 ¹	PA	lymphoma vs normal CNS lesions/AIDS	11	11			100											CT/MRI/follow-up/biopsy	
Staging																			
Delbecq, 2000	A	staging	27	27														pathology	31
Yap, 2000	A	management	58	58														not stated	50
Kostakoglu, 2000	A	staging	86	86							67	84					biopsy		
Martinez-Lizaro, 2000	A	low-grade NHL	101	24			100	94	88	75							pathol/radiol/scint/clin follow-up		
		high/intermediate grade NHL		49			85	86									histology		
Bohuslavzki, 2000	A	HD/staging	37	37			91		69		46	96	74				follow-up	8	
Dittmann, 2000	A	management	24	24													follow-up	50	
Selzer, 2000 ²	A	management	536	40													histology	6	
Hwang, 2000	A	CoDe-PET/staging lymphoma	59	59		yes					86	86					pathology		
Delbecq, 2000	A	NHL/HD management	45	45			58										Ga67/CT		
Tatsumi, 2000	A	staging/NHL	30	30	206		87												
		other PET		30		yes	77										CT/clinical correl	5	
Kostakoglu, 2000 ³	A	staging/dx/relapse	62	62			100										GaSPECT		
Tomas, 2000	A	staging/prior/during/post ther	10	10	32	yes										95			
Israel, 2000	A	staging/base/trtm/post trtm/follow-up	35	35	123												Ga scint/CT/biop/follow-up		
Jacobson, 2000 ⁴	A	dx/staging/recurr	95	177			89		93	62	88	84					clin/imaging/histopath		
Shah, 2000 ⁵	PA	management of lymphoma	29	29		yes	100			50							biop/clin observation	34	
Lin, 2000	A	staging/lymphoma	46	46													clinical staging/CT/Ga scint		
		extranodal B-cell lymphoma/MALT type		10	110		96										ophthal/otolaryng/gastros/endos		
Hoffmann, 1999 ⁶	PA	staging/lymphoma	17	17	42	yes	98										entero/colonosc/CT/biop		
Moog, 1999	PA	Hodgkin/NHL staging	56	56												100	biop/MRI/CT/radiol studies		
Jerusalem, 1999	PA	Hodgkin/NHL evaluation	60	60													clin ex/CT/MRI/local biop	3	
Zinzani, 1999	PA	Hodgkin/NHL prognosis	44	44			100	100	96	17							clin outcome/survival		
Finke, 1999	A	MH/primary staging	93	93			89	65	99	97							biop/MRI/ US/ follow-up	8	
		NHL		93			100	73	99	96							biop/MRI/ US/follow-up		
Lin, 1999	A	staging/lymphoma	17	17		yes	100										clinical staging/CT/Ga scint		
Bangerter, 1999	PA	chest lymphoma/staging/follow-up	89	147			96		94		90	98					CT/clin follow-up exam		
Jerusalem, 1999	PA	Hodgkin/NHL restaging	54	54							100	42					outcome		
Moog, 1998 ⁷	PA	detect extranodal lymphoma spread	81	50	58		97	63	100	93							biop/clin follow-up	16	
Carr, 1998	PA	detect lymphoma in bone marrow	50	50			81		76		90	78					unil iliac crest marrow aspir/biop		
Richter, 1998 ⁸	A	HD/NHL/staging/resid/recurr/pre-ther post-trtmt	17	17			100		100								histol/clin follow-up		
		Hodgkin/staging		46			90		100								histol/clin follow-up		
Bangerter, 1998	PA	Hodgkin/staging	44	44			86										conv staging/biop	14	

TABLE 4 (Continued)

LYMPHOMA	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded. PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT	
							PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)			PET (%)
Monitoring Response																			
Jenusalem, 2000 ²⁴	RA	early eval. rsnse/polychemo 2-5 cycles/NHL	28	26			42	100	100	100	100	67						relapse/biop/clin. follow-up	
Torizuka, 2000 ⁵	RA	response to RIT/NHL	14	8			100	100	100									phys ex/CT/bne mar biop/chem	
Tomas, 2000	A	staging/prior/during/post. ther	10		32	yes												GaSPECT	
Israel, 2000 ²⁶	A	staging/base/trtm/post-trtm/follow-up	35	13		yes	100		60	43	100	100	69					CT/biop/follow-up	
Wiedmann, 1999 ²⁷	RA	HD/eval response to chemoradiotherapy	23	22					77									CT/xray/US	
Bangertter, 1999	RA	chest lymphoma/staging/follow-up	89	147			96		94	90	98							CT/clin follow-up exam	
Richter, 1998 ²⁸	A	HD/NHL/staging/residual/recrur/pre-ther post-trtm	17	17			100		100									histo/clin follow-up	
			46	46			90		100									histo/clin follow-up	
Summary																			
by patients			262	279			9.0		9.3	8.8	9.4		6.9						
by lesions					32								9.5						
Other																			
No articles																			
¹ FDG uptake signif higher both qualitative/semiquantitative compared to nonmalign lesions.																			
² 50 major/10 minor.																			
³ 5% upstaged.																			
⁴ 95 pts/177 scans.																			
⁵ 10/29 pts=34% w chge in clin mgmt. 16 CT TP/5 CT FN/3 CT TN.																			
⁶ PET did not visualize histologically verified lesions in any of 10 pts studied. Study discontinued.																			
⁷ 13/81 pts=16% w reassignment of tumor stage from verified PET results.																			
⁸ PET:16TP/1TN.																			
⁹ PET:18TP/2FN/26TN.																			
¹⁰ For CT visual image interpretations, all findings except complete remission were considered pathological.																			
¹¹ 60 pts. 740 LN regions. 185 malign/555 benign. PET TP=167/CT TP=160. PET induced stage chge in 4 pts/60 pts=7%.																			
¹² 22 pts. 14 susp/8 treated. 16 pts had PET. Detection rate = 23/25.																			
¹³ 5% upstaged.																			
¹⁴ 95 pts/177 scans.																			
¹⁵ 38 pts. 21 pts w liver tumor. 15 pts w lymphoma. 1 pt w hemangioma.																			
¹⁶ 13 of 96 received immed secondary trtm.																			
¹⁷ 5% upstaged.																			
¹⁸ 95 pts/177 scans.																			
¹⁹ 61 pts. 58 pts used. 56 pts had MRI/24 PET/22 Both.																			
²⁰ For CT visual image interpretations, all findings except complete remission were considered pathological.																			
²¹ PET:16TP/1TN.																			
²² PET:18TP/2FN/26TN.																			
²³ Cannot duplicate Table 5 results from article reporting.																			
²⁴ 42 pts studied w PET. Sens based on 114 malign lesions.																			
²⁵ 2x2 of PET+/PET- vs Relapse/CR. 5TP/0FP/7FN/4TN. 1 init CR of PET+ ultim relap/7 of 21 CR of PET- ultim relap.																			
²⁶ 2 pts of init 23 PET- died.																			
²⁷ 2x2 defined by trtm response (CR&PR) vs no response and mean SUV-lean decline vs no decline at 33-70ds. N=8 pts.																			
²⁸ 6 TP=6 responders w signif decline. 2 TN=2 non-responders w no decline.																			
²⁹ 35 pts/13pts w complete data by pts:3TP/4FP/6TN.																			
³⁰ 23pts/42 exams/22 of 42 exams for rsnse to chemoradio. All pts in CR/5 FP/17 TN on PET.																			
PR PD, suspic of relapse considered not CR.																			
²⁸ PET:16TP/1TN.																			
²⁸ PET:18TP/2FN/26TN.																			

Head and Neck Cancer



Disease Background. Cancer of the head and neck is relatively uncommon in the western world, occurring in 2%–4% of all cancers. In contrast, it comprises up to 40% of all cancers in some Asian countries. In the western cases, the majority are squamous cell tumors with a variable aggressiveness that depends on site and histological appearance. Strong environmental links have been found with tobacco and alcohol usage and with other factors, such as chemicals, fumes, and viruses. Multidisciplinary teams of head and neck surgical oncologists, radiation oncologists, imaging specialists, and medical oncologists operating in specialized centers are required for good outcomes. Treatment is directed at maintaining the form and function of the head and neck structures as well as eradicating the disease. Because of the need to limit surgery and the fact that local nodal spread is the most important prognostic factor, imaging has an important role in the management of these tumors. After treatment, conventional anatomical imaging procedures prove less useful because of the distortion of anatomy caused by treatment. Therefore, FDG PET is of particular importance in follow-up imaging of suspected recurrence.

Case Example. A patient with a right alveolar ridge carcinoma was referred for FDG PET scanning before surgery for

staging purposes. Results of a CT scan (Fig. 5A, 5C) indicated that the tumor extended superiorly into the maxillary sinus. Registered PET and CT images showed uptake of FDG within the primary site arising from the alveolar ridge (Fig. 5B) but no evidence of tumor within the sinus itself (Fig. 5D). This illustrates how the FDG PET scan can identify the extent of disease when inflammatory tissue and tumor are co-located.

Why Did FDG PET Help? FDG PET helped because it determined that the tumor was localized and did not extend into the maxillary sinus. This directly aided the surgery for tumor removal.

Key Management Issues.

- Locating the site of primary disease
- Determining the extent of primary disease
- Staging of lymph node spread
- Detecting recurrence
- Assessing response to therapy

Summary of Evidence for FDG PET in Head and Neck Cancer.

For diagnosis/staging: An estimated 33% change was noted in management effect, based on 15 patient studies (Table 5).

For recurrence: An estimated 33% change was noted in management effect, based on 15 patient studies (Table 5). Because management effect for both diagnosis/staging and recurrence is based upon the same single study of 15 patients, results should be interpreted with caution.

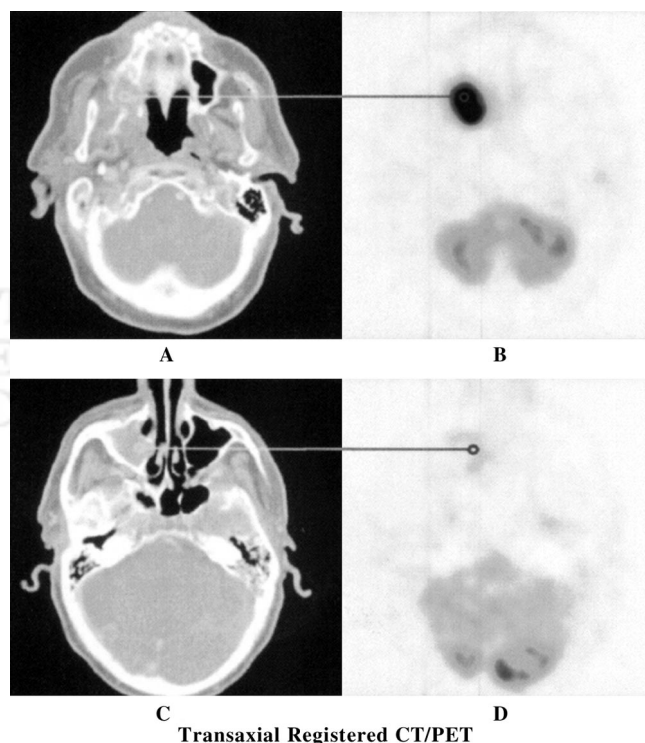


FIGURE 5. Case example, head and neck cancer. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.

TABLE 5
FDG PET in Head and Neck Cancer: Results of Literature Search

HEAD/NECK CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	SPC PET (%)	SPC CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT EFFECT (%)
Diagnosis																		
Zimny, 2000	A	dx/prim/recur/H&N prim/recur	36	36		yes	82	59	70	70					78	62	CT/MRI/US/histol/follow-up	
Beuthien-Baumann, 2000	A	LN mets/neck sides dx/oral mucosa carcinoma lymph node mets lymph node mets	39	39	68	yes	64	55	92	82					87	78	surg/histology	
Henze, 2000	A	larynx/hypopharynx	19				88	29		94			79			71	histological	
Nowak, 1999 ¹	RA	dx eval/kn or susp prim&recur/all prim recur	71		48		87	67	71	71	89	85	68	44	82	66	histol/clin follow-up	
Wong, 1997	RA	LN mets/nk sides assessmt/prim nodal disease recurring/residual disease	54	54	26		86	57	75	92	80	89	82	65	81	73		
Grünwald, 1997	RA	primary tumors/thyroid cancer	54		136		80	80	92	84	80	68	92	91	88	83	CT/MRI/histol/nk dissect	
Greven, 1994 ²	RA	detect prim	25	23	55		92			84								
	Summary	by patients by lesions	298	193	580		93	66	70	56					87	58	clin eval/histol/follow-up clin/radiographics	
Staging																		
Li, 2000	A	assessmt/recurrence	43	43			91		86						88	66	biopsy/6 mo follow-up other imaging/biops/clin follow-up	
Keyes, 2000 ³	RA	imaging thorax/H&N cancer	56	9			75		95	33							neck dissect/histol	
Lang, 2000	A	cervical lymph node staging	62		91		100	38	93	85							biop/pathol	
Lowe, 2000	RA	recur detect/ser post-thr/Stage IIIorIV	44	16			82	59	70	70					78	62	CT/MRI/US/histol/follow-up	
Zimny, 2000	A	dx/prim/recur/H&N prim/recur	36	36	68	yes	64	55	92	82					87	78		
Adams, 1998	RA	LN mets/neck sides cervical LN staging	60		1284	yes	90	82	94	85							histopathology	
Paulus, 1998	RA	initial lymph node staging local recurrence	38	25			50	40	100	100					80	76	surg/histopath	
				13			71	100	100	83					85	92		

TABLE 5 (Continued)

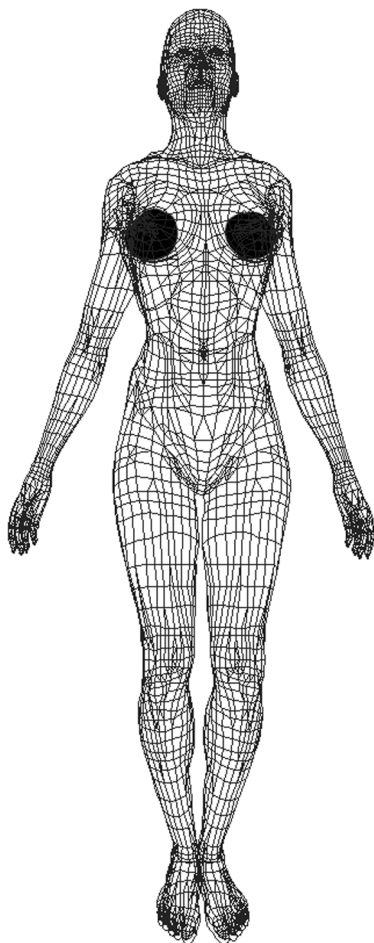
HEAD/NECK CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	Non-Ded		SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT				
						PET	CT	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)						
Staging Manolidis, 1998	RA	pre-op eval/local dis/prim&susp recurr	29		30			87	80	80									clin/radio/histopath				
		local dis/prim&susp recurr			21			94	80	80													
		local dis/recur malign only		22				71	66	66													
		local dis/squamous cell carcin only		15				92	100	100													
		regional disease/prim&recur		30		30		80	85	85													
Wong, 1997	RA	regional disease/prim&recur			20			57	77	77													
		regional disease/recr malign only		22			89	85	85														
		regional disease/recr malign only		14			82																
		regional dis/squamous cell carcin only		15			75	100	100														
		distant metastases		28		28		90	94	94													
Lowe, 1997	RA	assessmt/prim nodal disease	54	54			100																
		recurring/residual disease		16			67	67	100	25													
		post-treatment necks		12																			
Benchaou, 1996	RA	response to chemo	28	27			90	83	83														
		pre-op assessmt N-staging/H&N ca	48		468		72	67	99	97	89	74	99	95	96	93					tissue biopsies		
Braams, 1995	RA	sub-digestic LN gps			52		86	86	90	84	86	78	90	90	89	85							
		lymph nodes	12	12			91	88	88												clin/MRI/histopathology		
Zeitouni, 1994	RA	detect known tumors/newly dx&recur	7	7																			
		extracranial H&N ca/detect prim LN involvement	60	30			97																
Lindholm, 1993 ⁵	RA	recurring prim tum		34			90	100	100														
		detect known tumors/prior to ther	14	17			90															histol/cytol	
Dx/Staging	Summary	by patients	591	468			87	62	89	73	86	78	88	67									
		by lesions			2113		84	77	95	87	89	74	99	95	94	90							
Di Martino, 2000	A	pretherapeutic dx nodal spread	40	40			82	82	87	94	77	88	91	91									
		dx/prim/recr/H&N prim/recr	36	36			yes	82	59	70	70												
Pai, 1999	RA	LN mets/neck sides			68		yes	64	55	92	82												
		detect prim/pretherapy neck node mets/pretherapy	7	7			yes	71	86	100	100	100											
Cheon, 1999	RA	detect prim/posttherapy neck node mets/posttherapy	19	14			82	81	100	100	100	100	60	60	86	86							
		detect prim/posttherapy neck node mets/posttherapy		23			100	100	100	67	100	45	100	100	74								
Farber, 1999	RA	H&N/posttherapeutic recurrence <3mo post trmt	45	45			100	100	91	95	33	50	100	100	91	96							
		recurrent	18	18			97	73	88	85													
Conti, 1999	RA	susp local/ distant recur	28	28			100	67	86	71													
		residual/recurrent H&N-(primary)	30	30			100																
Fischbein, 1998	RA	residual/recurrent H&N-(primary)	44	44			100		64	65													

TABLE 5 (Continued)

HEAD/NECK CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PEI	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT
							PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)	PEI (%)	CI (%)		
Dx/Staging Paulus, 1998	RA	initial lymph node staging	38	25			50	40	100	100	80	76					surg/histopath	
Maldonado, 1998 Wong, 1995 ⁶	A RA	local recurrence tumor recurrence assess prim nodal mets	15 17	15 17			71	100	100	83	85	92					not stated	33
Lindholm, 1993	RA	possible local recurrence detect of known H&N	9	9			100				100						histol neck dissection	
Summary		by patients by lesions	360	330	179		88	69	83	85	76	88	90	91	88	73		33
Recurrence							83	78	94	85	66	60	93	93	89	84		
Haenggel, 2000 Lapela, 2000 ⁷	RA RA	detect resid tumor 3 mos post-trimt detect recurrent H&N ca	50	50	81		100		74	42	78		100		78		biop/follow-up 4 mos	
Mueller, 2000	A	pre-op dx/recur/hurthle cell ca	20		69		95		93	91	87				90		histol/cytol/biop/follow-up	
Lowe, 2000	RA	recur detect/ser. post-ther/Stage II/ortv	44	16	37		62										histol/cytol/follow-up	
Li, 2000	A	assessmt/recurrence	43	43			100	38	93	85							biop/pathol	
Zimny, 2000	A	dx/prim/recur/H&N prim/recur	36	36			91		86						88	66	biopsy/6 mo follow-up	
Pai, 1999	RA	LN mets/neck sides detect prim/pretherapy	7		68	yes	82	59	70	70					78	62	CT/MRI/US/histol/follow-up	
Stokkel, 1999	RA	neck node mets/pretherapy	19		7	yes	64	55	92	82					87	78	repeat MRI/endosc/3 mo follow-up	
Collins, 1998 ⁸	RA	detect prim/posttherapy	48	48	14		71	86		100	100				71	86		
Paulus, 1998	RA	neck node mets/posttherapy	28	37	23		82	81	100	100	60	60	60	60	86	86		
Maldonado, 1998	RA	loc relap/laryng/hypopharyng/post-rad	38	25	46	yes	100	100	100	67	100	45	100	100	74		endoscopy/biopsy	
Wong, 1997	A RA	susp recur/prim H&N initial lymph node staging local recurrence tumor recurrence	15	15			100	100	91	95	33	50	100	100	91	96		
Arzei, 1996 ⁹	RA	assessmt/prim nodal disease	54	54			100		71	100	85				85		FNAB	
Wong, 1995 ¹⁰	RA	recurring/residual disease	16	16			94		100	100	100				80	76	surg/histopath	
Lapela, 1995 ¹¹	RA	post-treatment necks previously treated/clin suspic assess prim nodal mets possible local recurrence detect suspected recurrence	12	12	23		50	40	100	100	80	76			85	92		
Summary		by patients by lesions	511	426	386		71	100	100	83					85	92		33
							100			25							CT/MRI/histol/hk dissect	
							67	67	100	25					54			
															100			
															100	54		
							88	25	100	75	100	67	80	33	92	42	histopath	
							100										histol neck dissection	
															100			
															100			
							88	88	86	93	75	87			87		histol/follow-up to 22 mos	
							92		50	79					78			
							93	54	83	74	73	67	91	33	87	65		33
							84	95	92	86	77	55	91	95	90	85		

TABLE 5 (Continued)

HEAD/NECK CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT(%) EFFECT	
Monitoring Response																			
Lowe, 2000	FA	recurr detect/ser post-ther/Stage II/III	44	16			100	38	93	85							biop/pathol		
Lowe, 1997	FA	response to chemo	28	27			90		83		95		71		89		tissue biopsies		
Wong, 1997	FA	assessmt/prim/recurr/residual	54	54			67	67	100	25					100	54	surg		
Berlangieri, 1994 ¹²	FA	radiother and chemo/loc adv/non-met	6	6	16		100										clin response		
Haberkm, 1993 ¹³	FA	assess early chemo effects	18	19	19		44										CT		
Chaiken, 1993 ¹⁴	FA	tumor rspnse&control post rad ther	19	19	6		100		100								pathol/other imaging		
		post rad/susp ex/inconclus MRI/H&N only		6			100		100		80								
Summary		by patients	169	128			84	60	95	39	92		71		96	54			
		by lesions			16		44												
Other																			
No Articles																			
¹ PET:71pts/78 studies/74 locations. CT:75 pts/74 locations. ² Sens based on 136 neck sides. ³ Sens by lesions = 24/27=89. ⁴ 60 pts w prim tumors. 9 studies w PET findings in the chest correlated w rel stds. ⁵ 60 pts total. 34 pts staging. 7 pts adv disease/laser excision. 19 pts recurrence. ⁶ Comparison to ¹¹ C-methionine tracer in 14 head & neck cancer pts prior to therapy. ⁷ 14 pts w H&N prim tumors. 2 pts w lung prim tumors. 1 pt w occult prim tumor never found. ⁸ Sens and spec of visual interpretation of PET depended on selected scheme of grading lesions. ⁹ 28 pts/37 FNAB & PET scans. ¹⁰ Values based upon defining rating of 4 as positive on 5 point rating system from 0 to 4 where 0=def nt recur and 4=def recur. ¹¹ 4 pts w H&N prim tumors. 2 pts w lung prim tumors. 1 pt w occult prim tumor never found. ¹² Values based upon counting only lesions w hi uptake @ visual assessmt as +. ¹³ Sens defined as exhibiting confirmed signif decline in tumor:ntumor. FDG ratios. ¹⁴ Sens=44 based on positive response being decrease in FDG uptake. If definition includes no change w decrease, sens=94. ¹⁵ 15 pts w H&N cancer. 4 pts w breast cancer.																			



Disease Background. In the United States, breast cancer is currently second only to lung cancer as the leading cancer causing death in women. It is the most common single cause of death for women ages 35–50 y. Cure can be achieved with early diagnosis and treatment, but a multidisciplinary approach is required. Treatment includes surgery, which is becoming progressively less radical, together with chemotherapy. Hormone and radiation therapy also are used ther-

apeutically. Imaging is an important part of detection, staging, and management of most breast cancer patients. Although mammography has helped to detect breast cancer in many women, many cancers are missed in women who have dense breasts, implants, or have been treated previously for breast cancer. In addition, more methods are needed to better detect the spread of breast cancer and to monitor treatment and recurrence.

Case Example. A 61-y-old woman with breast cancer showed several foci of tumor involvement in the chest and spine (Fig. 6, top row) on her initial FDG PET scan. After chemotherapy, an FDG PET study was requested to look for tumor response to chemotherapy (Fig. 6, bottom row). The small foci of FDG accumulation seen throughout the chest and spine clearly had resolved. Post-therapy CT was positive (still showed tumor mass) because of necrosis and edema from therapy.

Why Did FDG PET Help? FDG PET showed that the chemotherapy was working and that this patient's breast cancer had responded to this particular type of chemotherapy. These changes were evident long before the CT scan showed any signs of response to treatment.

Key Management Issues.

- Determining if a breast mass is benign or malignant (This is especially difficult in dense breasts, implants, and after treatment. Approximately 60%–85% of breast biopsies are benign.)
- Staging of axillary and internal mammary lymph nodes
- Detecting metastatic disease
- Detecting local or distant recurrence
- Assessing the response of the tumor to treatment

Summary of Evidence for FDG PET in Breast Cancer.

For diagnosis: An estimated 100% change was noted in management effect, based on six patient studies (Table 6). Because of the limited number of patient studies upon which this management change is based, this value should be interpreted with caution.

For staging: An estimated 24% change in management effect, based on 111 patient studies (Table 6).

For recurrence: An estimated 40% change in management effect, based on 23 patient studies (Table 6).

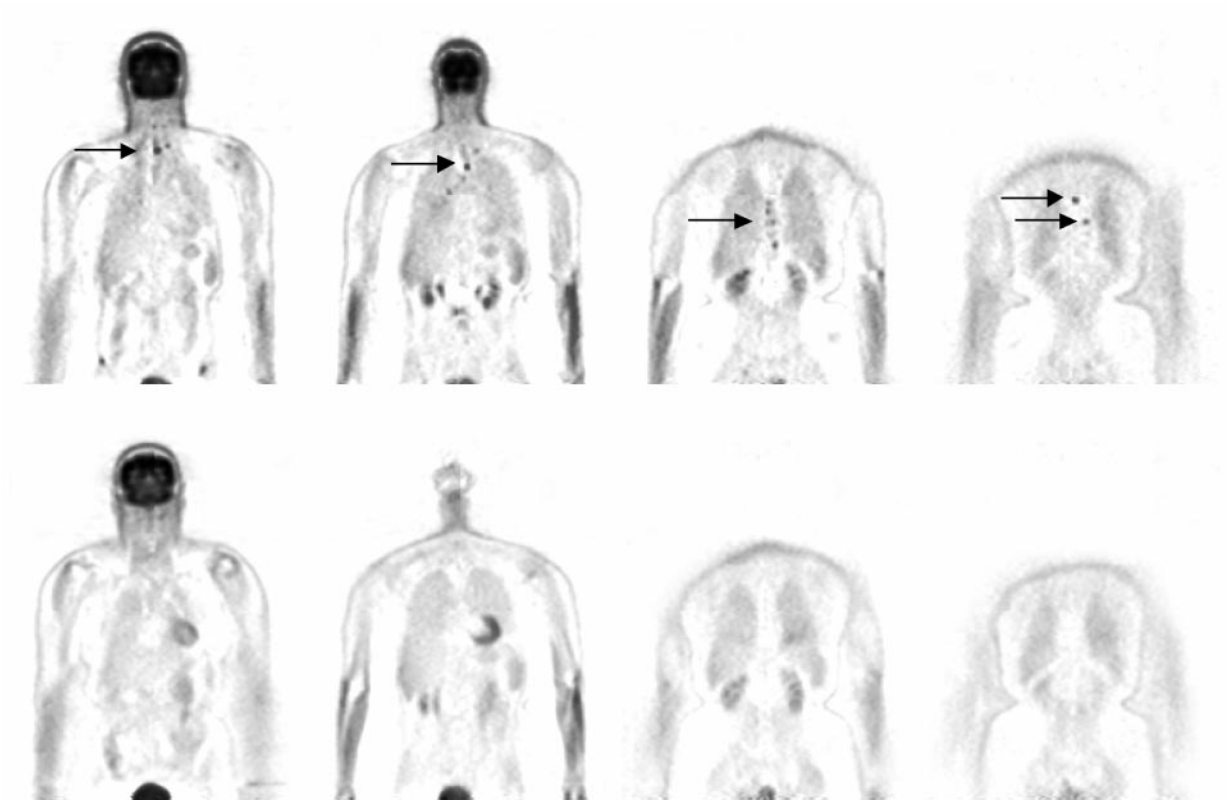


FIGURE 6. Case example, breast cancer.



TABLE 6
FDG PET in Breast Cancer: Results of Literature Search

BREAST CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD SID	MGMT(%) EFFECT	
							SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)			
Diagnosis																			
Fujii, 2000 ¹	A	screening	6	6													palpaton/US		100
Yutani, 1999	A	newly detected/prolif activity	28	28			83										lumpect/mastect		
Yutani, 1999 ²	RA	susp brst ca/prone vs supine image	52	18			94										excis biop/surg		
Noh, 1998	RA	primary breast mass	27	27			96		100								histology/phys ex/MM		
Palmedo, 1997	RA	axillary LN breast masses	20	20			100		92								histopathology		
Yutani, 1997	A	axillae newly detected/PETvsMIBI-SPECT	36	12			79										excis biopsya/nd/or mastect		
Scheidtner, 1996	RA	primary breast ca axillary lymph nodes	30	36		yes	77										histology/MM/US		
Avril, 1996	RA	distant mets primary tumor	51	23	72		100		100								histology		
Crowe, 1994	RA	primary breast ca axillary LN	28	33	33		100		100								phys ex/MM/axillary dissect		
Hoh, 1993 ³	RA	primary breast carcinoma	87	17	20		90		100								histol		
Adler, 1993	RA	breast masses axillary lymph nodes	28	35	35		96		100								axillary node dissection		
Nitzsche, 1993	RA	primary breast masses	37	20	20		94		100								biopsy		
Summary																			
			430	318	180		91		93										100
							90		92										88
Staging																			
Schirmelster, 2000	A	staging breast cancer lymph nodes	117	117			94		75								standard staging procedures		
Yap, 2000 ⁴	A	distant mets staging breast cancer	98	6			100										follow-up		21
Bellon, 2000	A	internal mammary nodes	30	34			50		86								CT/follow-up/xray		
Seltzer, 2000 ⁵	A	management	536	32			89		50								follow-up		42
Henze, 2000	A	dx clin susp LN/mets	14	13	13		100		80								histology		
Bernstein, 2000	A	preop staging/IMLN prim tumor	12	14			100										thoracic CT/biop biopsy/CT		13
Nitzsche, 1993	A	IMLN axillary LN	8	2			100												
Rostom, 1999	RA	prim tumor lymph nodes metastasis	109	4			75										histol/TCB/FNAB		
				93			91		83										89
				74			86		100										90
				19															100

TABLE 6 (Continued)

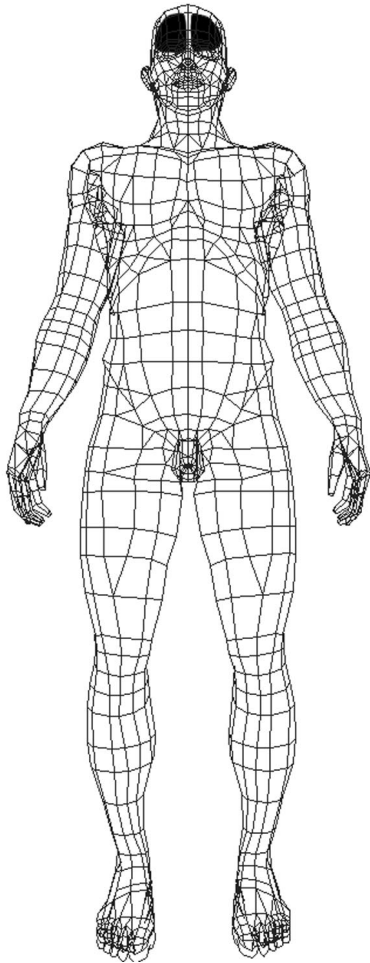
BREAST CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS (%)	SENS CI (%)	SPEC (%)	SPEC CI (%)	PPV (%)	PPV CI (%)	NPV (%)	NPV CI (%)	ACC (%)	ACC CI (%)	GOLD STD	MGMT(%) EFFECT	
Staging																			
Bleckmann, 1999	RA	prim/met	28		189		97										clin/other imaging/histopath		
Schirmmeister, 1999 ⁶	RA	detect met bone disease	34	33			100	100	100								MRI/planar xray/spiral CT/BS	12	
Noh, 1998	RA	primary breast mass	27	27			96	100	100								histology/phys ex/MM		
		axillary LN		27			100	92											
Crippa, 1998	RA	axillary lymph nodes	72	72			85	91									histopathology		
Smith, 1998	RA	axillary lymph nodes	50	50			90	97	95								FNA cytology/axillary dissect		
		T1 tumors		7			100	100											
		locally advanced disease		24			93	100											
Bombardieri, 1998	A	pre-operative staging	134	134			89	87	83								histopathology		
Crippa, 1997 ⁷	RA	pre-op detect axillary mets/overall	82	83			84	85									pathol/surg		
		palpable nodes only		18			92	50											
		nonpalpable nodes only		65			79	89											
Bender, 1997	RA	susp recurrence/lymph nodes	75	75			97	91	88								histology/CT/MRI		
		lymph nodes		63			74	91	95										
		local recurrence		75			80	96	89										
		local recurrence		63			93	98	98										
		bone mets		75			100	98	94										
		bone mets		63			46	98	98										
		lung mets		75			83	97	71										
		lung mets		63			83	96	96										
		liver mets		75			100	97	50										
		liver mets		63			50	95	95										
Adler, 1997	RA	axillary lymph nodes	50	50			95	66	63								axillary LN dissection		
Scheidhauer, 1996	RA	primary breast ca	30	30			91	86									histology/MM/US		
		axillary lymph nodes		18			100	89											
		distant mets		23			100	100											
Utech, 1996	RA	axillary lymph nodes	124	124			100	75	69								surg/biopsy/LN dissection		
Crowe, 1994	RA	primary breast ca	28	/	33		100	100									phys ex/MM/axillary dissect		
		axillary LN		20			90	100											
Adler, 1993	RA	breast masses	28		35		96	100									axillary node dissection		
		axillary lymph nodes			20		90	100											
Summary		by patients	1678	2034			91	88	96	76	74	97	92	90	90			24	
		by lesions			310		95	88	80	80	67	88							

TABLE 6 (Continued)

BREAST CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT (%) EFFECT
Dx/Staging																		
Yutani, 2000	RA	detect susp brst ca/prim axillary LN mets	40	40			79								80		biop/surg	
Noh, 1999 ⁸	RA	detect brst ca/augment brst/prim axillary LN mets	8	9 3			100		83						89 100		MM/US/pathol	
Summary		by patients	48	65			75		83		100				83			
Recurrence																		
Lonneux, 1999	A	recurrence isolated tumor marker elev	28	28			84		55		80		63		75		biop/PET imaging/follow-up	
Eubank, 1999	A	staging nodal disease	69	69			30	88	89	95	76		100		81			
Sugawara, 1999	A	dx brachial plexopathy	26	26			100		67						61		biop/follow-up CT	
Gimenez, 1999	A	axillary metastases	53	53			84		100		100		89		63		surg/biop	
Hathaway, 1999	RA	recurrence	10	10			100		100						90		histopath	
Moon, 1998	RA	recurrence/mets by pt recurrence/mets by les	57	57	41		93		79		82		92				MRI/follow-up/clin data/surg biop/clin follow-up/imaging	
Maldonado, 1998	A	recurrence	23	23			85		79								conv studies	40
Bender, 1997	RA	susp recurrence/lymph nodes lymph nodes local recurrence local recurrence	75	75 63 75			97		91		88		98		93		histology/CT/MRI	
		bone mets		75			80		96		89		86		87			
		bone mets		63			100		98		93		93		92			
		lung mets		75			83		97		94		100		99			
		lung mets		63			100		98		86		88		87			
		liver mets		75			83		96		71		99		96			
		liver mets		63			100		97		50		100		97			
				63			50		95		50		95		91			
Summary		by patients	341	977	41		80	90	85	96	88	93	89	98	82			40
		by lesions					85		79									

TABLE 6 (Continued)

BREAST CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients [§]	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT(%) EFFECT	
Monitoring Response																			
Schelling, 2000 ⁹	RA	rspnse to 1st chemo/locally adv 2nd chemo	22	16			100		85				88				histopath/GRD/MRD		
Smith, 2000 ¹⁰	RA	chemo_rspnse/prim&met/post 1st eval chemo response	22	22	31		83		94				91				pathol/surg		
Gupta-Burt, 1999	A	resp to chemo/locally adv	22	22			100		85								histopathology/MRD		
Smith, 1999	A	predict response to chemo	14	13			57		100				77				surg pathology		
Dehdashi, 1999 ¹¹	RA	rspnse to antiestr ther/ER+ met	24	24			75		100				100				surg		
Hoh, 1998	A	predict response to chemo	11	11									100				follow-up eval 3-24 mos		
Bassa, 1996 ¹²	RA	pre-op chemo/locally adv/prim residual prim init nodal involv residual nodal involv	22 16 16	22 17 16			100 75 77		100 100 100				100 100 100				pathol/surg/MM/US/follow 3y		
Jansson, 1995 ¹³	RA	efficacy of polychemo/post 1st	16	16			42		100								clinical/radiog		
Nieweg, 1993	RA	rspnse to chemo lymph nodes	20 5	11 5			91 100		67 100								pathology		
Wahi, 1993 ¹⁴	RA	mon_chemohormonoher/newly dx	11	11			100		100								clin/radiog/pathol		
Other																			
No Articles																			
		Summary	178	269	31		81		96				92						
		by patients																	
		by lesions																	
		FDG PET can find brst ca early enough for cur surg w few FP.																	
		2 Of 52 pts, 18 pts examined. 17 pts w brst ca/1 w fibrocystic disease. 16/17 detected by PET Irrespective of pt positioning.																	
		3 87 pts included mixed cancer types/prim and met.																	
		4 Clinical stage 33%/major management 21%/minor management 38%/ no change 35%/avert surgery or radiation therapy 9%/addit surgery or radiation 27%/overall 29%.																	
		5 42% pts w other cancers.																	
		6 Crige in clin mgmt=4/34=11.7%. 34 pts. S=17/17. sp=16/16. 1pt w degen lesions.																	
		7 82 pts. 83 cases (ALNDs).																	
		7 Reported acc=13/17=76. 2x2 shows acc=14/18=78.																	
		8 8 pts. 9 cases for prim. 3 TP, 1FP, 5 TN. 3 pts w brst ca examined for LN mets. 2 TP, 1TN.																	
		9 GRD=gross residual disease/nonresponding tumors. MRD=minimal residual disease/responding lesions. Values based on SUV decrease to <55% of baseline.																	
		10 Based on 20% reduction in DUR.																	
		11 Acc based on 2x2 defined as Responder/Nonresponder vs Met Flare +/- 7TP/4TN.																	
		12 16 pts. 1 pt had bilateral breast cancer.																	
		13 values for 1st PET 6 to 13 ds post 1st polychemo trtm. 12 responders/4 non-responders. 8/12 responders w signif decrease in tracer uptake.																	
		14 Sens defined as 8 responders w signif decrease in FDG uptake. Spec defined as 3 non-responders w no decrease in FDG uptake.																	



Disease Background. The incidence of primary brain tumors in the population is 11 in 100,000, with overall metastatic brain disease being more common. Typically, space-occupying lesions are caused by primary tumors, with >50% of patients presenting with some form of epilepsy. New treatments are being introduced, including guided biopsy and surgery (which are frequently image guided), targeted radiation, chemotherapy, and radioactive seed implantation. Outlook remains poor, with

survival <1 y for patients with high-grade tumors. Imaging is increasingly required to detect disease, particularly recurrent disease, and in planning and guiding therapy and biopsy. An especially difficult task is determining if cancer has come back after radiation therapy.

Case Example. The preferred treatment for brain tumors is surgical removal. FDG PET scans are useful for evaluating the efficacy of surgical procedures. A 64-y-old woman with a diagnosis of glioblastoma multiforme (aggressive brain tumor) was operated on to remove the tumor and was treated with radiation. Subsequent contrast-enhanced MRI (Fig. 7, left) suggested possible tumor recurrence. Note the area of contrast accumulation near the surgical region (white arrow). The lack of a corresponding FDG accumulation in that region in the FDG PET image (Fig. 7, right) suggested that the contrast enhancement observed in the MR image was the result of radiation necrosis and that no residual tumor was present at that time.

Why Did FDG PET Help? FDG PET helped by showing that an inconclusive finding on MRI was, in fact, the result of radiation and not residual tumor. Therefore, this patient did not need medical or surgical intervention.

Key Management Issues.

Initial management.

- Diagnosing and grading the malignancy
- Determining the extent for treatment planning
- Directing biopsy
- Determining prognosis

Post-treatment management.

- Differential diagnosis between recurrence and radiation necrosis
- Directing biopsy (This helps to determine where in the brain to sample the tissue, by differentiating tumor from necrosis and edema.)
- Determining the extent of tumor in treatment planning
- Monitoring response to treatment (surgery/radiotherapy/chemotherapy) (This involves differentiating tumor from necrosis and edema to determine how well the treatment affected the tumor.)

Summary of Evidence for FDG PET in Brain Tumors. For recurrence: An estimated 31% change was noted in management effect, based on 89 patient studies (Table 7).

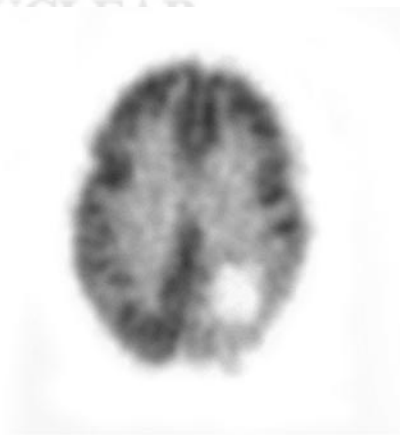
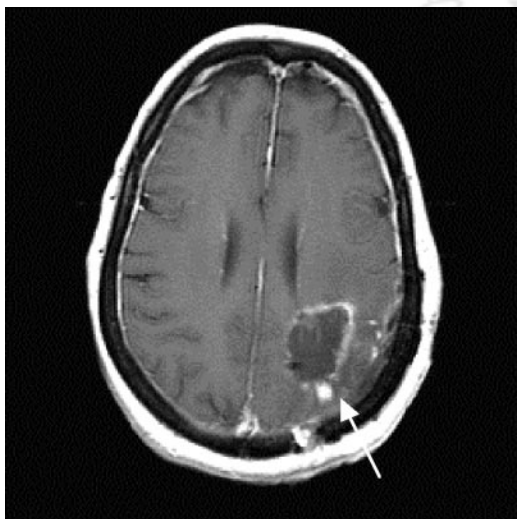


FIGURE 7. Case example, brain tumor.

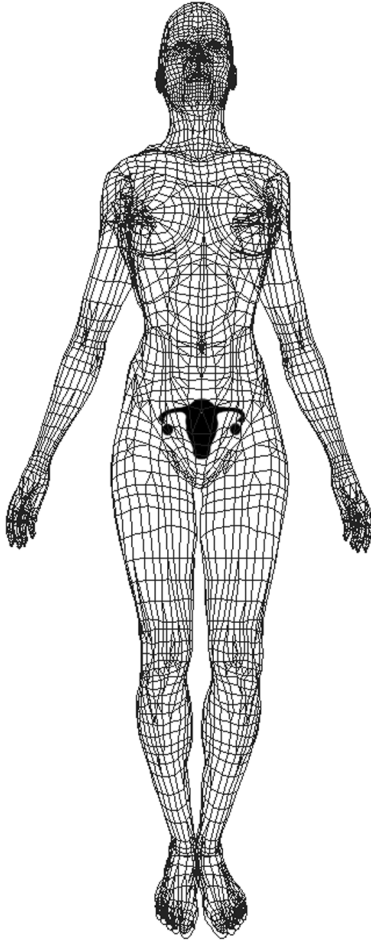
TABLE 7
FDG PET in Brain Tumors: Results of Literature Search

BRAIN TUMOR	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT
						SENS PET (%)	SENS CI (%)	SPEC PET (%)	SPEC CI (%)	PPV PET (%)	PPV CI (%)	NPV PET (%)	NPV CI (%)	ACC PET (%)	ACC CI (%)		
Diagnosis																	
Hustinx, 1999 ¹	RA	eval prim brain tumor	47	27		88										compare to normal gp	
Kincaid, 1998 ²	RA	predicting tumor grade	11	9		100										histol/TI/SPECT/CT/MRI	
Staging																	
Gupta, 1999 ³	RA	eval intracranial lesions	31	31		86										radiol/histol	
Dx/Staging																	
No articles																	
Recurrence																	
Cortes-Blanco, 2000 ⁴	A	recurrence children adults adults	112			81	100									pathol/radiol/follow-up	
Stokkel, 1999 ⁵	FA	recurrence	16	16		62										follow-up CT or MRI	
Thompson, 1999 ⁶	RA	rad necrosis vs recurr/prim glial	15	15	yes	92										stereo biop/cranio/histol	
Bader, 1998	RA	detect recurrence	39	39		82	100									IMT-SPECT/CT/MRI/biop	
Kim, 1998 ⁷	RA	met br tumor	20	13		85	69									histol	
Berry, 1997	A	recurrence	17	17		82										biopsy	
Bader, 1997 ⁸	A	recurrence	21	21		92										biopsy/MRI	
Raja, 1997 ⁹	A	recurrence	19	19		92										MRI/follow-up	
Deshmukh, 1996 ¹⁰	RA	recurrence	75	89		81	40									pathol	31
Kahn, 1994 ¹¹	FA	recurrence	19	21		81										biopsy/clin follow-up	
Janus, 1993 ¹²	RA	prim malig br tum/susp progress	50	20												surg	
Summary		by patients	403	367		79	69	77	90	56	76	31					

TABLE 7 (Continued)

BRAIN TUMOR	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		MGMT(%) EFFECT	
						PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	ACC	CI (%)
Monitoring Response																	
Ericson, 1996 ¹³	RA	stereotactically irradiated brain metastases	31	17		82		83									autopsy/surg/histol
Rozental, 1993 ¹⁴	RA	effects of BCNU on glucose uptake	6	6													
Holthoff, 1993 ¹⁵	RA	effect of chemo on tumor metabolism	15	7													close clin follow-up
	Summary	by patients	52	30		82		83									
Other																	
Weber, 1997 ¹⁶	RA	identification of tumor tissue	19	19		88		67		93		50		84			surg/biops
Holzer, 1993 ¹⁷	RA	prognostic indicator survival	15	15		100											clin follow-up/2y
	Summary	by patients	34	34		93		67		93		50		84			
¹² 0 normal pts=Gp1. 27 malignant primary CNS pts=Gp2. S=88 from visual analysis. ² All 9 low-grade gangliogliomas. All showed decreased or normal PET. Considered 100% correlative to tumor grade. ³ 31 pts. 22 pts w intracranial metastases/9 benign. ⁴ PET imaging in children reveals superior performance for detecting brain tumor recurrence and differentiating it than in adults. ⁵ 18F-FDG gave poorer results than 201TI SPET. ⁶ PET believed insufficient to resolve rad necrosis vs tumor progression. ⁷ 13/20 had metastatic brain tumor. PET found 11/13 and conv found 9/13. ⁸ 2 pts w recurrence had a change of grade. s=12/13=92. Was 1FN. ⁹ Listed in spots what was reported. Reported sens is actually PPV, and reported spec is actually NPV. ¹⁰ 28/89=.31 for change in therapy. 86/89=.97 for playing clinical role. 53/89=.59 for withholding aggressive therapy. 75pts/89scans. ¹¹ 19 pts. 21 scans. 13TP/3FP/3FN/2TN. Both 201TI SPECT and FDG PET were sensitive for lesions <1.6 cm or larger. ¹² 20 pts had surg. 9 w increase uptake and evidence of tumor. 6 w decrease uptake and no evidence of tumor. 5 pts w no correlation. Acc=15/20=.75. ¹³ 2x2 of tumor growth (in spite of therapy or regrowth post initial favorable response) vs FDG increase/decrease. 9TP/1FP/2FN/5 TN. ¹⁴ Patients w largest percentage change in FDG uptake following adjuvant BCNU had to have shortest survival. ¹⁵ Data suggests the more marked decrease in tumor metabolism post chemotherapy, the longer the period of initial clinical improvement. ¹⁶ 19 pts. 16 w tumor. 3 non-tumorous. In 2x2, discordant readings (between 2 observers) assumed as positive. ¹⁷ All patients had same treatment to assess prognostic value of PET for survival time and recurrence. Since not truly a response to treatment study, was put into OTHER.																	

Ovarian, Cervical, and Uterine Cancer



Disease Background. Ovarian cancer is the fifth leading cause of cancer death in women in the United States, with 14,500 deaths and 25,400 new cases diagnosed each year. Approximately one-third of all new cases will have metastatic disease at the time of diagnosis, with another third developing clinical metastases during the first year after surgical resection. The current recommendation for management of patients without evidence of metastatic disease at 1 y after diagnosis is to

perform second-look laparotomy for clinical staging and possible tumor resection. For early-stage ovarian cancer, accurate diagnosis is very difficult.

Cervical cancer is one of the most common cancers, accounting for 6% of all malignancies in women, with an estimated 16,000 new cases of invasive cancer of the cervix and 5,000 deaths in the United States each year. The prognosis for this disease is markedly affected by the extent of disease at the time of diagnosis.

Cancer of the endometrium, a common type of cancer in women, is a disease in which cancer cells are found in the lining of the uterus (endometrium). Cancer of the endometrium is different from cancer of the muscle of the uterus (sarcoma of the uterus). Cancer of the endometrium is the most common pelvic gynecologic malignancy and accounts for 13% of all cancers in women. It is a highly curable tumor.

Case Example. A 50-y-old woman with a history of ovarian cancer showed rising tumor markers in an annual blood test that looked for possible tumor recurrence. A follow-up CT scan was unable to find the source of the recurrence. An FDG PET study showed that the tumor had metastasized to the right lobe of the liver (Fig. 8, arrows on site of metastasis viewed on 4 different sections through the whole body). No other areas of metastasis were seen.

Why Did FDG PET Help? FDG PET showed that the blood study was correct (it was not falsely elevated) and that the source of recurrence was the liver. This patient was confirmed through follow-up to have recurrence in the liver.

Key Management Issues.

- Staging lymph nodes
- Identifying recurrent disease after surgery and radiation
- Assessing response to treatment

Summary of Evidence for FDG PET in Ovarian, Uterine, and Cervical Cancer. For recurrence: An estimated 17% change was noted in management effect, based on 30 patient studies (Table 8).

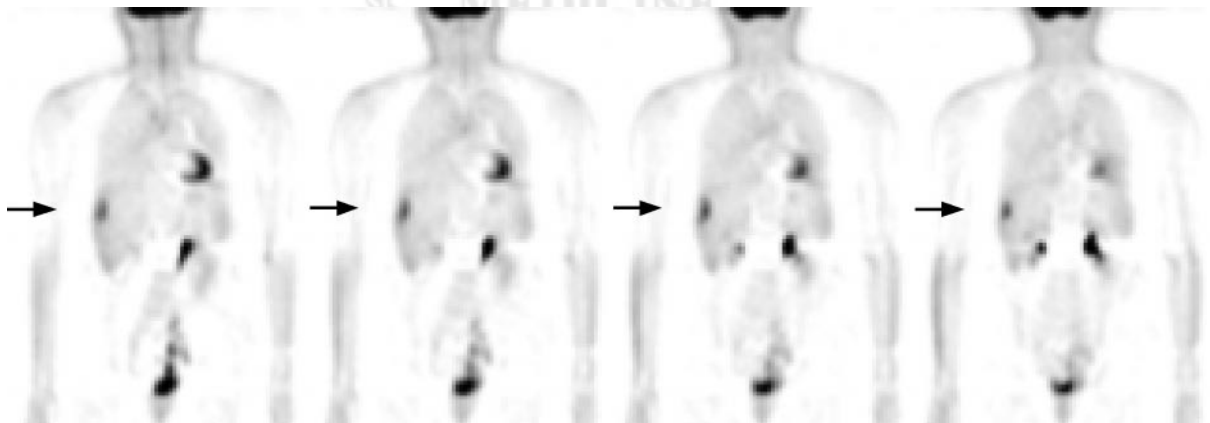


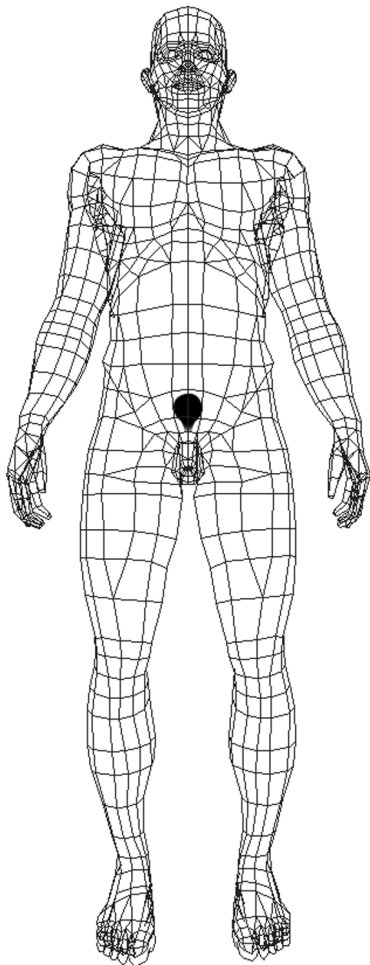
FIGURE 8. Case example, ovarian, cervical, and uterine cancer.

TABLE 8
FDG PET in Ovarian, Cervical, and Uterine Cancer: Results of Literature Search

OVARIAN/PELV MASS & UTERINE&CERVICAL	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL. Studies	Non-Ded. PET	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT
						SENS (%)	PET (%)	SPEC (%)	CT (%)	PPV (%)	CT (%)	NPV (%)	PET (%)	ACC (%)	CT (%)		
Diagnosis																	
Grab, 2000	RA	characterize asymp adnexal masses	101	101		58	80			28		93		77		laparoscopy/histol	
Kubik-Huch, 2000	RA	dx prim/susp ovar lesion/presentation	19	7		100	100	67	67					86	86	laparotomy/histol	
		dx recurr/susp @ follow-up		10		100	40	50	50					90	43		
Fenchel, 1999 ¹	RA	dx of pelvic mass	85	85		50	78			19		94		75		histopath	
Grigsby, 1999	A	prim tumor	36	36		97											
Zimny, 1997	RA	primary/recur ovarian	26	26		84	86			94		67		85		CI (chest xray/lymphangiography/CT)	
Römer, 1997	RA	dx of ovarian tumor	19	19		83	54			45		88		63		surg/cytol/histol	
																surg/histol	
	Summary	by patients	286	284		66	100	77	67	34		90		77	76		
Staging																	
Grigsby, 1999 ²	A	staging node&dist mets/prim carcin cervix	36	36													
		pelv lymph nodes		36		72	21										
		para-aortic lymph nodes		36		31	18										
		lit supraclavicular LN's		36		11											biopsy
		inguinal LN mets		36		3											biopsy
		pulmonary uptake		36		14											biopsy
Rose, 1999 ³	RA	staging/loc adv cerv ca	32	32													
		cervical tumors		32		91											histol
		para-aortic LN mets		32		75		92		75		92				retroperitoneal lymphadenectomy	
		pelv node mets		17		100	45	100	100	100		100				surg	
Smith, 1998	A	staging/recurrent	57	57		94	85	96	76							histol	
Karlan, 1993	RA	staging/recurrent	13	13		50	100									surg	
	Summary	by patients	138	399		54	48	96	76	84		95					
Dx/Staging																	
Both, 2000 ⁴	A	primary/staging	22	22		95								100		surg/MRI	
Sugawara, 1999 ⁵	RA	dx/staging recurr/cerv ca	21	21		76										biop/phys exam/correl imaging	
		postvoid imaging		11		100											
		LN mets		7		86	57	100	100							surg/clin follow-up/CT	
Hübner, 1993 ⁶	RA	primary tumor/staging	51	51		83	82	80	53	86	77	76	62	82	72	surg/histol	
		recurrent/follow-up scans		14		86		100						93		biop/clinical survival	
	Summary	by patients	94	126		86	79	82	59	86	77	76	62	87	72		

TABLE 8 (Continued)

OVARIAN/PELVY MASS & UTERINE/CERVICAL	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT EFFECT
						PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)	PET (%)	CI (%)		
Recurrence																	
Kubik-Huch, 2000	RA	dx prim/susp ovar lesion/presentation dx recurr/susp @ follow-up	19	7		100	100	67	67					86	86	laparotomy/histol	
Torizuka, 2000 ⁷	A	recurrence	11	11		100	40	50	50					90	43		
Kim, 2000	A	detect recurr/cerv ca* *NED post trtm	101	101		100	75	100	100	89	100	100	100	82	91	surg/histol/clin follow-up >4 mos clin follow-up/CT/MRI/biop	
Nakamoto, 2000	A	detect recurr/gynecol malign susp recurr	30	30		82	82	92	92					87	87	histopath/clin follow-up	25
Sugawara, 1999 ⁸	RA	dx/staging recurr/cerv ca postvoid imaging	21	21		76	76	91	91							biophys exam/correl imaging	7
Smith, 1998	A	LN mets staging/recurrent	57	7		86	57	100	100							surg/clin follow-up/CT histol	
Zimny, 1997	RA	primary/recr ovarian	26	26		94	85	96	76					94	85	surg/cyto/histol	
Römer, 1997	RA	dx of ovarian tumor	19	19		83	83	54	54	45	88	63	45	88	63	surg/histol	
Casey, 1994	RA	residual/recr/abdom & pelv tum	9	9		83	100	100	100	100	75	89	100	75	89	second look laparotomy	
Karlan, 1993 ⁹	RA	staging/recurrent	13	13		50	50	100	100							surg	
Hübner, 1993 ¹⁰	RA	primary tumor/staging recurrent/follow-up scans	51	51		83	82	80	53	86	77	76	62	82	72	surg/histol biop/clinical survival	
	Summary	by patients	357	417		88	76	90	75	85	77	92	62	87	43		17
Monitoring Response																	
Kerrou, 2000 ¹¹	A	detect recurr/respnse to chemo	40	11	yes	100										surg histol/LT follow-up	
	Summary	by patients	40	11		100											
Other																	
No articles																	
¹ Sens of PET in detection of borderline-tumors and early stage ovarian cancer seems to be limited. ² FDG-PET sens for detecting nodal and dist mets in pts w carcinoma of cervix. ³ PET-FDG accurately predicts both the presence and absence of pelvic and para-aortic nodal met disease. ⁴ Art shows 30/32=94 and not sens=91 which is reported. ⁵ sens=95 for primary tumor/acc=1.00 for staging. ⁶ Promising for detecting untreated or recurrent cervical cancer. ⁷ 14 pts w repeat scans for recurrence. 6 TP/7 TN as described in art. 1 FN derived from Table 7. ⁸ PET is effective for detection and staging of ovarian cancer. ⁹ Promising for detecting untreated or recurrent cervical cancer. ¹⁰ 13 pts. 1/7 pts w clin evid of recur dis refused lapar/biop for pathol cnfrm. Considered as PET neg (FN). 6TP/6FN/1TN. ¹¹ Abstract rpts overall sens=8/8, overall acc=11/11. 2x2's show sens=8/9, acc=10/11.																	



Disease Background. Bladder cancer is a disease in which cancer cells originate from the bladder wall. Approximately 70%–80% of patients with newly diagnosed bladder cancer will present

with superficial bladder tumors. Those tumors that are noninvasive are often curable, and those that are deeply invasive are sometimes cured by surgery, irradiation, or a combination of modalities that includes chemotherapy. Some patients with distant metastases have achieved long-term complete response after treatment with combination chemotherapy regimens. The major prognostic factors in carcinoma of the bladder are the depth of invasion into the bladder wall and the degree of differentiation of the tumor. Transurethral surgery, intravesical medications, and cystectomy (bladder removal) have been used in the management of patients with superficial tumors and are all associated with 5-y survival rates for 55%–80% of patients treated. As with many cancers, the key to management is determining if the bladder cancer has spread beyond the bladder to the local lymph nodes or to distant parts of the body.

Case Example. A patient with cancer of the bladder was scanned for staging purposes. Focal increased FDG uptake was seen within the posterior aspect (back) of the bladder, indicating primary disease only (Fig. 9, arrow). Mild accumulation of FDG also was seen around a right total hip replacement (Fig. 9A), possibly indicating active inflammation or infection, although the patient did not complain of any hip pain.

Why did FDG PET help? FDG PET helped by showing no evidence of cancer spread beyond the bladder, so that local treatment (e.g., removal of the bladder) likely would benefit the patient.

Key Management Issues.

- Primary nodal staging
- Systemic metastases staging

Summary of Evidence for FDG PET in Bladder Cancer. For staging: An estimated 17% change was noted in management effect, based on 12 patient studies (Table 9).

For recurrence: An estimated 17% change was noted in management effect, based on 12 patient studies (Table 9). Because management effect for both staging and recurrence is based upon the same single study of 12 patients, results should be interpreted with caution.

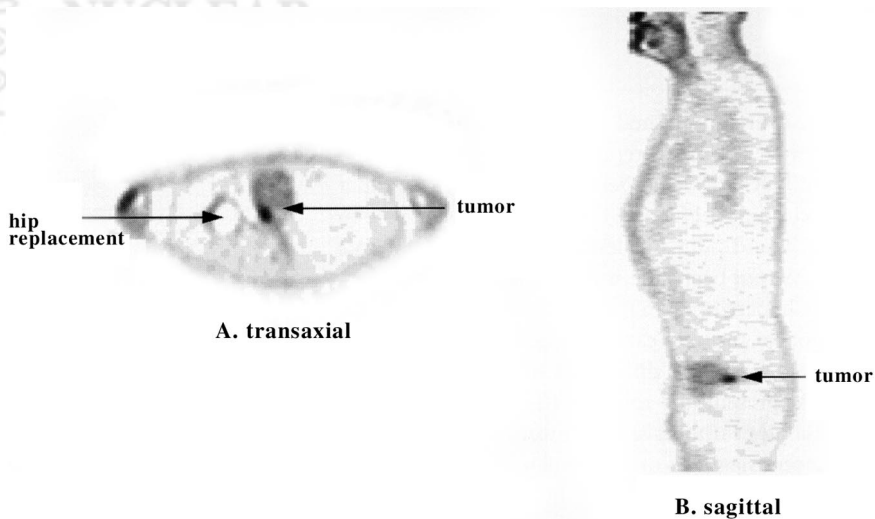
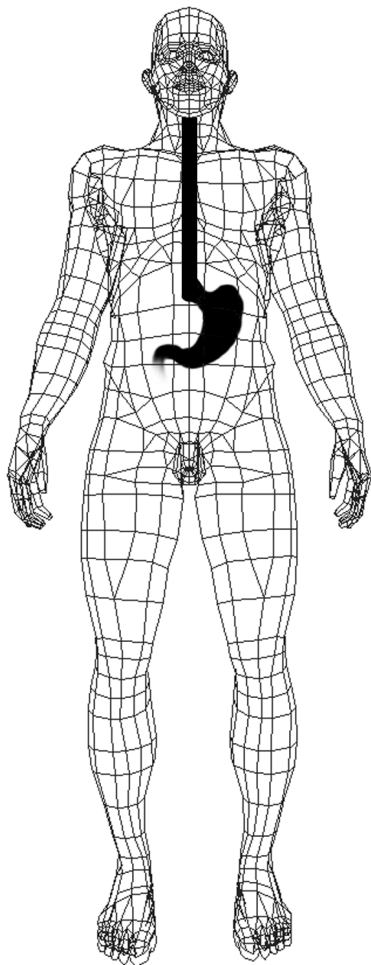


FIGURE 9. Case example, bladder cancer. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.

TABLE 9
FDG PET in Bladder Cancer: Results of Literature Search

BLADDER CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT
					PET	CT	PET	CT	PET	CT	PET	CT	PET	CT		
Diagnosis																
No Articles																
Staging																
Bachor, 1999 ¹	RA	lymph node staging	64	64	67	86	70	84	80					histol/lymphadenectomy		
Heicappell, 1999 ²	RA	pelv lymph node staging	8	8	67	100	100	83	88					histol/pelv LN dissection		
Kosuda, 1996 ³	A	staging/recur/LN mets/SUV 3.64	12	12	60									conv imaging		17
Bachor, 1995	RA	pre-op staging/prim staging LN's	26	26	85	86	63	100	88					surg/histol		
	Summary	by patients	110	136	76	87	71	88	83							17
Dx/Staging																
Bachor, 1995	RA	pre-op staging/prim staging LN's	26	26	85	86	63	100	88					surg/histol		
	Summary	by patients	26	52	93	86	63	100	88							
Recurrence																
Kosuda, 1996 ⁴	A	staging/recur/LN mets/SUV 3.64	12	12	60									conv imaging		17
	Summary	by patients	12	12	60											17
Monitoring Response																
No Articles																
Other																
No Articles																

¹PET results seem better than CT or MRI.
²FDG-PET may be valuable diagnostic tool in staging pelvic LN's in bladder and prostate ca.
³FDG PET may be useful in dx of perivesical tumor growth or dist met in adv stage dis. Recurr fd in 2/12=17% post-irrtmt.
⁴FDG PET may be useful in dx of perivesical tumor growth or dist met in adv stage dis. Recurr fd in 2/12=17% post-irrtmt.



Disease Background (Gastric Cancer). Cancer of the stomach, also called gastric cancer, is a disease in which cancer cells originate from the tissues of the stomach. Cancer of the distal half of the stomach has been decreasing in the United States since the 1930s. However, in the last 2 decades, the incidence of cancer of the cardia and gastroesophageal junction (upper half of the stomach) has been rising rapidly. The incidence of this cancer, especially in patients younger than 40 y, has increased dramatically. In localized distal gastric cancer, >50% of patients can be cured. However, early stage disease accounts for only 10%–20% of all cases diagnosed in the United States. The remaining patients present with metastatic disease in either regional or distant sites. The overall survival rate in these patients at 5 y ranges from almost no survival for patients with disseminated disease to almost 50% survival for patients with localized distal gastric cancers confined to resectable regional disease. Even with apparent localized disease, the 5-y survival rate of patients with proximal gastric cancer is only 10%–15%. Although the treatment of patients with disseminated gastric cancer may result in palliation of symptoms and some prolongation of survival, long remissions are uncom-

mon. Radical surgery represents the standard form of therapy with curative intent. Lesser surgical procedures also may play important roles in palliative therapy for patients with gastric cancer. Neoadjuvant or postoperative chemotherapy and/or radiation therapy are under clinical evaluation.

Disease Background (Esophageal Cancer). Carcinoma of the esophagus is increasing rapidly in frequency in the west, with the rise most apparent in patients with adenocarcinoma of the esophagus. Much of the increase is thought to be related to reflux esophagitis and Barrett's esophagus (conditions in which acid from the stomach damages the esophagus), but the exact cause is uncertain. Adenocarcinoma of the esophagus is now more prevalent than squamous cell carcinoma in the United States and western Europe, with most tumors located in the distal esophagus. Esophageal cancer is a treatable disease but is rarely curable. The overall 5-y survival rate in those cases amenable to surgery ranges from 5%–20%. The occasional patient with very early disease has a better chance of survival. Primary treatment modalities include surgery alone or chemotherapy with radiation therapy. Combined modality therapy (chemotherapy plus surgery or chemotherapy and radiation therapy plus surgery) is under clinical evaluation.

Case Example (Gastric Cancer). A 35-y-old patient underwent surgery for gastric cancer. At the time of surgery, a portion of the stomach was removed around the tumor site. During surgery, it was noted that lymph nodes near the stomach also were involved. The patient therefore underwent chemotherapy to treat for spread of the gastric cancer. A CT scan was performed after 6 mo and showed questionable enlargement of lymph nodes in the abdomen. An FDG PET scan was ordered to determine whether the lymph nodes seen on the CT scan were in fact consistent with tumor involvement. The FDG PET scan (Fig. 10) shows several areas of focal increased FDG accumulation in the midabdomen (arrow), confirming tumor recurrence.

Why Did FDG PET Help? FDG PET confirmed that the questionable findings on CT scan, in fact, were likely to be tumor. Sometimes the CT scan can show lymph node enlargement when no tumor has recurred. In the case shown, there was likely to be tumor recurrence, and the patient now could be managed with the maximal information in hand.

Case Example (Esophageal Cancer). A 59-y-old man with known esophageal cancer was referred for FDG PET scanning before surgery. CT demonstrated the presence of the primary tumor but no spread of disease. FDG PET showed uptake in the primary tumor (Fig. 11B, lower arrow) and a lymph node near the trachea (Fig. 11A, arrow, and 11B, upper arrow). The esophageal cancer had spread beyond the esophagus.

Why Did FDG PET Help? FDG PET showed that the cancer had spread beyond the esophagus. Esophageal surgery alone, therefore, was not the best way to manage this patient.

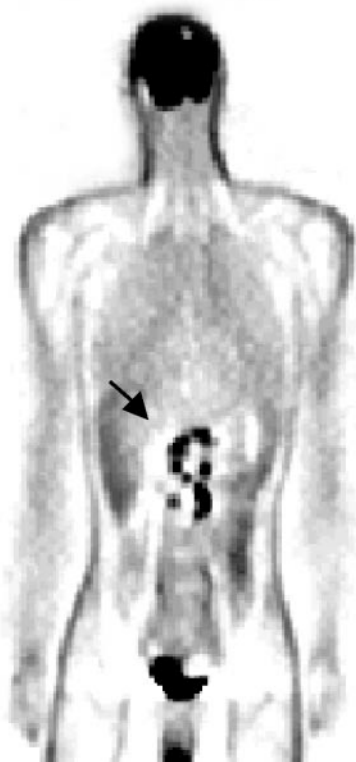


FIGURE 10. Case example, gastric cancer.

Key Management Issues.

- Staging for possible spread of tumor
- Assessing for recurrence

Summary of Evidence for FDG PET in Gastroesophageal Cancer. For diagnosis: An estimated 14% change was noted in management effect, based on 99 patient studies with 276 lesion sites (Table 10).

For staging: An estimated 20% change was noted in management effect, based on 229 patient studies (Table 10).

For diagnosis/staging: An estimated 14% change was noted in management effect, based on 109 patient studies (Table 10).

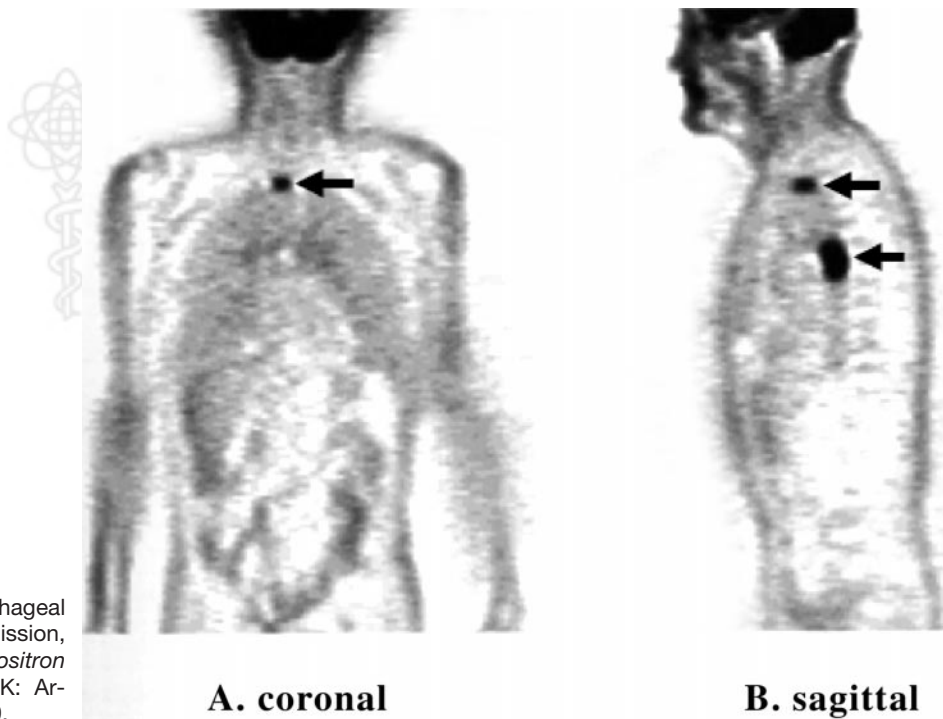


FIGURE 11. Case example, esophageal cancer. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.

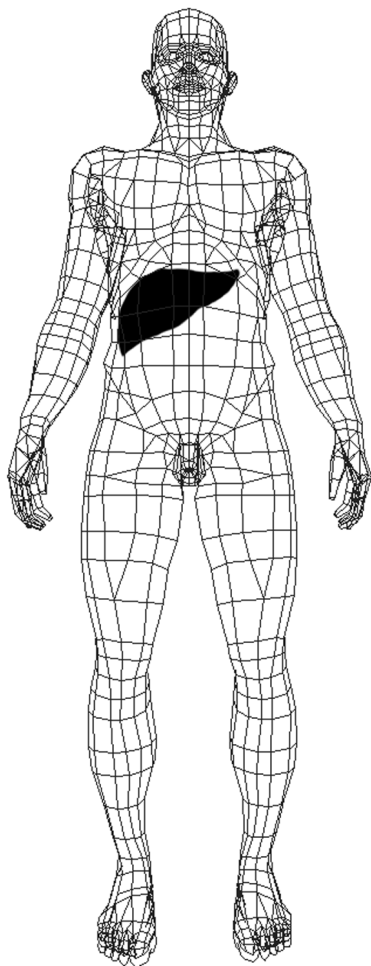
TABLE 10
FDG PET in Gastroesophageal Cancer: Results of Literature Search

GASTRO-ESOPHAGEAL CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT
						CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)		
Diagnosis																	
Yeung, 1999 ¹	RA	dx/staging/follow-up	109		276	80	95							86		histo/clin follow >6mo	14
Fukunaga, 1998 ²	RA	eval esophageal ca	48	48	269		68	81						98	73	surg/follow-up 7y/CT/US surg/pathol	
Kole, 1998	RA	primary tumor	26	26		96	81										
Block, 1997	RA	primary tumor	58	58		94										biop/pathol	
Flanagan, 1997	RA	primary tumor	36	36		100										surg/biop	
Summary		by patients by lesions	277	168	545	80	81	95	81					98	73		14
Staging																	
Choi, 2000	RA	eval individ LN groups	61	48		57	18	97	99							esophagectomy/LN dissect surg/histol	
Melitzer, 2000 ³	RA	init staging/esoph ca locoreg LN	47	47		41	75	90	29	93							
Cambier, 2000	A	dist mets	41	10		70		90		70	29						
Que, 2000 ⁴	A	metastatic disease	17	41	15	96	67							89		pathol/radiol/clin follow-up operative/pathological	
Luketich, 1999 ⁵	RA	staging of distant mets	91	100		69	46	93	74					84	63	surg	16
Kole, 1998	RA	prim/lymph nodes	26	26		96	81									surg/pathol	
Rankin, 1998	RA	pre-op esophageal ca/prim peri-esophageal nodes	25	19		100	95									histol	
Luketich, 1997 ⁶	RA	left gastric nodes	50	35		38	50										
		staging esophageal ca/dist mets				11	56							91		surg	20
		local-regional nodal mets				88		93						48			
Block, 1997	RA	prim	58	58		45	100									pathol/ biop	29
		lymph node mets				97											
		distant metastases				52	29										
Flanagan, 1997	RA	prim	36	36		100	29									surg/tissue sampling	14
		lymph nodes				76	45										
Summary		by patients by lesions	452	545	15	73	50	90	69	89	29			83	68		20
Dx/Staging																	
Yeung, 1999	RA	dx/staging/follow-up	109	109		80	68	95	81					86	73	surg/pathol	14
Summary		by patients	109	109		80	68	95	81					86	73		14

TABLE 10 (Continued)

GASTRO-ESOPHAGEAL CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	SENS		SPEC		PPV		NPV		ACC		GOLD SID	MGMT(%) EFFECT
						SENS PEI (%)	SENS CI (%)	SPEC PEI (%)	SPEC CI (%)	PPV PEI (%)	PPV CI (%)	NPV PEI (%)	NPV CI (%)	ACC PEI (%)	ACC CI (%)		
Recurrence																	
Cambier, 2000	A	recurrence	41	41		100	43							73		pathol/follow-up	
	Summary	by patients	41	41		100	43							73			
Monitoring Response																	
Couper, 1998 ⁷	PA	chemo rrsponse/oesophageal & gastric ca	14	13		100	36							46		CT/dysphagia scores/wt chge	
	Summary	by patients	14	13		100	36							46			
Other																	
No articles																	
¹ 109 pts/PET: 276 sites (99pts) 131TP/6FP/32FN/107TN. CT: 269 sites 109TP/21FP/51FN/88TN. ² SUV Cutoff = 2.0. ³ Hi-sens mode(equiv is +). Mid-pt of range given(63-87). ⁴ PET is better than CHOL for gastro cancer. ⁵ 91 pts/100_sens. PET:27TP/4FP/12FN/57TN. CT:18TP/16FP/21FN/45TN. 16/100 cases directed to not perform esophagectomy based on PET. ⁶ 20% of pts identified for unsuspected dist mets. ⁷ Values based on 2x2 defined as Responder/Nonresponder vs FDG descr/FDG Incr. 4 TP/7FP/2TN.																	

Hepatocellular Cancer



Disease Background. Adult primary liver cancer is a disease in which cancer cells start to grow in the tissues of the liver. People who have hepatitis B or C or cirrhosis, a disease of the liver, are more likely than other people to get adult primary liver cancer. Primary liver cancer is different from cancer that has spread from another place in the body

to the liver. Hepatocellular carcinoma is a relatively uncommon tumor in the United States, although its incidence is rising. It is the most common cancer in some other parts of the world. Hepatocellular carcinoma is potentially curable by surgical resection, but surgery is the treatment of choice for only the small fraction of patients with localized disease. Prognosis depends on the degree of local tumor replacement and the extent of liver function impairment. Therapy other than surgical resection is best administered as part of a clinical trial. Hepatocellular carcinoma is associated with cirrhosis in 50%–80% of patients. Five percent of patients with cirrhosis eventually develop hepatocellular cancer, which is often multifocal. Childhood liver cancer, also called hepatoma, is a rare disease in which cancer cells are found in the tissues of a child's liver. Two types of cancer (hepatoblastoma and hepatocellular cancer) start in the liver and are identified by the way the cancer cells look under a microscope. Hepatoblastoma is more common in children younger than 3 y and may have a genetic cause. The overall survival rate for children with hepatoblastoma is 70% but is only 25% for hepatocellular carcinoma.

Case Example. A patient presented to his doctor with vague abdominal symptoms. The work-up, which eventually included a CT scan, revealed that the patient had enlarged lymph nodes near the portal region of the liver. An FDG PET scan was ordered to further evaluate for tumor. The scan revealed uptake of FDG within a focus in the right lobe of the liver (Fig. 12, center). No other foci were present, indicating that the tumor was confined to the liver. The patient went on to have an appropriate surgery for localized hepatoma.

Why Did FDG PET Help? FDG PET indicated that the tumor was localized and that the patient was a candidate for surgery.

Key Management Issues.

- Distinguishing between cirrhosis and hepatoma
- Assessing response to treatment and differentiating tumor from necrosis, edema, and scarring
- Identifying multifocal lesions

Summary of Evidence for FDG PET in Hepatocellular Cancer. For staging: An estimated 60% change was noted in management effect, based on 20 patient studies (Table 11).

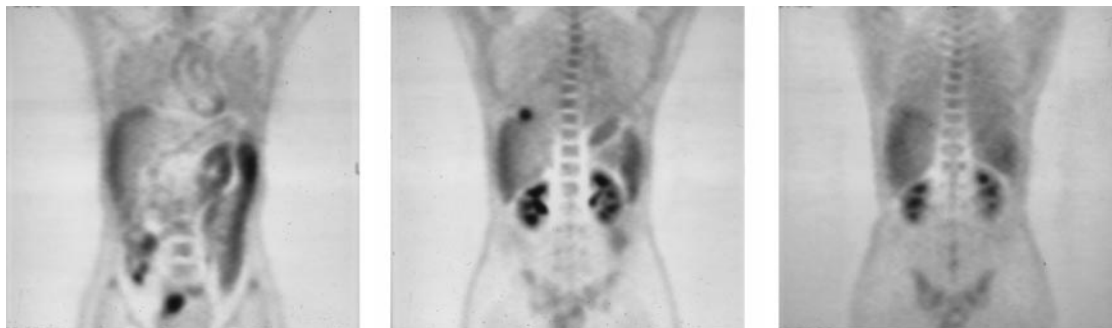


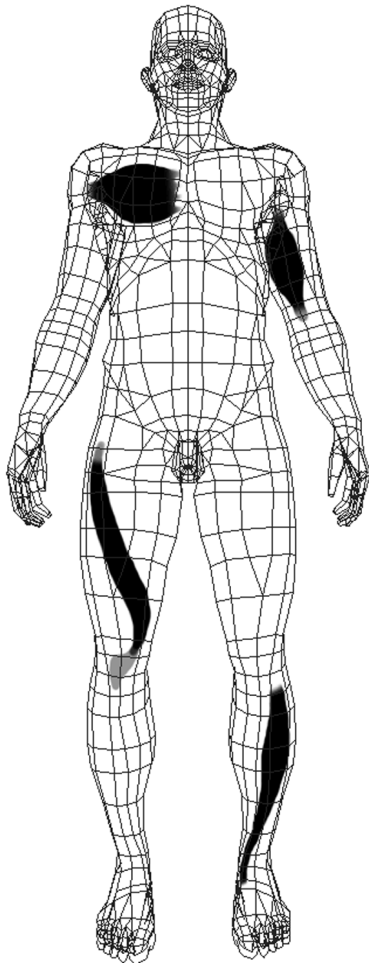
FIGURE 12. Case example, hepatocellular cancer.

TABLE 11
FDG PET in Hepatocellular Cancer: Results of Literature Search

LIVER/HEPATOCELLULAR Cancer	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		NPV		ACC		GOLD STD	MGMT/ (%) EFFECT	
							PET (%)	CT (%)	SPEC PET (%)	CT (%)	NPV PET (%)	CT (%)	ACC PET (%)	CT (%)			
Diagnosis No Articles																	
Staging																	
Schoenberger, 2000	A	indeterm liver lesions	19	19			100	100	100	100	100	100	100		compare w hybrid PET		
Abdel-Nabi, 1999 ¹	A	pre-op staging liver mets	20	20		yes	87	100	100	100	67	89					
Fröhlich, 1999 ²	RA	staging for surgery lesions > 1 cm	168	168			100	68	95	65	95	91		CT/histol/follow-up surg/CT follow-up		60	
Delbecke, 1998 ³	RA	pre-op eval/hep lesions/hepatocell ca	110	23			43							biop/surg/pathol/CT/follow-up			
Abdel-Nabi, 1998 ⁴	RA	staging liver mets lesions <= 1 cm	43	43			70	88	100	97	100	50	97	86	98	81	CT/surg/histopath
Summary		by patients by lesions	317	292			77	38	97	97	76	50	94	86	93	81	
Dx/Staging																	
Trojan, 1999	RA	detect/staging HCC/liver cirrhosis mod or poor differen HCC only	14	14			50	79							US/helical CT/histol/p53/AFP		
Summary		by patients	14	22			64	79									
Recurrence																	
Peterson, 2000 ⁵	A	ident resid or recurr	7	7	9		88	38							serial CT/CEA/biop		
Summary		by patients by lesions	7	7	9		88	38									
Monitoring Response																	
No articles																	
Other																	
No articles																	

¹ FDG PET more sensitive than CT in pre-op eval of liver mets; can select pts to benefit fm cur resect of liver mets.
² FDG provides reliable hepatic staging for lesions >1cm.
³ Did not report the TN's by lesions so did not have total lesion count for weighted average.
⁴ 110 pts. Used pts=23 for hepatocellular carcinoma.
⁵ CT spec liver mets reported and listed as .97. It calcs to be 32/35=.91.
⁶ 2 pts had additional scans following additional ablation.

Muscle and Connective Tissue Tumors



Disease Background. Adult soft tissue sarcoma is a disease in which cancer cells are found in the soft tissue of part of the body. The soft tissues of the body include the muscles, connective tissues (tendons), vessels that carry blood or lymph, joints, and fat. The prognosis for a patient with adult soft tissue sarcomas depends on several factors, including the patient's age and the size, histologic grade, and stage of the tumor. Factors associated with a poorer prognosis are age older than 60 y, tumors >5 cm, and high-grade histology. Although low-grade tumors usually are curable by surgery alone, higher-grade sarcomas (as determined by the mitotic index and the presence of hemorrhage and necrosis) are associated with higher local treatment failure rates and increased metastatic potential. Soft tissue sarcomas are rare in children and adolescents. There are many different kinds of soft tissue sarcoma, depending on the soft tissue in which the cancer begins. Rhabdomyosarcoma is the most common type of childhood soft tissue sarcoma. It begins in muscles around the bone and can be found anywhere in the body.

Case Example. A 41-y-old man had surgery and radiotherapy, first for a liposarcoma in the right thigh and 3 mo later for a solitary metastasis in the abdomen. He developed recurrent disease within the right thigh and was referred for FDG PET scanning. The FDG PET scan showed focal increased metabolism within the right thigh (Fig. 13A, B, and C), indicative of recurrent disease, surrounded by diffuse metabolism secondary to inflammation after surgery. High metabolism of FDG was also noted within lung metastases (Fig. 13C and D).

Why Did FDG PET Help? The FDG PET scan indicated lung and mediastinal metastases, in addition to local disease in the thigh. This meant that the patient would not benefit from treatment of the thigh region alone and would likely require chemotherapy.

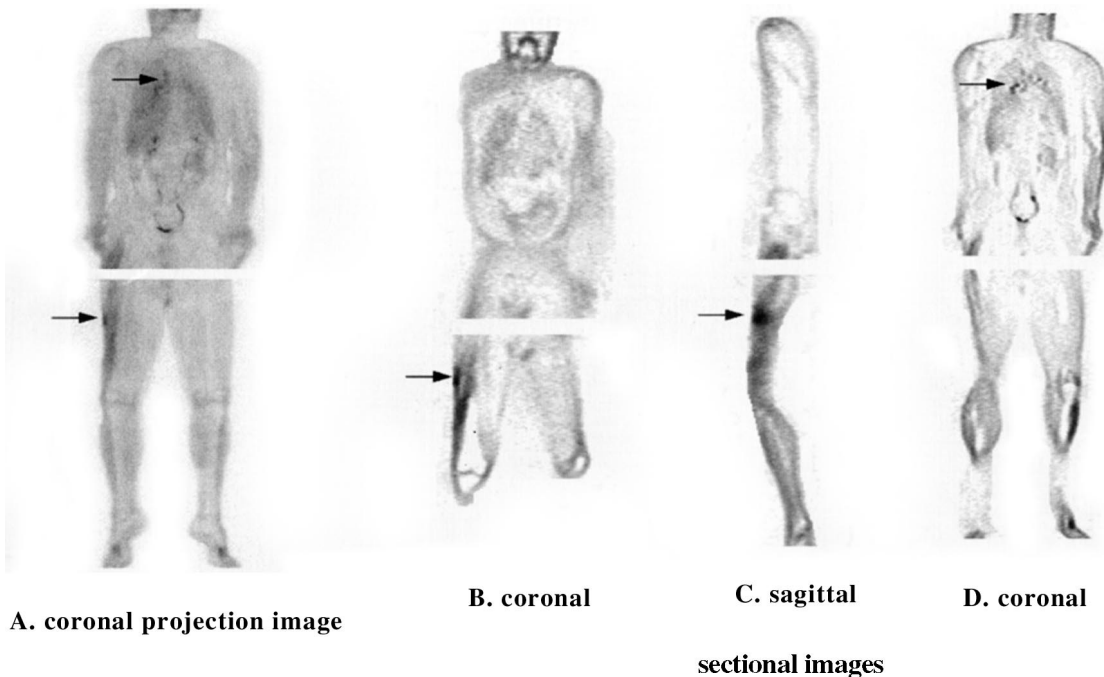


FIGURE 13. Case example, muscle and connective tissue tumors. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.

Key Management Issues.

- Following up sarcoma treatment
- Grading sarcoma
- Separating benign from malignant masses
- Selecting biopsy sites
- Assessing extent of sarcomas

Summary of Evidence for FDG PET in Muscle and Connective Tissue Tumors. Management change data for diagnosis and staging and other applications are not directly available from the literature (Table 12).



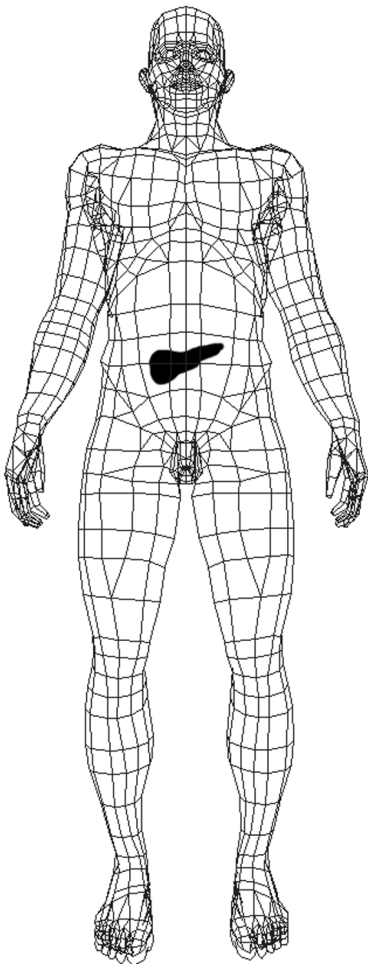
TABLE 12
FDG PET in Muscle and Connective Tissue Tumors: Results of Literature Search

MUSCLE&CONNECTIVE TUMOR	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT(%)/EFFECT
Diagnosis																
Schwarzbach, 2000 ¹	RA	pre-op assessmt STS/susp prim susp local recurr	50	59	91	88	88	88							histopath/surg/follow-up	
Dimitrakopoulou-Strauss, 2000 ²	A	dx/prim/recur/STS	50	59	88	92	92	92							not stated	
Lucas, 1999 ³	RA	eval soft tissue masses/sarcoma/qualitative quantitative/SUV cutoff 2.0	30	31	91	88	88	88	91	88	88	89	89	89		
Gauthier, 1999 ⁴	A	assessmt/progress dis/soft tissue sarcoma	18	18	90	63	63	63	75	83	78	78	78	78	follow-up(av9mo)/histol	
Schwarzbach, 1999 ⁵	RA	prim susp loc recurr	14	14	100	100	100	100							surg_pathol/follow-up	
Schulte, 1999 ⁶	RA	eval of soft tissue tumors	102	102	97	66	66	66	84	92	86	86	86	86	biopsy/histol	
Nieweg, 1996 ⁷	RA	defect STS	22	21	100	67	67	67							histopath/biopsy	
	Summary	by patients	286	374	94	72	72	72	84	90	85	85	85	85		
Staging																
Lenzo, 2000 ⁸	A	staging/overall dx/prim response to their/poor LT response response to their/good LT prognosis	20	20	93	88	88	88							anat_imag/clin_hist/LT_follow-up(-3.3y)	
Lodge, 1999	RA	assess tumor malignancy	29	29	100	76	76	76							biop/histol/surg excis	
Lodge, 1998	A	differentiating STS from benign	27	27	82	94	94	94							biop/histol	
	Summary	by patients	76	84	91	85	85	85								
Dx/Staging																
Dimitrakopoulou-Strauss, 2000 ⁹	A	dx/skeleton sys/sp_occ lesions	83	55	90	88	88	88	100	90	90	90	90	90	histol	
	Summary	by patients	83	55	90	98	98	98	100	90	90	90	90	90		

TABLE 12 (Continued)

MUSCLE&CONNECTIVE TUMOR	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(% EFFECT)
					SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)		
Recurrence																
Jacobson, 2000 ¹⁰	A	recurrence dx/prim/recr/STS	95	177	47	93	62	88	84					clin/imag/histopath		
Dimitrakopoulou-Strauss, 2000 ¹¹	A	prim recurr	50	19	91	88	91	88	89					not stated		
Hain, 1999 ¹²	RA	eval local recurr/ampu/soft tissue sarcoma	16	31	88	92								clin follow-up (up to 8 y)		
Schwarzbach, 1999 ¹³	RA	prim susp loc recur	14	14	100	100								surg pathol/follow-up		
Lucas, 1998 ¹⁴	RA	soft tissue sarcoma/local recurrence lung mets	62	72	74	94	82	91	89					MRI/CT/histo/biopsy/follow-up 3y2mo		
Kole, 1997 ¹⁵	RA	detect local recurrence	17	70	87	100	100	96	100	97	97			biop/pathol/clin follow-up 6 mos		
Monitoring Response																
Lenzo, 2000 ¹⁶	A	staging/overall dx/prim response to ther/poor LT response response to ther/good LT prognosis	20	20	93									anat imag/clin hist/LT follow-up(-3.3y)		
van Ginkel, 1996 ¹⁷	RA	hypertherm rpsnse/loc limb perfus/loc adv STS	20	19	88									surg/pathol		
Other																
No articles																
¹ 50 pts. 59 masses. ² PET is helpful for the Dx of prim & recur STS. There is corrol of FDG uptake and grading which can result in FN for G I tumors. ³ Abst did not provide enough data to complete 2x2 and verify spec--92. ⁴ With hi NPV, PET can help clinician in mgmt of pts for earlier dx of progressive disease. ⁵ FDG PET is suitable for functional imaging of soft tissue sarcomas and detecting sarcoma recurrence. ⁶ 102pts/102soft tiss tumors/35 benign tumors(25 benign, 10 tumor-like lesions)/67 malig tumors (66 sarcoma, 1 NHL). ⁷ 22 pts. 18 pts w STS. 4 pts w benign lesions. 1 benign lesion read as equivocal; not included in analysis.18TP/1FP/2 TN. ⁸ FDG is useful for staging & monitoring response to therapy in childhood STS. Promise for detecting occult met disease. Persistent FDG post-ther indic of poor lt prognosis. Neg FDG dur or post-ther suggest lt dis free surv. Overall stated sens=93(18pts/21scans). Calcs to be 18/20=90. ⁹ The analysis of the dynamic FDG data offers a hi acc for differential Dx of space occupying lesions of skeleton system. ⁹ Sarcoma(122scns/47pts)NHL(26scns/21pts)HL(21scns/18pts). ¹⁰ Sarcoma(122scns/47pts)NHL(26scns/21pts)HL(21scns/18pts) + other cancers. ^{2x2} is based on 177 scans. ¹¹ PET is helpful for the Dx of prim & recur STS. There is corrol of FDG uptake and grading which can result in FN for G I tumors. ¹¹ Abst did not provide enough data to complete 2x2 and verify spec--92. ¹² Of 16 pts, only 2 w recur. Many FPs in lower limb amputees persisting >=18 mos; many due to prosthesis pbms. ¹³ FDG PET is suitable for functional imaging of soft tissue sarcomas and detecting sarcoma recurrence. ¹⁴ 62 pts. 72 comparisons for local recurrence. 70 comparisons for lung mets. ¹⁵ PET:14TP/1FN/2TN. CT/MRI:12TP/3FN/2FP. ¹⁶ FDG is useful for staging & monitoring response to therapy in childhood STS. Promise for detecting occult met disease. Persistent FDG post-ther indic of poor lt prognosis. Neg FDG dur or post-ther suggest lt dis free surv. Overall stated sens=93(18pts/21scans). Calcs to be 18/20=90. ¹⁷ Overall 17/19 responses correctly indicated by PET.																

Pancreatic Cancer



Disease Background. Pancreatic carcinoma is common in the United States, with approximately 30,000 patients each year diagnosed with pancreatic adenocarcinomas. Patients with inflammatory processes in the pancreas

(pancreatitis) but no cancer can sometimes have high FDG uptake that is indistinguishable from cancers and, thus, must be differentiated from patients with cancer. FDG PET is being applied increasingly in pancreatic cancer diagnosis. Considering the very poor prognosis of pancreatic carcinomas, PET's greatest role may prove to be in helping to characterize masses appearing in the pancreas, as opposed to more general tumor staging. This is an active area of current investigation.

Case Example. A 52-y-old woman with a calcified pancreatic mass on CT (Fig. 14A, arrow) was referred for FDG PET scanning because of rising blood tumor markers. No uptake of FDG was seen within the mass (Fig. 14B). The patient was treated conservatively, under the assumption that she had inflammation of the pancreas (pancreatitis). Follow-up over 2 y with CT revealed no changes, indicating that FDG PET was correct and no tumor existed.

Why Did FDG PET Help? FDG PET demonstrated that there was no pancreatic tumor, sparing the patient pancreatic surgery.

Key Management Issues.

- Differentiating chronic pancreatic masses from cancer
- Staging nodal and liver metastases
- Assessing response to chemotherapy

Summary of Evidence for FDG PET in Pancreatic Cancer. For diagnosis: An estimated 50% change was noted in management effect, based on 26 patient studies (Table 13).

For diagnosis/staging: An estimated 43% change was noted in management effect, based on 65 patient studies (Table 13).

For staging: An estimated 36% change was noted in management effect, based on 33 patient studies (Table 13).

For recurrence: An estimated 53% change was noted in management effect, based on 19 patient studies (Table 13).

For monitoring response: An estimated 16% change was noted in management effect, based on 19 patient studies (Table 13).



A. CT



B. FDG PET

FIGURE 14. Case example, pancreatic cancer. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.

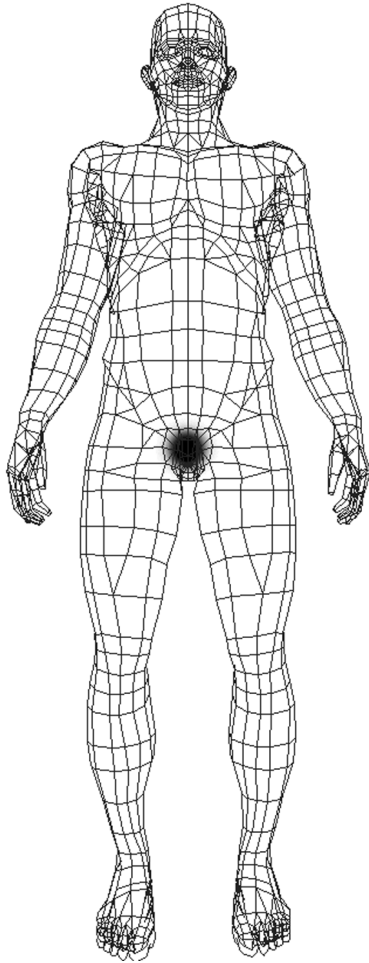
TABLE 13
FDG PET in Pancreatic Cancer: Results of Literature Search

PANCREATIC CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	SPC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT EFFECT (%)
Diagnosis																		
Nakamoto, 1999 ¹	RA	dx/liver mets	34	34	51	92	69	91	100	85	95	100	91	87	90	82	histol/surg/clin follow-up	
Imdahl, 1999 ²	RA	dx	48	48		90	81	91	100	93	100	91	87	71	90	82		
Nitzsche, 1997 ³	A	differential diagnosis	30	26		96	81	100	89	100	91	94	94	76	100	50	histol/surg/endosc cholang/CT	
Chierichetti, 1997 ⁴	A	differential diagnosis	57	12		92												
Friess, 1995 ⁵	RA	dx	80	80		94	79	88	69	91	77	98	71	91	74		histology	
Inokuma, 1995 ⁶	RA	dx	46	46		94	89	82	73	94	91	82	67	91	85		histol/surg/clin follow-up	
Stollfuss, 1995 ⁷	RA	dx	73	73		95		90		93	90	90	93	93	82		histol/surg/biop	
				68			80	74			83		71		78			
Dx/staging																		
	Summary	by patients	368	387	51	94	82	90	75	93	84	92	71	92	78			50
		by lesions				90	69	91	100	93	100	87	71	90	82			
Martin, 2000 ⁸	A	dx/staging	49	49		77		74		82		67					tiss dx/explor lapar/CT/clin	
Debelke, 1999 ⁹	RA	dx/staging	65	65		92	65	85	61	96	87	73	31	91	65		histol/clin & radiol follow-up	43
Zimny, 1997	RA	dx/staging	106	106		85		84		93		71		85			histol	
		lymph node involvement		26										46				
		metastases		31										52				
Bares, 1996 ¹⁰	RA	dx/staging	85	85		85		77		87		74		82			histopath/surg	
		lymph node metastases		31		61												
		liver metastases		13		54												
Bares, 1994	RA	different dx/susp pancr ca	40	40		93		85		93		85		90			histol	
		lymph node mets		17		76		18				67		93			histol/surg	
Bares, 1993	RA	dx/staging/detect pancr mass	15	15		92		100		100		67						
Staging																		
	Summary	by patients	360	461	17	83	65	82	61	91	87	73	31	81	65			43
		by lesions				76	18											
Frank, 1999 ¹¹	RA	follow-up/pancreatic ca	19	19													surg/biop/clin follow-up	53
Fröhlich, 1999 ¹²	RA	staging	168	168		68		95		65		95		91			surg/CT follow-up 6 mos	
		>1cm		29		97												
		≤1cm		37		43												
Jadvar, 1999 ¹³	A	impact to mgmt/all stages	14	14		100		67						93			histol/LT clin follow-up 5-17 mos	14
Summary		by patients	201	201	66	70	93	67		65	95	91	91	91	36			
		by lesions																

TABLE 13 (Continued)

PANCREATIC CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT
						CT	PET	CT	PET	CT	PET	CT	PET	CT	PET		
Recurrence																	
Frankle, 1999 ¹⁴	RA	follow-up/pancreatic ca	19	19												surg/biop/clin follow-up	53
	Summary	by patients	19	19													53
Monitoring Response																	
Shields, 1999 ¹⁵	A	eval trtmt rrsnse/local adv	19	19			94									clin follow-up	16
Higashi, 1999 ¹⁶	RA	mon/unresect/pre&post IORT detection of liver mets	12	12			100									surg/compare to CT	
							83		100								
	Summary	by patients	31	45			92		100								16
Other																	
No Articles																	
¹ Data suggests PET as adjuvant modality to confirm liver mets when other modals inconclusive. ² NPV=21/22=95 where art reports .94. ³ All prim lesions were classified correctly on PET. ⁴ To obtain a 100% spec in detecting malignancies w PET, SUV w cut-off of 3 applied w sens=875. 12 pts w pancr ca. Other pts w other maligns and non-malign conditions. ⁵ Art reports PET PPV=91 NPV=98. 2x2 from reported results gives PPV=92 NPV=90. ⁶ Focal increase of FDG uptake seems to be a highly specific sign of malignant tumor. ⁷ Art reports PET NPV=90. This is actually the spec. From 2x2, NPV=93. ⁸ PET alone unable to eval extrapancreatic (Stig II) & nodal involvmt (Stig III) or accurately identify confounding bowel activity. ⁹ PET values listed are from cutoff level of SUV 3.0. Optimal cutoff value to differentiate benign from malign was 2.0. ¹⁰ Use of PET in classifying pancreatic masses may lead to decrease in unnecessary laparotomies in pts w benign disease. ¹¹ 10/19=53% where PET gave addnl info to clinicians which changed therap proced. In 5/19=26% PET directed to locoreg chemo. 9 pts non-resect tum dis & 10 pts suspic for tumor recurr after surg. ¹² 66 malign lesions found in 22 pts. 29>1cm & 37<=1cm. ¹³ 2/14=14% redirected to additional chemo. ¹⁴ 10/19=53% where PET gave addnl info to clinicians which changed therap proced. In 5/19=26% PET directed to locoreg chemo. 9 pts non-resect tum dis & 10 pts suspic for tumor recurr after surg. ¹⁵ 3/19(16%) post 1cycle trtmt had surg. Sens=94=15/16 that PET predicted correctly whether resect or not post 1 cycle ther. ¹⁶ Define decr in SUV from before to after IORT as indicative of partial response, then sens of PR=10/10=100 i.e. 10 w PR had decr SUV, 2 w NC had incr'd SUV (due to abscesses). ¹⁶ Follow-up PET after IORT on 12 pts/14 scans [2 pts had 2nd PET after IORT].																	

Prostate Cancer



One out of every six men is at lifetime risk for prostate cancer. Approximately every 13 min, a life is lost to prostate cancer in the United States. African-Americans have the highest prostate cancer incidence rates in the world, exceeding those for white males in the United States by 34%. Prostate cancer mortality rates are two times higher for African-American men than for white American men.

Case Example. A 75-y-old man, who was diagnosed with prostate cancer, was followed by blood levels for prostate specific antigen (PSA, a prostate tumor marker). A rising PSA was followed up with a CT scan (Fig. 15, left), which revealed lymph node involvement in the pelvis near the removed prostate. An FDG PET study confirmed what was seen on CT and, in addition, showed spread of cancer into the abdomen and chest (Fig. 15, middle and right).

Why Did FDG PET Help? FDG PET helped because it showed that the cancer had spread to distant sites and that local radiation to the pelvis alone was not likely to benefit the patient.

Key Management Issues.

- Further evaluation of equivocal bone lesions found with conventional imaging
- Differentiating benign from malignant lesions in bone
- Assessing treatment response when lesion is imaged initially
- Identifying metastatic disease in soft tissue

Summary of Evidence for FDG PET in Prostate Cancer. Management change data for staging patients are not directly available from the literature (Table 14).

Disease Background. Prostate cancer rates increased 141.8% between 1973 and 1994. In 1998, new prostate cancer cases totaled 184,500, or, in other terms, one new case every 3 min. Prostate cancer continues to be the most frequently occurring malignancy (aside from skin cancers), representing 29% of all new cancer cases in American men.

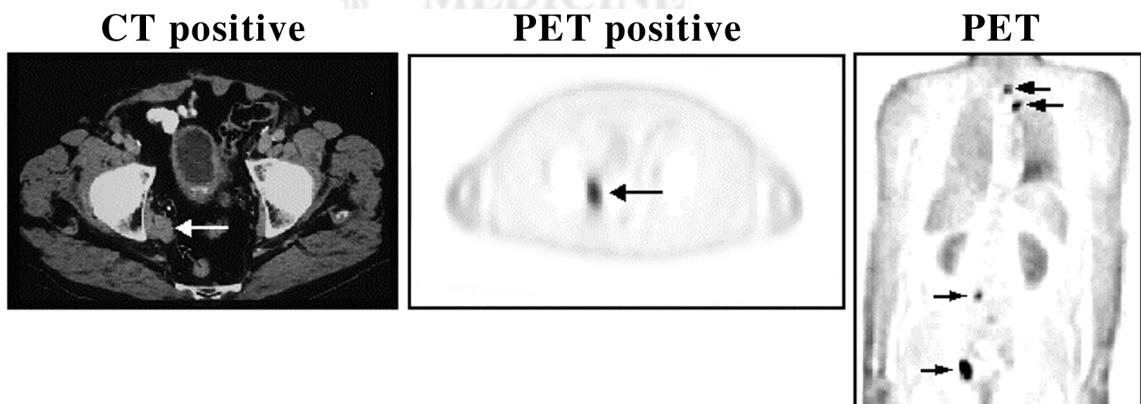


FIGURE 15. Case example, prostate cancer.

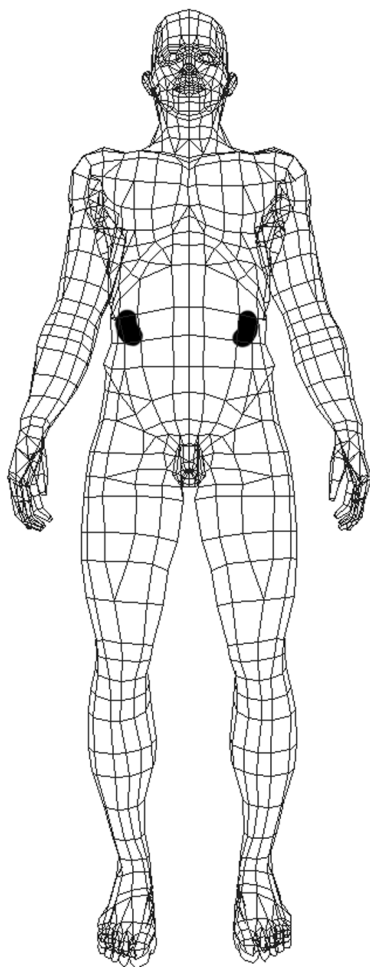
TABLE 14
FDG PET in Prostate Cancer: Results of Literature Search

PROSTATE CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	_MGMT(%) EFFECT
Diagnosis No Articles																	
Staging																	
Seltzer, 2000	A	prostate region advanced prostate	22	5		0										not stated	
Sanz, 1999	RA	rising PSA and/or refrac horm staging iliac & obturator LNS	21	11		12				73						surg pathol CT/bne scint	
Oyama, 1999 ¹	RA	post-trtm/biochem progress/recurr	49	49		64		100		43						histopath/bne scint	
Oyama, 1999 ²	A	untreated pca bone mets	11	12		75										histol	
Melchior, 1999 ³	A	trtm rsnse/endocrine ther	32	11		91										seum PSA/pelv MRI ev 3 mo	
Heicappell, 1999	RA	trtm rsnse/adv/andro with T3 tumors/adv	17	16		81										histol	
Hara, 1998	RA	staging pelv lymph nodes	10	10		67										histol	
Caputo, 1997	A	p cal/mag pelv to lowabd staging bone mets	21	21		50										compare w bone scan	
Shreve, 1996 ⁴	RA	staging soft tiss mets	34	21		40											
Hoh, 1996	A	staging of osseous mets	8	8	202	65			98							bne scan/CT/clin follow-up	
Yeh, 1995	A	trtm rsnse/partial/adv dis trtm rsnse/stable clin dis	8	8		100										PSA deers-50%/meas dis >50% dear	
	A	staging bony mets/horm resis	11	11		75										compare w Tc-99m-MDP	
	Summary	by patients by lesions	244	227	202	57	100			73							
Dx/Staging No Articles																	
Recurrence																	
Seltzer, 2000	A	prostate region advanced prostate	22	5		0										not stated	
Sanz, 1999	RA	rising PSA and/or refrac horm staging iliac & obturator LNS	21	11		12				73						surg pathol CT/bne scint	
Seltzer, 1999	RA	post-trtm/biochem progress/recurr dist mets/PSA relapse/PSA-4 PSA-4	45	22		50	50			43						CT/monoclonal antibody/FNA	
Jacobson, 1999 ⁵	A	eval LN mets	11	12		4	17										
Haseman, 1996	RA	dx recurr/lev tum markers occult recurr/lev PSA 3mo post ther	14	10		67										radial exam/histol biop prostate bed/CYT-356	
	Summary	by patients	113	121	202	26	33			33							

TABLE 14 (Continued)

PROSTATE CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	SENS		SPEC		PPV		NPV		ACC		GOLD	MGMT(%) EFFECT
						SENS (%)	CT (%)	SPEC (%)	CT (%)	PPV (%)	CT (%)	NPV (%)	CT (%)	ACC (%)	CT (%)		
Monitoring Response																	
Meichior, 1999 ⁶	A	trtmt resp/adv/andro with T3 tumors/adv	32	16	81											serum PSA/peiv MRI ev 3 mo	
Oyama, 1999 ⁷	A	trtmt rspnse/endocrine ther	11	11	91											histol	
Hoh, 1996	A	trtmt rspnse/partial/adv dis	8	8	100											PSA dect>50%/meas dis >50% dect	
		trtmt rspnse/stable clin dis	8	8	75												
	Summary	by patients	59	43	86												
Other																	
	No articles																
¹ 44 pts w histol proven pca. 5 benign control pts w BPH. ² Glucose util may be independent fm PSA or prost size for eval of endocrine ther. ³ Serial assessmt by FDG PET may be useful for optimizing neoadjuvant or intermittent hormonal ther. ⁴ FDG PET can help identify osseous and soft-tissue mets w hi PPV. Less sens than brn scint for osseous mets. ⁵ Yields for prostate pts w elev Tg. C, PSA are prob lower due to lower metabolic activ. ⁶ Serial assessmt by FDG PET may be useful for optimizing neoadjuvant or intermittent hormonal ther. ⁷ Glucose util may be independent fm PSA or prost size for eval of endocrine ther.																	

Renal Cell Cancer



Disease Background. Renal cell cancer, also called renal adenocarcinoma or hypernephroma, can often be cured if diagnosed and treated when still localized to the kidney and to immediately surrounding tissue. The probability of cure is directly related to the stage or degree of tumor dissemination. Even when regional lymphatics or blood vessels are involved with tumor, a significant number of patients can achieve prolonged survival and probable cure. When distant metastases are present, disease-free survival is poor, although occasionally, patients will survive after surgical resection of all known tumor. Because a majority of patients are diagnosed when the tumor is still relatively localized and amenable to surgical removal, approximately 40% of all patients with renal cancer survive 5 y. Occasionally, patients with locally advanced or metastatic disease may exhibit indolent courses lasting several years. Late tumor recurrence many years after initial treatment occurs occasionally. Renal cell cancer is one of the few tumors in which well-documented cases of spontaneous tumor regression in the absence of therapy exist, but this occurs very rarely and may not lead to long-term survival. Surgical resection is the

mainstay of treatment of this disease. Even in patients with disseminated tumor, locoregional forms of therapy may play an important role in palliating symptoms of the primary tumor or of ectopic hormone production. Systemic therapy has demonstrated only limited effectiveness.

Case Example. A 59-y-old man with a history of metastatic renal cell cancer and left kidney removal developed left-sided flank pain. Abdominal-pelvic CT was negative on initial review (Fig. 16, bottom row). FDG PET revealed a focus in the apex of the right lung and in the left flank (Fig. 16, top row, arrows). Because of the abnormality in the region of the left flank, the CT was reviewed again and a mass located in the posterior abdominal wall was found. Biopsy revealed metastasis from renal cell cancer.

Why Did FDG PET Help? FDG PET showed a lesion missed on CT and also showed that the renal cell cancer had spread to the lungs. The patient, therefore, could be managed better with systemic therapy and, because of the spread of the disease, was not likely to do well.

Key Management Issues.

- Detecting metastatic disease
- Assessing response of metastases to chemotherapy
- Determining nature of renal masses

Summary of Evidence for FDG PET in Renal Cell Cancer. Management change data for diagnosis and staging and other applications are not directly available from the literature (Table 15).

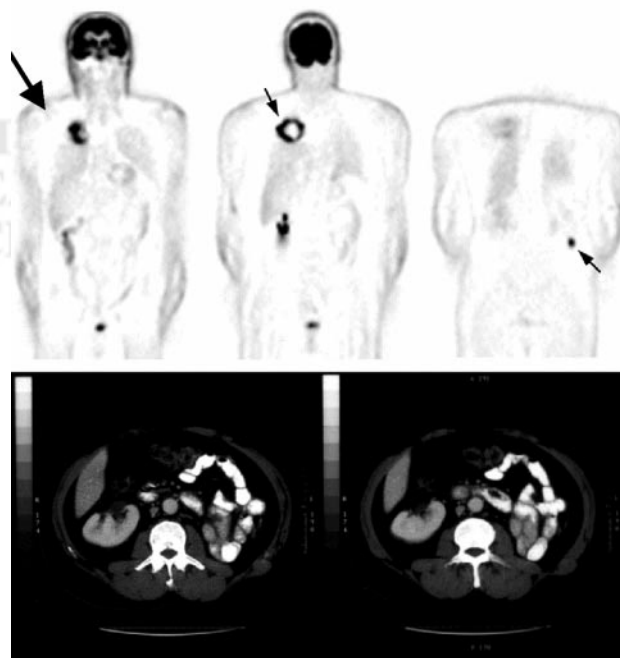
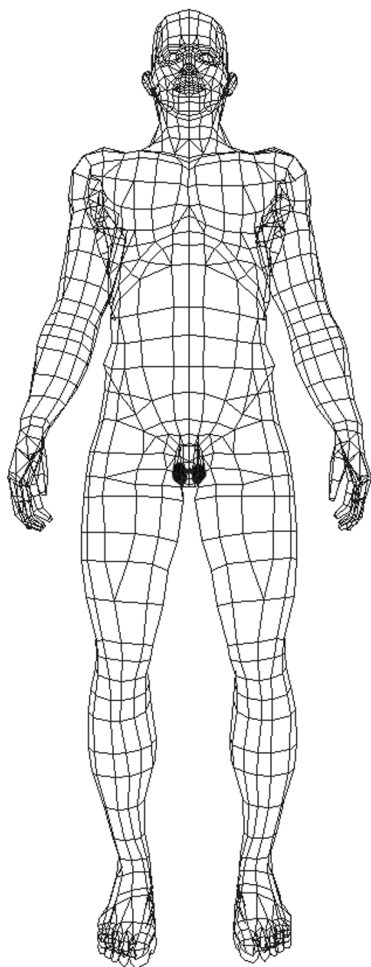


FIGURE 16. Case example, renal cell cancer.

TABLE 15
FDG PET in Renal Cancer: Results of Literature Search

RENAL CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT
					PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)		
Diagnosis																
Bachor, 1995	RA	primary dx	11	11	89										surg	
	Summary	by patients	11	11	89											
Staging																
Harrison, 2000 ¹	RA	pre-op staging/adrenalect	8	4									100		surg/CT/follow-up 43 mos	
Lang, 2000	A	staging	46	46	74										clin follow-up	
Bachor, 1996	RA	staging	29	29	77										surg/histol	
Hoh, 1996	A	staging	22	22	80										clin follow-up/ CI	
	Summary		105	101	76								100			
Dx/Staging																
No Articles																
Recurrence																
No Articles																
Monitoring Response																
No articles																
Other																
No articles																
¹ 14 pts evaluated w PET. 2 pts showing isol mets/successful surg. 2 pts showing dissem mets/death.																

Testicular Cancer



abdomen. Furthermore, the tumor did not appear to have spread elsewhere. Knowing the presence of tumor in that region changed management for the patient, who would have received only testicular surgery but now could receive additional treatment for spreading testicular cancer.

Key Management Issues.

- Monitoring response to treatment
- Staging of primary disease
- Assessing residual mass
- Further evaluating raised markers

Summary of Evidence for FDG PET in Testicular Cancer. For staging: An estimated 22% change was noted in management effect, based on 27 patient studies (Table 16).

For recurrence: An estimated 51% change was noted in management effect, based on 53 patient studies (Table 16).

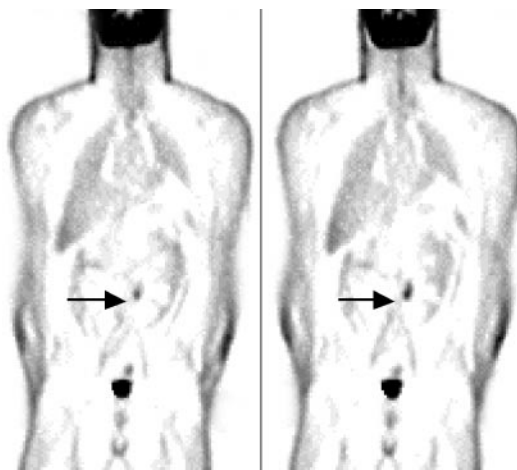


FIGURE 17. Case example, testicular cancer.

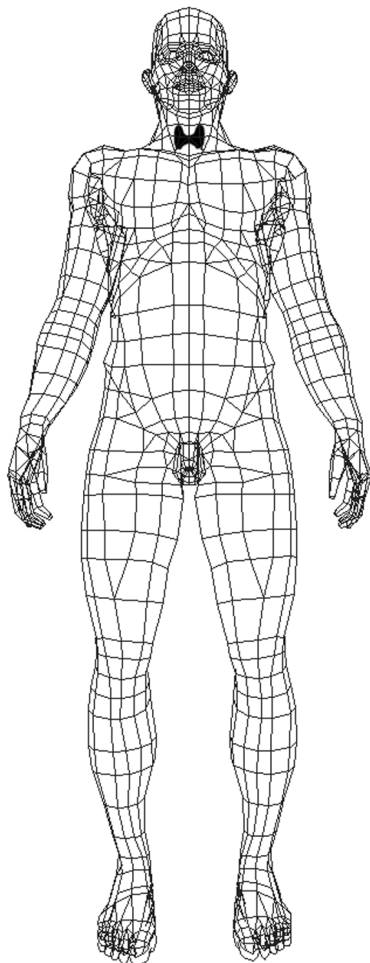
Disease Background. Cancer of the testicle, a rare type of cancer, is a disease in which cancer cells are found in the tissues of one or both of a man's testicles. Cancer of the testicle is the most common cancer in men 15–35 y old. Men who have an undescended testicle (a testicle that has never moved down into the scrotum) are at higher risk of developing cancer of the testicle. This is true even if surgery has been performed to place the testicle in the appropriate place in the scrotum. Prognosis and choice of treatment depend on the stage of the cancer and the patient's general state of health.

Case Example. A 27-y-old man with testicular cancer had his left testicle removed. An abdominal CT scan indicated an enlarged lymph node in the lower abdomen. A CT-guided biopsy was performed but did not reveal cancer. An FDG PET scan was ordered to make sure the biopsy was not wrong. The scan showed a focus of activity in the abdominal lymph node (Fig. 17, arrows), suggesting cancer spread. A repeat biopsy confirmed tumor in the abdominal lymph node site.

Why Did FDG PET Help? FDG PET showed that the biopsy was wrong, and, in fact, tumor was present in the

TABLE 16
FDG PET in Testicular Cancer: Results of Literature Search

TESTICULAR CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS		SPEC		PPV		NPV		ACC		GOLD	MGMT(% EFFECT)
					PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)		
Diagnosis																
No articles																
Staging																
Albers, 1999	RA	staging	37	37	70	40	100	78								
Cremerius, 1999	RA	staging	50	50	87	73	94	94	87	85	94	89	92	68	LN dissect/clin follow-up	
Hain, 1999	A	staging	27	27											clin follow-up/retroper lymphad	
Nuutinen, 1996	A	metastatic cancer	13	13	100		89								histol/clin follow-up	22
Harms, 1995	A	metastatic cancer	29	29	79		90								histol/surg/follow-up	
	Summary	by patients	156	156	82	59	94	87	87	85	94	89	92	68	clin course/histol	22
Dx/Staging																
No articles																
Recurrence																
Hain, 1999	A	recurrence	53	53											biop/follow-up	51
	Summary	by patients	53	53												51
Monitoring Response																
No articles																
Other																
de Wit, 1999	A	germ cell tumor	133	133	80	88	92	48	88	57	86	83	87	66	histol/clin follow-up	
	Summary	by patients	133	133	80	88	92	48	88	57	86	83	87	66		66



Disease Background. Cancer of the thyroid is a disease in which cancer cells are found in the tissues of the thyroid gland. People who have been exposed to large amounts of radiation or who have had radiation treatment for medical problems in the head and neck have a higher chance of getting thyroid cancer. The cancer may not occur until 20 y or longer after radiation treatment. The four main types of cancer of the thyroid are: papillary, follicular, medullary, and anaplastic. The chance of recovery depends on the type of thyroid cancer, whether it is only in the thyroid or has spread to other parts of the

body (stage), and the patient's age and overall health. Some types of thyroid cancer grow much faster than others. Although thyroid cancer is relatively uncommon, it is nonetheless the most common malignancy of the endocrine system. Differentiated tumors (papillary or follicular) are highly treatable and usually curable. Poorly differentiated cancers (medullary or anaplastic) are much less common but aggressive, metastasize early, and have a much poorer prognosis. The incidence of this malignancy has been increasing over the last decade. The prognosis for differentiated carcinoma is better for patients younger than 40 y and who have no extracapsular extension or vascular invasion. Age appears to be the single most important prognostic factor. Thyroid cancer commonly presents as a cold nodule within the thyroid gland. The overall incidence of cancer in a cold nodule is 12%–15% but is higher in patients younger than 40 y.

Case Example. A 62-y-old patient underwent surgery of the left thyroid for thyroid cancer. Routine yearly monitoring revealed elevated blood levels of calcitonin. A CT scan was ordered and was normal. An FDG PET scan revealed increased FDG accumulation in the neck (Fig. 18, arrows), which was confirmed by biopsy to be residual thyroid cancer.

Why Did FDG PET Help? FDG PET identified the source of the rising tumor marker and thereby allowed removal of residual thyroid cancer.

Key Management Issues.

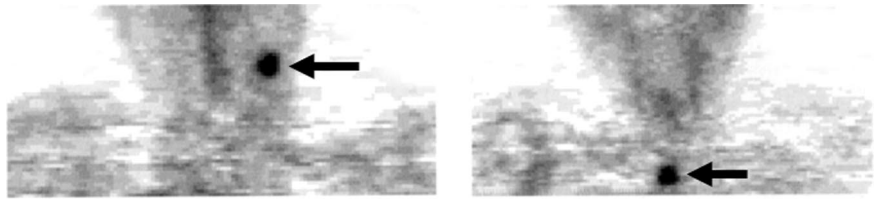
- Further evaluation when whole-body ^{131}I scan is negative but thyroglobulin (Tg) levels are rising in a patient with known differentiated thyroid cancer
- Further evaluation for medullary thyroid cancer when rising calcitonin level and initial imaging with dimer-captosuccinic acid V, octreoscan, or metaiodobenzylguanidine is negative.

Summary of Evidence for FDG PET in Thyroid Cancer.

For staging: An estimated 22% change was noted in management effect, based on 60 patient studies (Table 17).

For diagnosis/staging: An estimated 9% change was noted in management effect, based on 58 patient studies (Table 17).

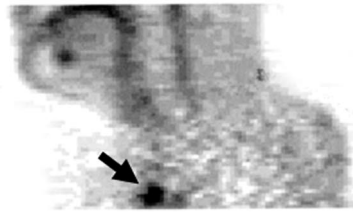
For recurrence: An estimated 53% change was noted in management effect, based on 21 patient studies (Table 17).



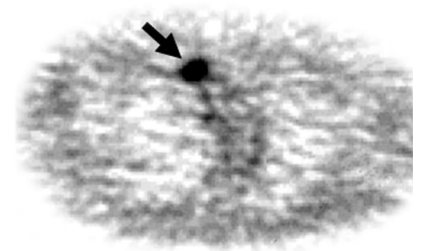
A.

B.

coronal



C. sagittal



D. transaxial

FIGURE 18. Case example, thyroid cancer. Reproduced, with permission, from Maisey et al. *Atlas of Clinical Positron Emission Tomography*. London, UK: Arnold, Hodder Headline Group; 1999.



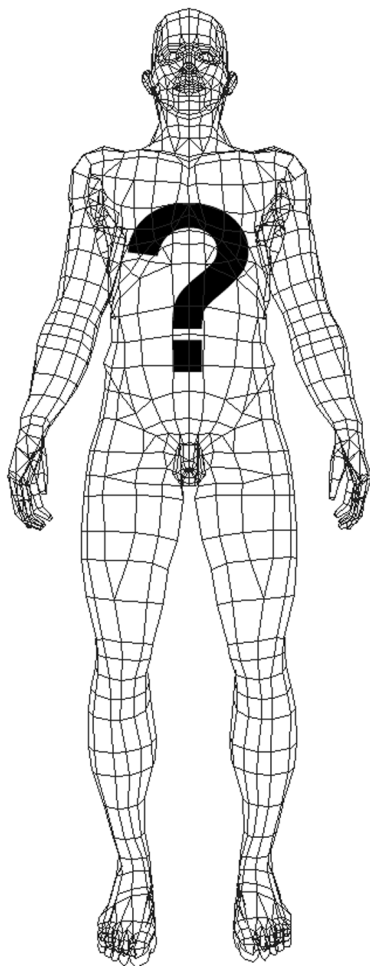
TABLE 17
FDG PET in Thyroid Cancer: Results of Literature Search

THYROID CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)	GOLD STD	MGMT(%) EFFECT
							SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)			
Diagnosis No articles																	
Staging																	
Brandt-Mainz, 2000	RA	follow-up/MTC/elev calcit/overall neck mets	20	18	14		76									histol/CT/venous cathet	
		mediastinal mets			8		86										
		pulm mets			3		100										
		bone mets			2		100										
Kurtaran, 2000 ¹	A	recurrent medullary thyroid carcin	20	20			32									CT/US/MRI/histol	
Shanker, 2000 ²	A	follow-up/iodine-neg thyroid ca	23	27			89	88								serum thyroglob&antibod/neck US&MRI	
Hoda, 2000 ³	A	detect recur/met papillary thyrd ca	11	11			91									IWBS/serum Tg assay	
Liu, 2000	A	detect recur & mets/thyroid ca	19	19			79									clin/path follow-up	
Wang, 1999	RA	resid thyrd ca/131I-elev Tg hi Tg	37	18			71			92						all imaging/serological study/biopsy	51
		lb Tg		19			67	81		40			93				
Jacobson, 1999	A	dx recur/thyrd/elev markers	60	26			35									radiol exam/histopath	
Grönwald, 1999	RA	differentiate thyroid carcinoma	222	222			75	90		88			79			histol/cytoI/thyroglob/US/CT/clin	83
Schluter, 1998 ⁴	RA	early surg/131I- differen thyrd carcin	60	60												surg	22
Dietlein, 1998	RA	follow-up/differen thyrd ca/all mets LN mets	50	50			50									MBI-scintig/MRI/lung CT	
		local recurrence		50			60									histol/iodine-uptake	
		pulm mets <1cm		50			100									histol/morphol imaging	
				50			0									spiral-CT/rising Tg-levels	
Summary		by patients	522	640			69	89		85			80				84
		by lesions			27												
Dx/Staging																	
Dietlein, 1997 ⁵	RA	dx/staging/follow-up to trimit subgp/finer Tg level/meg WBS	58	58			50									histol/anatom imag/hod dissect	9
		LN mets			41		82										
Bloom, 1993 ⁶	RA	Dx/eval thyroid nodules	19	19			49									surg	100
Adler, 1993 ⁷	RA	Dx/susp thyrd nodule/pre-op	9	9			100	67								surg excis	
Summary		by patients	86	144			68	67									100
		by lesions			41		49										9

TABLE 17 (Continued)

THYROID CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	Non-Ded PET	SENS (%)	SPEC (%)	PPV (%)	NPV (%)	ACC (%)	GOLD STD	MGMT(%) EFFECT
							CT	CT	CT	CT	CT		
							PET	PET	PET	PET	PET		
							(%)	(%)	(%)	(%)	(%)		
Recurrence													
Altafisi, 2000 ⁹	RA	recurr or met 131I neg papill thyrd ca	11	11					64			CT/sonog/biop nk lesions	64
Kurtaran, 2000 ⁹	A	recurrent medullary thyroid carcin	20	20			32					CT/US/MRI/histol	
Shankar, 2000 ¹⁰	A	follow-up/iodine-neg thyroid ca	23	27			89	88			89	serum thyroglob&antibod/neck US&MRI	
Hoda, 2000 ¹¹	A	detect recurr/met papillary thyrd ca	11	11			91					IWBS/serum Tg assay	
Liu, 2000	A	detect recur & mets/thyroid ca	19	19			79					clin/pathol follow-up	
Chung, 1999	RA	athyrotic papill thyrd ca/131 I -	54	54			94	95	97	91	94	pathol/other imag/clin crse/dissect	
Jacobson, 1999	A	dx recurr/thyrd/elev markers	60	26			35					radiol exam/histopath	
Grünwald, 1999	PA	differentiate thyroid carcinoma	222	222			75	90	88	79	83	histol/cytol/thyroglob/US/CT/clin	
Stokkel, 1999	PA	recurr/rising Tg level/neg I-131 WBS	11	11		yes	100	100			100	US/CT/MRI/FNA cyt	40
Jadvar, 1998 ¹²	PA	eval susp recurr papill thyrd carcin	10	10			100	80	83	100	90	US/CT/MRI/FNA nk LN dissect	
Altenvoerde, 1998 ¹³	PA	differen thyrd carcin/elev Ig-I 131	32	12					50			CT/MRI	
Grünwald, 1997 ¹⁴	PA	differen thyrd carcin/versus WBS	54		66		73	86	81	79	80	hist/cytol/Tg/US/CT/MRI/follow-up	
Feine, 1996 ¹⁵	PA	detect recurr	41	41			89					131-I-WB gamma cam/nk&abd US	
Grünwald, 1996 ¹⁶	PA	susp recurr/incr Tg level/131-IWBS	33	33			83					CT/nTg levels	
Summary													
			601	497	66		77	91	87	82	86		53
							73	86	81	79	80		
Monitoring Response													
No articles													
Other													
No articles													
¹ sens=6/13=32 1FP discovered from histol which reduced 7 TP to 6 TP. And 13 FN. 20 pts. Conclus:PET has to sens in localizing recurr/metast MTC lesions. Should not be performed routinely. ² 23 pts/27 scans. Spec actually rounds up to .89. ³ Not clear from abstr if there was 1 FN or 1 TN. If FN, then sens=10/11 for PET. ⁴ 13/60=22% operated on after positive PET findings. ⁵ Change in mgmt=5/8=9% sent to surg from +PET where -I uptake for LN met pts. ⁶ 19 pre-op pts. 12 pts w solitary thyrd nodules. 7 multinodular goiters. 4 w malign had FDG>8.5. 15 benign w FDG <7.6. Acc=19/19=100. ⁷ Mean FDG DUR for 3 malign lesions signif > 6 benign lesions. 3 malign and 4/6 benign lesions w incr FDG uptake. ⁸ PET redirected trinit in 7/11 pts=64. ⁹ sens=6/13=32. 1FP discovered from histol which reduced 7 TP to 6 TP. And 13 FN. 20 pts. Conclus:PET has to sens in localizing recurr/metast MTC lesions. Should not be performed routinely. ¹⁰ 23 pts/27 scans. Spec actually rounds up to .89. ¹¹ Not clear from abstr if there was 1 FN or 1 TN. If FN, then sens=10/11 for PET. ¹² FDG PET provided additional info in 4/10=40% pts affecting their clinical mgmt. ¹³ 500 pts w diff thyrd carc. Subgp of 32 pts w elev hTg. 1 131, neg US and chest xray. 12 pts w PET scans. ¹⁴ Based on Table 2 results:22TP/5FP/8FN/31TN. ¹⁵ Referred to in Stokkel '99 discussion. ¹⁶ Referred to in Stokkel '99 as sens=83. From abst, sens=82=14/17.													

Unknown Primary Tumor



Disease Background. Detection of the unknown primary lesion is very difficult. In many cases, patients present with obvious metastatic disease, often adenocarcinoma, in which the location of the primary lesion may never be found. In some cases, knowledge of the primary site is important, because the type of treatment may vary (e.g., breast cancers are more responsive to some treatments than are renal cancers). This knowledge also can be helpful in resection or treatment for cure of the primary lesion and metastases (e.g., head and neck cancers). FDG PET is useful in locating primary tumors after metastatic disease has appeared in regional lymph nodes. FDG PET is being applied increasingly in the search for unknown primary lesions. This application is still in evolution, but FDG PET should be considered strongly in the work-up of the unknown primary.

Case Example. A 49-y-old woman presented with lymph node enlargement in the neck. Physical examination, CT, and mammography performed twice all failed to reveal the source of the primary cancer. An FDG PET

study showed that the primary cancer was in the left breast (nonpalpable). The lymph node involvement was not seen in the sections shown in Figure 19.

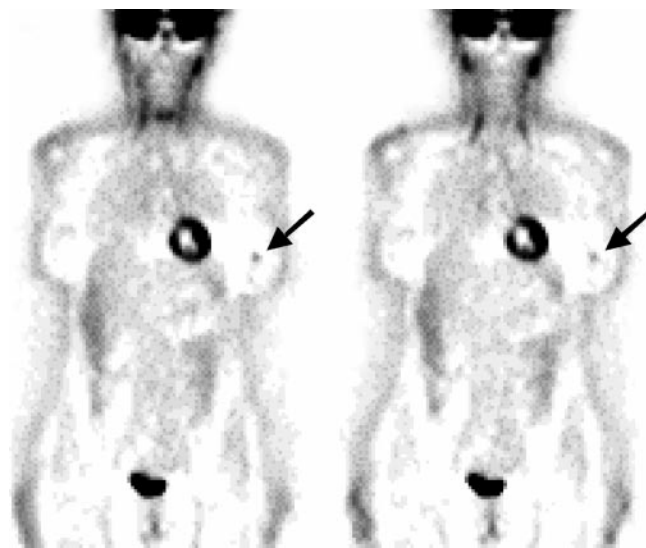


FIGURE 19. Case example, unknown primary tumor.

Why Did FDG PET Help? FDG PET identified the source of the patient's cancer when no other study could do so, thereby allowing the patient to be treated appropriately for breast cancer with spread to the lymph nodes.

Key Management Issues.

- Identifying primary site to determine treatment and evaluate for possible resection

Summary of Evidence for FDG PET in Unknown Primary Cancer. For staging: An estimated 29% change was noted in management effect, based on 285 patient studies (Table 18). (See Table 19 for the results of the literature search on FDG PET in miscellaneous tumors.)

Summary

Table 20 is a summary of results from the literature search on FDG PET in cancer.

TABLE 18
FDG PET in Unknown Primary Cancer: Results of Literature Search

UNKNOWN PRIMARY CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT(%) EFFECT
							CI (%)	CI (%)	CI (%)	CI (%)	CI (%)	CI (%)	CI (%)	CI (%)	CI (%)	CI (%)		
Diagnosis																		
No articles																		
Staging																		
Bohuslavizki, 2000 ¹	RA	detect of unk prim/overall	53	52			100	81			77	100	88				clin/surg/histopath	
Haase, 2000 ²	A	detect unk prim/overall	32	32		yes	80										biop/CT/MRI/OPET follow-up	38
Kuehnel, 2000 ³	A	detect unk prim	34	34						75							histol/clin course	44
Lassen, 1999 ⁴	RA	detect unk prim	20	20			100			69							histol/clin course	15
		detect mets		20														
Safa, 1999 ⁵	RA	detect of unk prim/hd & nk	14	14			75	33	80	60	60	20	89	78	79	57	clin/CT/histopath	21
Hanasono, 1999 ⁶	RA	detect of unk prim/hd & nk	84	20			70	60	60	64	64	67	65	65			CT/MRI	35
Greven, 1999 ⁷	RA	detect of unk prim/hd & nk	13	13			50	45	45	14	83	83	46	46			biop/panendoscopy	8
AAassar, 1999 ⁸	RA	detect of unk prim/hd & nk	17	15			100	63	63	70	70	100	80	80			surg/clin/histopath	53
Lang, 1999	A	detect of unk prim	40	40														28
Kiurmann, 1999 ⁹	A	detect of unk prim/cerv mets	28	28						60							histol/subseq conv imag	32
Yang, 1999 ¹⁰	A	detect of unk prim/cerv mets	8	8			80										physical ex/cheest x-ray/CT/histol	50
Kole, 1998 ¹¹	RA	detect of unk prim	29	29			70										clin follow-up/addit dx study	10
Braams, 1997 ¹²	RA	detect of unk prim/H&N	13	13			80										biop/endosc/histol	30
Kole, 1995 ¹³	A	detect of unk prim	19	19			67										histol/clin follow-up 2-30 mos	21
Hubner, 1995	RA	charact prim unk lung masses	54	23	24		100	67	67	90	90	100					histol/surv/SUV vs DNA prolif index	
		recurr lung carcin/lymphoma		13	19		83	80	80	63	63	91						
		extrathor/susp_pulm mets		18	21		87	83	83	93	93	71						
Summary		by patients	458	411	64		82	33	71	64	66	20	91	78	77	57		29
		by lesions					91	76		83	83	88						
Dx/Staging																		
No articles																		
Recurrence																		
No articles																		

TABLE 18 (Continued)

UNKNOWN PRIMARY CANCER	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Total Lesions	Non-Ded PET	SENS		SPEC		NPV		ACC		GOLD STD	MGMT(%) EFFECT
							PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)		
Monitoring Response																
No articles																
Other																
No articles																
<p>¹ 1 pt denied further workup, so analysis based on 52 pts. 20TP, 6FP, 26TN, 44pts had cerv mets/sextracerv mets.</p> <p>² Sens=12/15=80 based on calling 14 pts w nothing found as TNs. In reality, they may have CUP, but no imaging can find it. Assumption is that for calcs, if isn't found, is considered as TN.</p> <p>Chge in mgmt determined from % pts w discovered unk prim as this affects trtmt given 12/32=38%.</p> <p>³ PET influenced trtmt in 15/34 pts=44%. Data given as 15 TP and 5 FP for PET. So calc'd PPV and not sens, as no results re FNs. And assuming as above, that anything not found is TN.</p> <p>⁴ In 13 pts, PET suggested prim tum which was verified in 9. TP=9/FP=4. Also 2 FN/TN/4 not confirmed. PET directed therapy change in 3/20=15%.</p> <p>⁵ Have listed what is in Table 2 as ratios. Abst agrees w 1 FN. Art also states that 9 pre-trtmt PET negs had neg rmdm biops(which would be 9 TNs). But 1 found to be FN in post-trtmt PET scan and this is in Table.</p> <p>⁶ Chge in mgmt=7/20=35% from detecting unk prim correctly in 7 pts. Art also reports this as detection and sens rte. But states that 10 prims ultimately proven histologically in these 20 pts.</p> <p>So if use 7TPs, 3FNs, 4FPs, and 6 TNs which agree w Table 1, would get sens=7/10=70.</p> <p>⁷ Chge in mgmt=1/13=8% for discovered unk prim. 3 occult prims proven. PET had 1TP, 1FN, 1FP that it picked up in site other than actual location.</p> <p>⁸ 2 lung prims discovered and excluded from rest of analysis. 2x2 based on remaining 15 H&N pts. 7 TP, 3FP, 5TN. Chge in mgmt=9/17=53% (9pts total w lung(2) and H&N(7)prims discovered).</p> <p>. They used same assumptions as above; the 8 pts they consider to still have occult prim disease are still listed as FPs and TNs.</p> <p>⁹ Of 16 PET+, 9 TP, 6FP, and 1 refused further cnfrm. PPV=9/15=60. Chge in mgmt=9/28=32%. 12 PET-, but no info on FN vs TN w LT follow-up.</p> <p>¹⁰ PET tst results reported for 7 pre-surg PET; 1 post-surg PET; 1 post-surg PET no rslt. Sens=4/5=80 based on 4 TP, 2TN, 1FN for 7 pts. Chge in mgmt=4/8=50% for discovered prim tumors.</p> <p>¹¹ They report chge in mgmt for 3 pts and 3/29=10%. For other unk prim studies, #prim discov/tot pts = chge in mgmt as potent for trtmt specific chges. Also, they report sens rte= detect rate=7/29=24%.</p> <p>From 2x2, 7 TP, 3 FN, 19 TN, and sens=7/10=70.</p> <p>¹² PET results reported are 4 TP, 1 FN, 8 TN. Technically, sens=4/5=80. Again, they report detection rate of 4/13=30% which is sens if you consider all 13 as 'known' prims to be detected.</p> <p>But we rpt 2x2 as if assumption is none may ever be found (see other footnotes above).</p> <p>¹³ 9pts. 4TP, 2 FN, 13 TN for primary tumor detection. Chge in clin mgmt=4/19=21%. 2x2 sens=4/6=67. In this example, the 4 pts w chge in mgmt were not all the same 4 that were discovered primaries.</p>																

TABLE 19
FDG PET in Miscellaneous Tumors: Results of Literature Search

Miscellaneous Tumors	ARTICLE TYPE	PURPOSE	Total No. Patients	Total PL Studies	Total Lesions	Non-Ded PET	SENS PET (%)	SENS CT (%)	SPEC PET (%)	SPEC CT (%)	PPV PET (%)	PPV CT (%)	NPV PET (%)	NPV CT (%)	ACC PET (%)	ACC CT (%)	GOLD STD	MGMT(%) EFFECT
Kunkel, 2000 ¹	PA	verification of oral cancer recurr screening for oral cancer recurr	44	23	26		88											morphological correlative
Turtakow, 2000 ²	A	detection of peritoneal carcinomatosis	92	16	10		90											
Kluge, 2000	A	detection of perihilar cholangiocarcinoma	29	29			44											biopsy
	A	detection of perihilar cholangiocarcinoma	29	29			90	88	88	95	78	90						MRI/histol/control gp PET
	A	staging regional lymph nodes	8	8			13											
	A	staging distant mets	6	6			100											
De Winter, 2000 ³	A	dx of chronic osteomyelitis	37	33			100	91	100			94						invasive/clinical
	A	vertebral osteomyelitis	16	16			100	100			100							
Forster, 2000	A	mon thir rsnse/preop irrad/oral cancer	30	30			88	88	94									histology
Bongers, 2000 ⁴	A	detection of recurrent laryngeal cancer	75	75		yes	100	80	88	88	100	92						dx endoscopy/biop
Kato, 2000	A	dx/biliary carcinoma	13	13			70	80	67	67	88	89	40	50	69	77		histopath/cin/abdom CT
Beuthien-Baumann, 2000	A	dx/oral mucosa carcinoma	39	39			95	72										histology
	A	lymph node mets	70				93	87	87	56	98	86						
	A	lymph node mets	59				29		94	36	79	71						
Shulkin, 1999 ⁵	PA	assess sens/pheochromocytomas	29	29			88		42	68	71	69						bne scn/MRI/MBG
Lowe, 1999 ⁶	PA	identify early stge prim & recur laryngeal ca	12	12			92											biop/CT
Myers, 1998	PA	eval NO nk/squamous cell carcin/oral cavity	11	11			100	40	100	88	100	67	100	70	100	69		neck dissection
Schirmeister, 1998 ⁷	A	RNB/bne imag/osteoblastic & osteolytic mets	44	44			82											MRI/131-I scint/xray/CT
Musholt, 1997 ⁸	PA	recur or persist MTC & pheochromocytoma	10		30													surg/pathol
	PA	recur or persist MTC & pheochromocytoma	27				23											
Shulkin, 1995 ⁹	PA	assess FDG uptake in pediatric neoplasms	22	22			81		100	100	20	86						surg/bne scn/CT
Patz, 1995	PA	bronchogenic cal/overall thor nodal staging	42	42	62		83	43	82	85	73	63	89	72	82	69		pathol/CT
	PA	hilat/lobat lymph nodes	40		40		73	27	76	86	53	43	88	76	75	70		
	PA	mediastinal node	22		22		92	58	100	80	100	78	91	62	95	68		
Austin, 1995	PA	dx residual laryngeal carcinoma	10	10			67		57	67	80	60						biopsy/ clin follow-up
Greven, 1994	PA	recur vs irrad sequelae/carcin larynx	11	11			100		80	86	100	91						laryngectomy/CT/pathol
	Summary	by patients	550	496			88	58	81	82	86	69	79	67	88	71		
	Summary	by lesions	346				83	35	85	88	56	52	92	74	83	79		

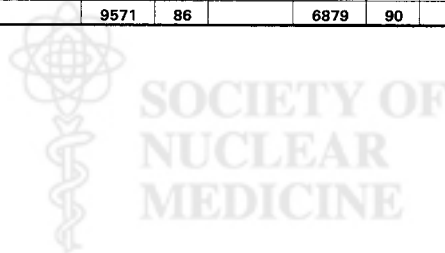
¹44 pts. 50 scans. 23 scans in Gp A/susp recurr. 27 scans in Gp B/screening.
²Pts included stomach,ovarian,adrenal cancers, and mesothelioma.
³From 2x2, sp=90 but is reported as .91. 33pts:13TP,2FP,18TN.
⁴16 pts w vert osteo. 5TP,11TN.
⁵75 pts:45TP,6FP,24FN. Counted init PET+ readings as TP where for 16 pts biop was init neg and became + w LT follow-up.
⁶Ratios based on 29pts:15TP,7FP,2FN,5TN.
⁷12pts:7ND,5Recur. PET:11TP,1FN. CT:2TP,7FN.
⁸44 patients with thyroid, lung, or prostate cancer.
⁹PET+not identified=not resected. FNs determined by surg/follow-up. No info on TNs.PET:16TP,4FP,10FN. CT:6TP,1FP,20FN.
⁹Cases included neuroblastoma,ewing sarcoma,lymphoma,other malignancies.

TABLE 20
FDG PET in Cancer: Summary of Results of Literature Search

CANCER	PURPOSE	Total Pt.	Total	SENS	Total Pt.	Total	SPEC	Total Pt.	Total	ACC	Total Pt.	Total	MGMT
		Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	EFFECT (%)
Bladder	Staging	136		76	98		87	98		83	12		17
	Dx/Staging	52		93	26		86	26		88			
	Recurrence	12		60							12		17
Brain	Dx	36		91									
	Staging	31		86									
	Recurrence	258		79	213		77	161		76	89		31
	Mon Respons	17		82	17		83						
Breast	Other	34		93	19		67	19		84			
	Dx	202		91	97		95	105		95	6		100
			140	90		140	92		105	88			
	Staging	1407		91	1373		88	1328		90	111		24
			242	95		53	88		33	88			
	Dx/Staging	65		75	9		83	52		83			
	Recurrence	414		80	414		85	268		82	23		40
Colorectal			41	85		41	79						
	Mon Respons	206		81	174		96	84		92			
			31	90		31	74						
	Staging										236		36
			24	96									
	Dx/Staging	101		85	87		71	87		94			
	Recurrence	1426		94	1166		87	418		94	915		32
Gastro-Esoph			981	93		912	96		331	87			
	Mon Response												
			34	100		23	90		11	100			
	Dx	120		96				48		98	99		14
Head&Neck			276	80		276	95		276	86			
	Staging	545		73	302		90	245		83	229		20
			15	93									
	Dx/Staging	109		80	109		95	109		86	109		14
	Recurrence	41		100	41		43	41		73			
	Mon Respons	13		100				13		46			
	Dx	129		93	36		70	61		87			
Lung			311	84		267	83		267	85			
	Staging	363		87	279		89	301		88			
			2020	84		1999	95		596	94			
	Dx/Staging	296		88	249		83	184		88	15		33
			179	83		151	94		158	89			
	Recurrence	342		93	271		83	283		87	15		33
			278	84		241	92		241	90			
Hepatocellular	Mon Respons	128		84	122		95	81		96			
			16	44									
	Staging	292		77	249		97	249		93	20		60
	Dx/Staging	22		64									
Lymphoma	Recurrence		9	88									
	Dx	919		96	797		73	719		90			
			278	91		259	68		101	82			
	Staging	1867		83	1495		91	1272		82	1565		37
			1721	83		1553	92		1478	90			
	Recurrence	209		98	193		92	180		96			
			39	100		39	62		39	87			
Melanoma	Mon Respons	161		94	161		90	126		96			
	Other	101		83									
	Dx	11		100									
	Staging	1179		90	826		93	158		88	407		21
			1156	91		58	100		32	95			
Lymphoma	Dx/Staging	254		92	177		93				62		5
	Recurrence	557		87	453		93	155		88	158		10
			114	100									
	Mon Respons	257		90	279		93	13		69			
									32	95			
Lymphoma	Staging	888		83	863		91	125		91	283		26
			899	87		461	68		83	84			

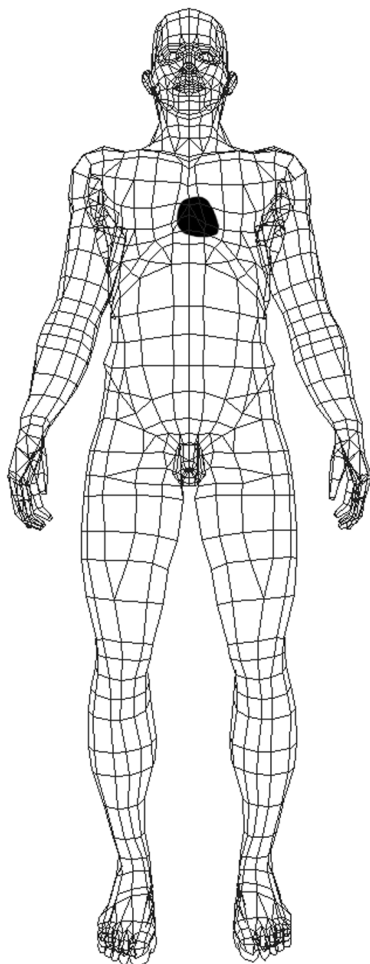
TABLE 20 (Continued)

CANCER	PURPOSE	Total Pt.	Total	SENS	Total Pt.	Total	SPEC	Total Pt.	Total	ACC	Total Pt.	Total	MGMT
		Studies	Lesions	PET	Studies	Lesions	PET	Studies	Lesions	PET	Studies	Lesions	EFFECT
(cont.)				(%)			(%)			(%)			(%)
Muscle&Conn	Dx	274		94	250		72	139		85			
	Staging	84		91	56		85						
	Dx/Staging	55		90	55		98						
	Recurrence	393		66	360		95	346		89			
	Mon Respons	40		86				19		89			
Ovar/Uter/Cerv	Dx	274		66	238		77	153		77			
	Staging	331		54	119		96						
	Dx/Staging	112		86	58		82	73		87			
	Recurrence	359		88	327		90	220		87	30		17
	Mon Respons	11		100				11		100			
Pancreas	Dx	293		94	281		90	259		92	26		50
			51	90		51	91		51	90			
	Dx/Staging	404		83	360		82	368		81	65		43
			17	76									
	Staging	182		70	182		93	182		91	33		36
			66	67									
	Recurrence										19		53
	Mon Respons	45		92	14		100				19		16
Prostate	Staging	196		57	49		100						
			202	65									
	Recurrence	100		26									
	Mon Respons	43		86									
Renal	Dx	11		89									
	Staging	97		76	22		100	4		100			
Testicular	Staging	129		82	129		94	37		92	27		22
	Recurrence										53		51
	Other	133		80	133		92	133		87			
Thyroid	Staging	430		69	268		89	249		84	60		22
	Dx/Staging	125		68	9		67	19		100	58		9
			41	49									
	Recurrence	474		77	324		91	324		86	21		53
			66	73		66	86		66	80			
Unknown Prim	Staging	235		82	114		71	114		77	285		29
			64	91		64	76						
Misc Tumors		372		88	321		81	335		88			
			260	83		194	85		194	83			
Total Pt. Studies		18402		84	14264		88	9994		87	5062		30
Total Lesions			9571	86		6879	90		4094	89			



CARDIAC APPLICATIONS

Myocardial Viability



Disease Background. A key issue for imaging is to determine whether a given portion of the heart is viable. This means looking at areas of the heart that are not functioning properly and determining whether tissue is still alive and can recover if the blood supply is restored by revascularization. This is a biochemical question. Biochemists and biologists have shown that glucose is a protective substrate to the heart when blood flow is limited. FDG PET helps to determine viability, because those areas of the myocardium that are viable will have glucose metabolism. On the other hand if the myocardial muscle is dead, it will not have any glucose metabolism. The patient whose myocardial muscle demonstrates no glucose metabolism will not benefit from having blood supply re-established to the muscle. Such a patient would need medical therapy or a heart transplant. About 35% of coronary artery disease patients who receive bypass surgery or angioplasty to revascularize the heart do not show improvement in cardiac function because the affected tissue is not reversible (i.e., is dead).

Case Example. A 57-y-old patient with a previous heart attack was evaluated by echocardiography (echo), which showed that the patient's left ventricular ejection fraction (percentage of blood ejected from the heart during cardiac cycle) was compromised at 35% (normal >55%) and that wall motion was abnormal. An FDG PET cardiac study was requested to evaluate for viable, reversible myocardium. The PET image on the left in Figure 20 was obtained by using ^{13}N ammonia in a study of blood perfusion to the heart. ^{13}N ammonia has been approved by the U.S. Food and Drug Administration for imaging blood flow in the heart. The donut-like structure is the heart muscle, and the chamber it encloses is the left ventricle. The defect (arrow) seen toward the right side of the donut is an area of compromised blood flow. The image on the right in Figure 20 is the FDG PET glucose metabolism study, and it clearly shows FDG metabolism in the same area that is compromised with regard to blood flow. This patient, therefore, would be likely to benefit from revascularization (bypass surgery to restore blood to a portion of the heart). This is referred to as a mismatch pattern (i.e., low blood flow with high glucose metabolism).

Why Did FDG PET Help? FDG PET showed that the patient had viable myocardial tissue, which, if blood flow could be restored, could return the function of the heart closer to normal. The patient, therefore, could avoid a heart transplant by undergoing bypass surgery instead. This patient underwent bypass surgery. The ejection fraction returned to 50% and the wall motion to normal levels.

Key Management Issues.

- Determine whether patients with ischemic heart disease and symptoms of congestive heart failure are best treated with coronary artery bypass surgery, cardiac transplantation, or conservative medical therapy

Summary of Evidence for FDG PET in Myocardial Viability Assessment. Myocardial viability studies with FDG PET should be performed in patients with ischemic heart disease and impaired left ventricular function who are potential candidates for coronary revascularization (Table 21).

Presence of myocardial viability as determined by FDG PET predicts functional improvement, improved daily life activity levels, and improved survival after revascularization.

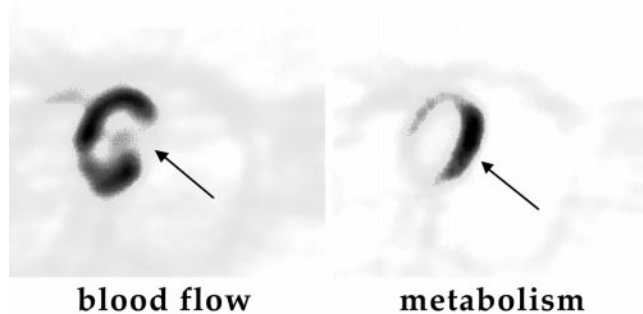
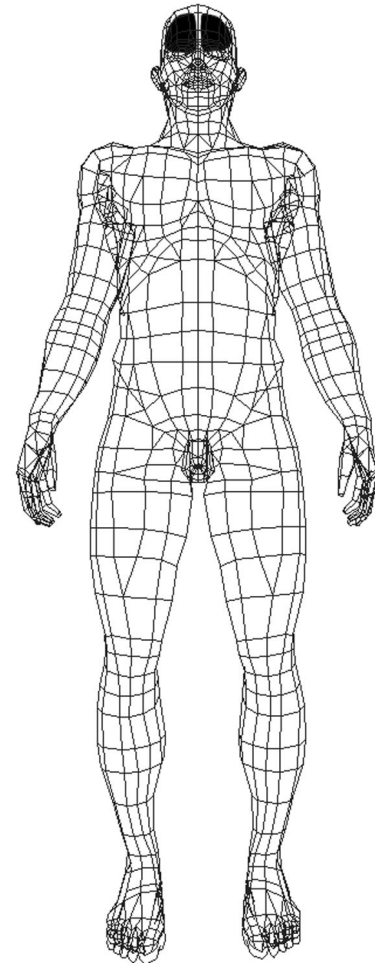


FIGURE 20. Case example, myocardial viability.

TABLE 21
FDG PET in Myocardial Viability: Results of Literature Search

MYOCARDIAL VIABILITY	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pl. Studies	SENS (%)		SPEC (%)		PPV (%)		NPV (%)		ACC (%)		GOLD STD	MGMT(%) EFFECT
					PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)	PET (%)	CT (%)		
Viability																
Inubushi, 2000	A	assess viable myocardium	6	6					77		85				functional improvement	
Schöder, 1999	RA	assess viable myocardium	40	40	93		82								functional improvement	
Maes, 1997	RA	assess viable myocardium	30	30	83		91		91		83				functional improvement	
Baer, 1996	RA	assess viable myocardium	121	42	96		69		83		92				functional improvement	
Gerber, 1996	RA	assess viable myocardium	39	39	75		67		78		63				functional improvement	
Tamaki, 1995	RA	assess viable myocardium	61	43	88		82		76		92				functional improvement	
Knuuti, 1994	RA	assess viable myocardium	48	48	100		63		54		100				functional improvement	
Gropler, 1993	RA	assess viable myocardium	34	34	83		50		52		81				functional improvement	
Lucignani, 1992	RA	assess viable myocardium	14	14	93		80		95		80				functional improvement	
Tamaki, 1989	RA	assess viable myocardium	22	22	78		78		78		78				functional improvement	
Tillisch, 1986	RA	assess viable myocardium	17	17	95		80		85		92				functional improvement	
Summary		by patients	432	335	89		73		74		86					



Disease Background. Dementia is defined as loss of memory and at least one other area of complex behavior sufficient to interfere with day-to-day function. The magnitude of the problem is increasing, and it is estimated that 5% of the population older than 65 y and up to 25% of the population older than 80 y has some form of dementia. Causes of dementia include degenerative changes (e.g., Alzheimer's disease, Pick's disease, Parkinson's disease, Huntington's disease), vascular insufficiency, trauma, endocrine changes, and other causes. Metabolic changes in the brain have been shown to precede structural changes by at least 5 y. Treatment for the degenerative forms of dementia, such as Alzheimer's, is improving with the use of cholinesterase inhibitors and treatment options continue to grow. The diagnosis of early Alzheimer's disease and its differential diagnosis from other organic dementias or the benign effects of aging remain clinically difficult today. PET with FDG has been shown to provide an accurate and positive differential diagnosis of Alzheimer's and of other forms of organic dementias. In some ways, the diagnosis of dementia

is similar to that for cancer in the separation of benign from malignant disease. In the case of dementia, it is the separation of benign from organic degenerative disease.

Case Example. A 67-y-old man presented with a 3-y history of progressive loss of memory and day-to-day function and a clinical diagnosis of possible Alzheimer's. A brain MR image showed no anatomic indications of disease. An FDG PET scan was ordered to evaluate for possible Alzheimer's disease. Shown in the left column in Figure 21 is a normal FDG PET scan from a 64-y-old man. In the right column is the FDG PET scan from the patient in this case. Two representative slices are shown from each individual. The right column clearly shows low FDG metabolism in the back portion of the brain (arrows) in the parietal and temporal regions. This hypometabolism pattern is consistent with Alzheimer's disease.

Why Did FDG PET Help? FDG PET established with a high degree of accuracy that the patient's symptoms were

the result of Alzheimer's disease and not other causes of dementia. The diagnosis of Alzheimer's disease was confirmed 6 y later at autopsy.

Key Management Issues.

- Early diagnosis of dementia versus benign memory loss
- Differential diagnosis of dementia from frontotemporal disease, diffuse Lewy bodies, or cerebrovascular diseases
- Differentiation from pseudodementia/depression (This is a dementia-like state that is caused by depression and not Alzheimer's disease.)

Summary of Evidence for FDG PET in Dementia Work-Up.

Primary neurodegeneration is the most common process underlying dementia, and Alzheimer's disease alone accounts for approximately two-thirds of cases. Regional cerebral metabolic patterns reflect pathophysiologic changes in brain that will lead to Alzheimer's disease, even before they give rise to symptoms. In addition to the diagnostic value FDG PET may have in evaluation of dementia, it can also serve as a prognostic tool to determine the likelihood of deterioration of mental status during the years after scanning, thereby facilitating planning by the patient and family members. Although results have varied, depending in part on the severity and diagnostic mix of patients, nearly all studies designed to assess the accuracy of FDG PET in the diagnosis of dementia have found sensitivity for Alzheimer's disease to be >90%, with specificity typically approximating 75% (range, 67%–97%). Meeting the challenge of accurately identifying minimally affected patients to allow them to reap the greatest potential therapeutic benefits requires making the diagnosis with a high degree of sensitivity and overall accuracy at the earliest possible stage of disease. The consistently high sensitivity of FDG PET in patients with even mild impairment makes it well suited for assisting with that task (Table 22).

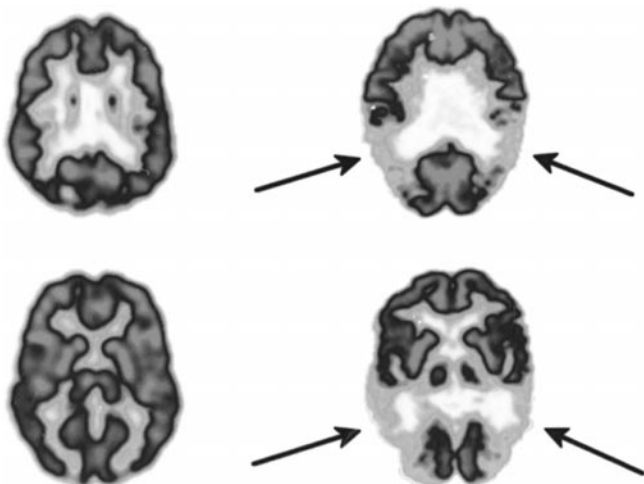


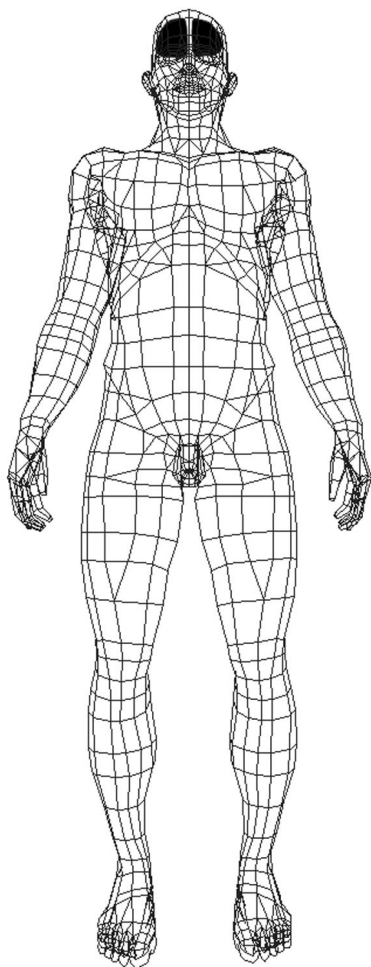
FIGURE 21. Case example, dementia.



TABLE 22
FDG PET in Dementia Work-Up: Results of Literature Search

DEMENTIA WORKUP	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	SENS		SPEC		PPV		NPV		ACC		GOLD STD	MGMT/EFFECT
					SENS (%)	SENS CI (%)	SPEC (%)	SPEC CI (%)	PPV (%)	PPV CI (%)	NPV (%)	NPV CI (%)	ACC (%)	ACC CI (%)		
Silverman, 2000 ¹	A	eval of neurodegen dem dis/PND eval of alzheimer's	70	70	93	92									autopsy	
Silverman, 2000 ²	A	eval of ACT effect/PET in dem pts	128	128	90	76			82	86					autopsy	
Hoffman, 2000 ³	RA	dx for possible & probable AD	22	22	93	75			87	86					follow-up pathology	
Silverman, 1999	A	cognit or behav change	88	88	93	74			80	91					follow-up 3 y	
Mielke, 1998	RA	normal and impaired	45	45											clin eval nr time of scan	
Tedeschi, 1995; Salmon, 1994; Mielke, 1996	RA	cognitively impaired	20	20	92	71									histopathology	
Salmon, 1994 ⁴	RA	cognitively impaired	104	104	93	80									clin eval nr time of scan	
Mielke, 1994	RA	normal/probable AD/vasc dem	45	45	(ROC)	(ROC)									clin eval nr time of scan	
Herholz, 1993	RA	normal and probable AD	71	71	95	97									clin eval nr time of scan	
Szelies, 1992	RA	normal and probable AD	57	57											clin eval nr time of scan	
Summary		By Patients	650	720	93	80			82	88			87			

¹PND- Progressive Neurodegenerative Disease.
²ACT- Anticholinesterase Therapy.
³AD- Alzheimer's Disease.
⁴Note the value of specificity of .90 in the study [Salmon, 1994] is based on all patients with clinical evaluation as gold standard excepting those with Parkinson's disease dementia.



Disease Background. Epilepsy is a common condition, with a prevalence in the population of about 1 in 200 people. Several abnormalities within the brain can lead to abnormal “synchronous firing” of neurons, causing a seizure. Depending on which part of the brain is epileptogenic, seizures will have different outward appearances. In a grand mal seizure, all extremities move as a result of abnormal neuronal firing, which spreads within the brain to cause a diffuse motor seizure. Imaging of all types helps to locate abnormalities within the brain, and, when coupled with electroencephalography (EEG, scalp electrical signal monitoring), can help to manage epilepsy patients. Many patients can be controlled well on medications. Patients who have seizures despite having tried several medications are referred to as patients with intractable seizures. In these patients, identifying the source of the seizure within the brain often can lead to surgery that can stop or reduce the seizures. Imaging, including FDG PET, can play an important role in determining whether a patient is a candidate to be operated on for seizure control. The alternative (invasive electronic moni-

toring) requires putting electrodes into the brain parenchyma or meninges, with attending morbidity and mortality.

Case Example. An 11-y-old boy, diagnosed with epilepsy at age 7, had been treated with medications for 4 y. During the last year, he had continued to have seizures, even with a change in antiseizure medications. An FDG PET scan was ordered to evaluate for the possible source of the seizure. MRI showed no structural abnormality. The FDG PET scan (Fig. 22) showed moderate-to-severe hypometabolism (lower than normal glucose utilization) in the right parietal, posterior, frontal, occipital, and temporal lobes (arrows) in the interictal period (i.e., between seizures).

Why Did FDG PET Help? PET showed epileptogenic tissue in the localized brain. Surgery was performed to resect the dysfunctional tissue. The child, after surgery, was seizure free.

Key Management Issues.

- Diagnosis of partial epilepsy (MRI negative)
- Localization of seizure focus
- Prediction of surgical outcome (prognosis)

Summary of Evidence for FDG PET in Seizure Work-Up. In patients who have medically intractable epilepsy, neurosurgery to resect epileptogenic foci can decrease or eliminate seizure episodes and reduce neurologic impairment resulting from recurrent seizures and/or high doses of anti-convulsants. Patients with complex partial seizures, particularly those who have EEG evidence of a temporal lobe focus but inconclusive findings on MRI, often are referred for functional brain imaging to assess interictal metabolism. PET with FDG can identify epileptogenic zones through localization of hypometabolic brain tissue interictally. Interictal FDG PET has been demonstrated to be as useful for presurgical planning in most patients with temporal lobe epilepsy as the more logistically cumbersome ictal SPECT or more invasive EEG monitoring with depth electrodes. Patients with unilateral foci of hypometabolism identified by PET have been found in numerous studies to have a high likelihood of benefiting from neurosurgery, regardless of whether invasive electrode monitoring is also undertaken. Patients can thus be saved risks and costs otherwise incurred with invasive monitoring. Further study is needed to define more specifically the role of depth electrodes and surgical therapy in patients with findings of bilateral hypometabolism (Table 23).

SUMMARY

For a summary of all FDG PET literature searched, see Table 24.

APPENDIX A. LITERATURE SEARCH CRITERIA AND DATA ANALYSIS METHODS

Literature Search Criteria

The literature search was performed using the databases Medline/Healthstar 1993–2000 and Biosis Previews 1993–

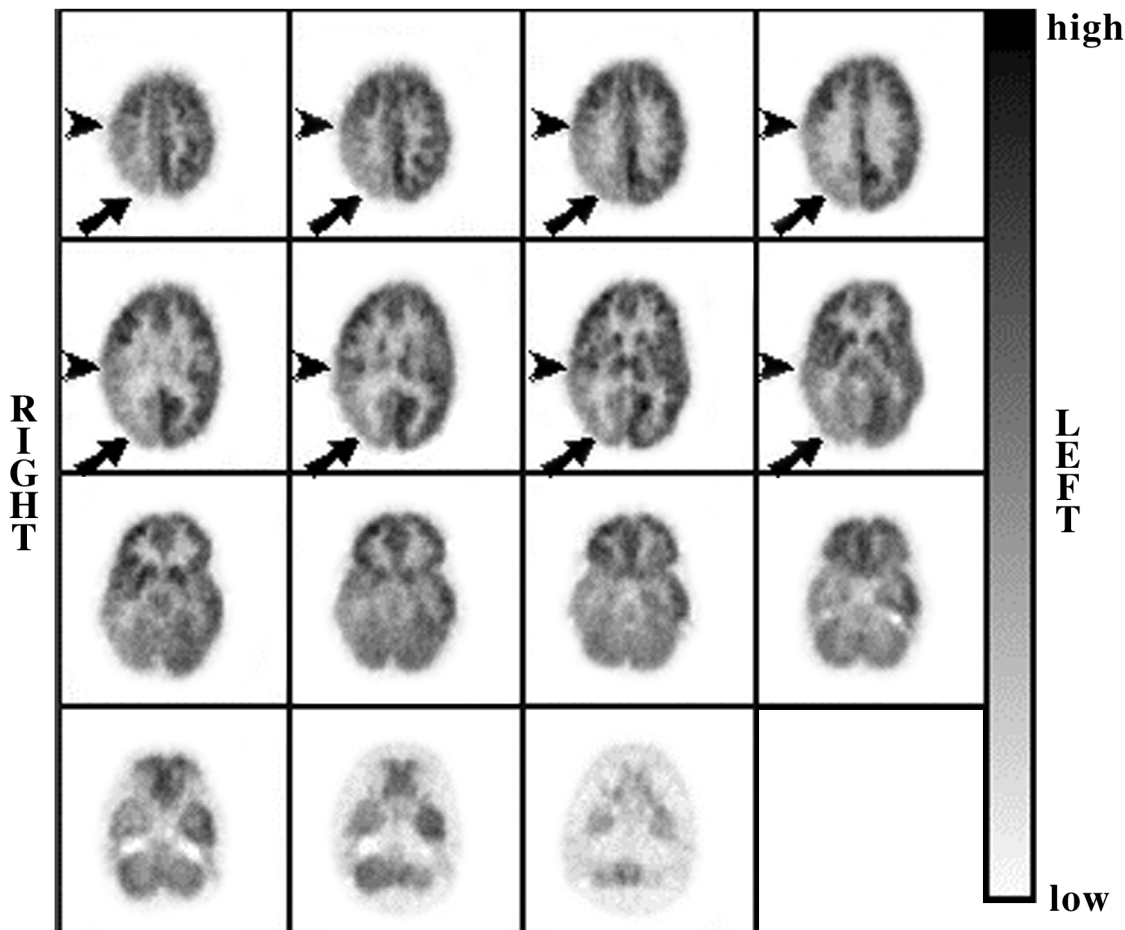


FIGURE 22. Case example, seizure.

2000 for articles and abstracts published from January 1993–June 2000. All key word combinations, including FDG PET, PET, and specific oncologic, neurologic, and cardiac applications were searched. Printed copies of *The Journal of Clinical Positron Imaging* (1998–2000) and *The Journal of Nuclear Medicine* abstracts (1996–2000) also were used. Only articles/abstracts in English were used, with the exception of a few English abstracts of non-English-language articles that provided complete information. Both dedicated PET and newer low-cost PET technology (for example, coincidence imaging) studies were included.

The only exceptions to our search time period occurred in the neurological and cardiac application categories. Specifically for myocardial applications, the Medline search extended back to 1986, with a focus on literature assessing viable myocardium. For dementia and seizure workup, the Medline search extended back to 1980, with respective foci on literature assessing accuracy in diagnosing individual patients with dementia and on literature assessing PET performance with respect to evaluating potential candidates for neurosurgery.

All literature that was not clear with respect to methods and/or reporting was excluded. Furthermore, any article/abstract that reported on a study with five or fewer individ-

uals also was excluded. A total of 775 articles/abstracts were retrieved from the literature for our review. Approximately 8 articles could not be obtained from interlibrary requests to outside libraries. The data analysis used 473 unique articles/abstracts (specifically 151 abstracts and 322 articles), and 302 were excluded as per the inclusion/exclusion criteria. The spreadsheets listed a total of 561 article/abstract entries, of which 17 were repeated across several spreadsheets to which they were applicable and 71 were repeated within spreadsheets in multiple applications.

Inclusion Criteria. (1) Abstracts and articles reporting data within which sensitivity (sens), specificity (spec), positive predictive value (ppv), negative predictive value (npv), accuracy (acc), and management change (mgmt) values were either partially or fully listed or could be partially or fully derived for FDG PET imaging in the 22 different oncologic areas, cardiac viability area, and dementia and seizure work-up areas. In addition, some studies (e.g., seizure) were listed with FDG PET contributions to clinical issues without accompanying accuracy data. Only data with stated or derived total patient studies or total lesions were incorporated into the weighted averages. In those instances in which CT data were found in the PET literature satisfying the inclusion criteria, these were also listed.

TABLE 23
FDG PET in Seizure Work-Up: Results of Literature Search

SEIZURE WORKUP	ARTICLE TYPE	PURPOSE	Total No. Patients	Total Pt. Studies	Comments	PET HELPS
Salanova, 1999	RA	presurg eval/ outcome/temp lobe	22	14	PET suggested that focal functional deficits appear early in pts w medically refractory TLE which may help in early ident.	yes
Akimura, 1999	RA	clarify improvment from epil surg	9	9	4/9 pts showed improved glucose metabolism in formerly hypometabolic zones.	yes
Barrington, 1998	RA	determine if routine EEG justified	236	6	PET scan interpretation w single seizure occurrence not influenced by CSEEG recordings. The value of routine CSEEG in oulpts treated w medication should be reappraised w pot cost savings.	yes
Kraemer, 1998	RA	eval lesionectomy outcome	15	15	PET can be part of careful pt selection where lesionectomy is procedure of choice for occult vascular malformations.	yes
Blum, 1998	RA	predict good surg outcome/ident UTH	10% Instit PET sens	10% Instit PET sens	PET has proven useful in epilepsy surg to ID unilateral temporal hypometabolism (UTH). Pts w BTH have a worse prognosis for seizure remission after surg.	yes
Mukahira, 1998	RA	surg/intractible childhood epilepsy	14	14	PET revealed abnormalities providing crucial information regarding the epileptic focus.	yes
Salanova, 1998	RA	predict outcome/TLE surg	38	38	PET scans found complementary to head MRIs. Concordance between PET temporal hypometabolism and MRI hippocampal sclerosis correlated w better outcome.	yes
Achtern, 1998	RA	lateralization/refrac TLE	29	29	Proton MR spectroscopy more sensitive in depicting metabolic abnormalities than PET for TLE. Pts w neg PET will benefit from MR spect for lateralization.	no
Da Silva, 1997	RA	eval Landau-Kleffner syndrome	17	17	PET enabled study of pathophysiology of syndrome. Suggested importance of temporal lobe structures, and revealed cortical abnormalities indicating common extensive brain functional disturbance.	yes
Markand, 1997	RA	localize epilept focus/MI-CPS	67	36	Interictal PET equally sensitive to ictal SPECT. Both play critical role providing localization in MRI neg pts allowing surg wout addit invas electrographic monitoring.	yes
Duncan, 1997	RA	presurg PET mapping/loq brain	15	15	Suggested noninvasive presurgical PET brain mapping has potential to reduce risk and improve neurologic outcome.	yes
Holopainen, 1997	RA	verify ext hypometabolic area	5	5	PET can be used in conjunction w proton magnetic resonance spectroscopy for clinical assessment of children w intractable TLE.	yes
Theodore, 1997	RA	seizure focus detection	46	46	PET provides valuable data in pts w unlocalizd surface ictal EEG and can reduce the number of pts who require IEEG studies.	yes
Tanaka, 1997	RA	presurg ident of epileptic foci	10	10	BP images delineated the epileptic foci more precisely than either PET or ictal perfusion SPECT.	no
Rintahaka, 1997	RA	tuberous sclerosis complex	23	23	PET provides addit localizing info to CT and MRI in pts w tuberous sclerosis complex. If EEG, CT, and MRI are unifocal or unilateral, and surg is considered, more detailed eval w PET may help to determine if contralateral tubers present and eval the functional integrity of brain as a whole.	yes
Lucignani, 1996	RA	SEEG&PET/severe part epilepsy	16	16	PET consistently allowed localization of temporal hypometabolism, but is not specifically related to SEEG patterns.	no
Helveston, 1996	RA	comparison w MRI/intract TLE	16	16	Ea imag technique yields useful info for sz lateraliz in TLE w hippocamp formation volumetric assessmt (HV MRI) yielding more than QPET or OMR.	yes
Ferrie, 1996	RA	epileptic encephalopathies	32	32	PET suggests that some children w epil encephalopathies previously thought to have prim generalized seizures due to multifocal pathology may have unifocal cortical origin which may be amen to surg.	yes
Delbeke, 1996	RA	postsurg outcome/uncontrol part sz	38	38	A focus of interictal temporal hypometabolism on PET is associated w marked improvement of seizure control after surg in 94% of pts.	yes
Snead, 1996	RA	pediatric epil surg	100	56	Found insuffic corrl of interict hypometab area on PET and epilept zone in terms of anat locat & size to justify foregoing chronic invasive intracran monitoring in children w intract part sz in eval for surg unless absolute concord of all neuroimaging,clin.&video-electroenceph data.	no
Fois, 1995	RA	intract childhood epil/surg corral	30	30	Functional imaging w SEEGs appears superior to only CT and MRI for selecting children w epil for surg espec w CPS resistant to therapy.	yes
De Reuck, 1995	RA	late onset/cryptogenic symp sz	10	10	Suggested that late-onset seizures could be premonitory signs of progressive encephalopathy of unknown origin.	yes
Heinz, 1994	RA	compare PET and MRI w histol	27	27	PET sens for mesial temporal sclerosis=12/15(80). MRI sens=13/15(87).	yes
Shih, 1994	RA	compare PET/MRI w outcome	27	27	PET sens for good outcome=17/24=71, incl 3/4 pts missed by MRI.	yes
Savic, 1993	RA	PET vs SPECT/MRI vs CT/cw ECOG&outcome	30	30	FDG-PET more accurate than SPECT. MRI more sens than CT.	yes
Savic, 1993	RA	compare PET w intra- & extra-cranial EEG	8	8	FDG laterality agreed w EEG in 5/8. [C-11]flumazenil-PET laterality agreed w EEG in 8/8.	yes
Leiderman, 1992	RA	compare FDG-PET w [O-15]water-PET	28	28	FDG-PET more specific for localization of epileptic zones.	yes
Engel, 1990	RA	compare depth-SEEG w PET+ictal surf EEG	153	153	Concordant PET and ictal surface EEG studies generally obviate the need for SEEG.	yes
Pawlik, 1990	RA	retrosop rev of exper w 213 PET studies	124	124	PET had sens of >.90, and was superior to other diagnostic methods in typical TLE.	yes
Swartz, 1989	RA	compare CT/MRI/PET in frontal lobe epil	22	22	Sens of CT=.32;MRI=.45;PET=.64. Frontal hypometab signif correlated w ictal semiology.	yes
Chuganji, 1988	RA	compare PET to outcome & neuropathol	8	8	In the 4 pts with outcome data, all were correctly classified; PET more sens than CT and MRI.	yes
Stefan, 1987	RA	compare PET/SPECT/MRI/CT w EEG	10	10	PET was the best imaging modality for definition of epileptic focus.	yes
Theodore, 1986	RA	compare PET/CT/MRI w EEG	36	36	PET more sensitive than CT or MRI.	yes
Summary			1261	948		

TABLE 24
Summary of Results of FDG PET Literature Search

CANCER	PURPOSE	Total Pt.	Total	SENS	Total Pt.	Total	SPEC	Total Pt.	Total	ACC	Total Pt.	Total	MGMT
		Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	EFFECT (%)
Bladder	Staging	136		76	98		87	98		83	12		17
	Dx/Staging	52		93	26		86	26		88			
	Recurrence	12		60							12		17
Brain	Dx	36		91									
	Staging	31		86									
	Recurrence	258		79	213		77	161		76	89		31
	Mon Response	17		82	17		83						
Breast	Other	34		93	19		67	19		84			
	Dx	202		91	97		93	105		95	6		100
			140	90		140	92		105	88			
	Staging	1407		91	1373		88	1328		90	111		24
			242	95		53	88		33	88			
	Dx/Staging	65		75	9		83	52		83			
	Recurrence	414		80	414		85	268		82	23		40
Colorectal	Mon Response	206		81	174		96	84		92			
			31	90		31	74						
	Staging										236		36
			24	96									
	Dx/Staging	101		85	87		71	87		94			
	Recurrence	1426		94	1166		87	418		94	915		32
			981	93		912	96		331	87			
Gastro-Esoph	Mon Response		34	100		23	90		11	100			
	Dx	120		96				48		98	99		14
			276	80		276	95		276	86			
	Staging	545		73	302		90	245		83	229		20
			15	93									
	Dx/Staging	109		80	109		95	109		86	109		14
	Recurrence	41		100	41		43	41		73			
Head&Neck	Mon Response	13		100				13		46			
	Dx	129		93	36		70	61		87			
			311	84		267	83		267	85			
	Staging	363		87	279		89	301		88			
			2020	84		1999	95		596	94			
	Dx/Staging	296		88	249		83	184		88	15		33
			179	83		151	94		158	89			
Hepatocellular	Recurrence	342		93	271		83	283		87	15		33
			278	84		241	92		241	90			
	Mon Response	128		84	122		95	81		96			
			16	44									
	Staging	292		77	249		97	249		93	20		60
	Dx/Staging	22		64									
	Recurrence		9	88									
Lung	Dx	919		96	797		73	719		90			
			278	91		259	68		101	82			
	Staging	1867		83	1495		91	1272		82	1565		37
			1721	83		1553	92		1478	90			
	Recurrence	209		98	193		92	180		96			
			39	100		39	62		39	87			
	Mon Response	161		94	161		90	126		96			
Lymphoma	Other	101		83									
	Dx	11		100									
	Staging	1179		90	826		93	158		88	407		21
			1156	91		58	100		32	95			
	Dx/Staging	254		92	177		93			62			5
	Recurrence	557		87	453		93	155		88	158		10
			114	100									
Melanoma	Mon Response	257		90	279		93	13		69			
									32	95			
	Staging	888		83	863		91	125		91	283		26
		899	87		461	68		83	84				

TABLE 24 (Continued)

CANCER	PURPOSE	Total Pt.	Total	SENS	Total Pt.	Total	SPEC	Total Pt.	Total	ACC	Total Pt.	Total	MGMT
		Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	PET (%)	Studies	Lesions	EFFECT (%)
(cont.)				(%)			(%)			(%)			(%)
Ovar/Uter/Cerv	Dx	274		66	238		77	153		77			
	Staging	331		54	119		96						
	Dx/Staging	112		86	58		82	73		87			
	Recurrence	359		88	327		90	220		87	30		17
	Mon Response	11		100				11		100			
Pancreas	Dx	293		94	281		90	259		92	26		50
			51	90		51	91		51	90			
	Dx/Staging	404		83	360		82	368		81	65		43
			17	76									
	Staging	182		70	182		93	182		91	33		36
			66	67									
	Recurrence										19		53
	Mon Response	45		92	14		100				19		16
Prostate	Staging	196		57	49		100						
			202	65									
	Recurrence	100		26									
	Mon Response	43		86									
Renal	Dx	11		89									
	Staging	97		76	22		100	4		100			
Testicular	Staging	129		82	129		94	37		92	27		22
	Recurrence										53		51
	Other	133		80	133		92	133		87			
Thyroid	Staging	430		69	268		89	249		84	60		22
	Dx/Staging	125		68	9		67	19		100	58		9
			41	49									
	Recurrence	474		77	324		91	324		86	21		53
			66	73		66	86		66	80			
Unknown Prim	Staging	235		82	114		71	114		77	285		29
			64	91		64	76						
Misc Tumors		372		88	321		81	335		88			
			260	83		194	85		194	83			
Total Pt. Studies		18402		84	14284		88	9994		87	5062		30
Total Lesions			9571	86		6879	90		4094	89			
CARDIAC	Viability	329		89	329		73	289		79			
DEMENTIA	Workup	573		93	573		80	720		87			
SEIZURE	Workup	948											

(2) Oncologic studies drawn from the period January 1993–June 2000; dementia and seizure studies from January 1980–June 2000; and cardiac studies from January 1986–June 2000.

(3) Response-to-treatment articles were included in the spreadsheets where a 2 × 2 table could be created from the reported data for: responders/nonresponders versus increased FDG/decreased FDG. In those instances in which a 2 × 2 table could not be formulated, the article was excluded.

Note that three articles were also included that provided no numerical information about FDG PET accuracy but had some useful features, which are described in the comments field. These articles, therefore, have no

bearing on the weighted averages summarizing all the literature data. These studies by Bischoff et al. (46), Holthoff et al. (197), and Rozental et al. (354) were all part of the monitoring response application.

Exclusion Criteria. (1) Case reports, review/tutorial articles, and studies with 5 or fewer patients.

(2) Articles not in English. However, abstracts in English of articles not in English but with relevant information were included.

Data Analysis

Data analysis was performed using simple weighted averages. Therefore, studies with more patients were weighted more than studies with fewer patients to arrive at estimates

of the sensitivity, specificity, and, when possible, management changes. Weighting is the easiest method to use on such a large number of studies, each of which may or may not present a full 2×2 table of outcomes. No attempt was made to perform a formal meta-analysis.

In instances in which articles and abstracts included data for multiple categories (e.g., diagnosis/staging/recurrence), the entire article entry was listed in each of the three individual categories (diagnosis, staging, and recurrence) to preserve the entirety of a study's reporting and to represent that study's contribution to data for that category for both this report and possible future analyses that might be looking for all references including data for a given category (e.g., specifically for recurrence.) Only data relevant to a specific category was used in the weighted average for that category (e.g., in calculating the weighted average in the recurrence category, only the recurrence portion of the article's data was used, even though data for diagnosis also may have been listed).

The number in the total patient studies column sometimes exceeded that in the total number of patients column for a given entry line (e.g., in instances in which patients may have had multiple FDG PET scans). For each line entry of data, the total patient studies or total lesions were listed upon which the 2×2 table was based for calculating a given line of data (e.g., if 58 patients had 62 scans from which the true positive (TP), true negative (TN), false positive (FP), and false negative (FN) values were counted, 62 was listed for total patient studies).

In those instances in which articles/abstracts had data broken down for various reported subgroups (e.g., mediastinal and hilar lymph nodes or lymph nodes <1 cm), total patient studies for each subgroup would be listed (as explained above), but often these subgroups would have overlapping patients. In terms of the data analysis, when a given study provided overall values in addition to listing various subgroup values, the overall value was used in the weighted average. When an overall value was not listed, the subgroup data was weighted in by the total patient studies value from which it was generated (or by total lesions, if listed by lesions). The only exceptions occurred in the lung cancer spreadsheet/staging section in the four articles by Baum et al. (36), Tatsumi et al. (424), Ryu et al. (356), and Marom et al. (290). When these studies reported subgroup values for the full patient study count multiple times, the subgroup

values were averaged and weighted into the weighted average formula by the total patient studies for one group only.

APPENDIX B. DATA ANALYSIS SUMMARY

Because the default analysis (analysis 1) of our 22 spreadsheets listed all the literature values used that included multiple listings of abstracts/articles both within spreadsheets (e.g., both in the diagnosis/staging and staging applications of a particular cancer) and across spreadsheets, some overlapping of patient studies occurred. To analyze the effects of listing the broad view of the literature values used we performed 5 additional data analyses to study the effects on PET sens/spec/acc by selecting out certain studies according to the following criteria:

(1) Analysis 1 (default analysis): Included all literature used that had some overlap of total patient studies within and across spreadsheets.

(2) Analysis 2: Used data from only those studies that reported jointly the sensitivity and specificity values for PET and PET accuracy values that included one other PET statistical ratio column (e.g., reported PET accuracy and also PET sensitivity).

(3) Analysis 3: Repeated the default analysis, including data from only full research articles. All abstracts were specifically excluded.

(4) Analysis 4: Repeated analysis 2, including data from only full research articles. All abstracts were excluded.

(5) Analysis 5: Pooled sens/spec/acc values for PET across all available studies that provided TP/TN/FP/FN values for each cancer and for all cancers together. This is a formal pooling analysis, using data from each study to construct a large 2×2 table. Note that all four cells (TP/TN/FP/FN) must be available, that is, information for patients both with and without disease is required. Therefore, some studies used in some of the weighted averaging subanalysis formulations (e.g., reporting sens only) could not be included in the pooling subanalysis, and thus slight discrepancies exist between the article subsets used in the weighted averages and those used in the pooling.

(6) Analysis 6: Repeated the default analysis excluding all data from nondedicated PET machines (e.g., coincidence imaging).

Results are provided in Table 25.

TABLE 25
Data Analysis Summary

	Total Patient Studies	Total Lesions	Sens (%) PET	Total Patient Studies	Total Lesions	Spec (%) PET	Total Patient Studies	Total Lesions	Acc (%) PET
Analysis 1* [*Default]	18402		84	14264		88	9994		87
		9571	86		6879	90		4094	89
Analysis 2	14212		86	14212		88	8892		88
		6879	85		6879	90		3934	89
Analysis 3	12004		86	9823		88	7313		89
		7397	86		5385	91		2806	90
Analysis 4	9801		86	9801		88	6850		89
		5385	86		5385	91		2795	90
Analysis 5	14458		85	14458		91	14458		88
		8187	85		8187	93		8187	90
Analysis 6	17783		84	13954		88	9663		88
		9244	87		6607	90		3666	89

APPENDIX C. ABBREVIATIONS LEGEND

Abdom: abdominal
 Abst: abstract
 Acc: accuracy
 Activ: activity
 Addit: additional
 Adenocarc: adenocarcinoma
 Adjuv: adjuvant
 Adrenalect: adrenalectomy
 Adv: advanced
 AFP: serum alpha-fetoprotein level
 AIDS: acquired immunodeficiency syndrome
 ALNDs: axillary lymph node dissections
 Amen: amenable
 Amput: amputations
 Anat: anatomical
 Andro: androgen
 Antibod: antibodies
 Antiestr: antiestrogen
 Art: article
 Aspir: aspirate
 Assessmt: assessment
 Asymp: asymptomatic
 Autop: autopsy
 Av: average
 Avdmaj surg: avoid major surgery
 BCNU: carmustine
 Behav: behavioural
 Biochem: biochemical
 Biop: biopsy
 Bne: bone
 BPH: benign prostatic hyperplasia
 Br: brain
 Brst: breast
 BS: bone scintigraphy
 BTH: bilateral temporal hypometabolism
 B/w: between
 C: calcitonin
 Ca: cancer
 Calcit: calcitonin
 Calcs: calculates

Cam: camera
 Carcin: carcinoma
 Cathet: catheterization
 CDM: conventional diagnostic methods
 CEA: carcino-embryonic antigen
 Cerv: cervical
 Chem: chemistry
 Chemo: chemotherapy
 Chemohormonother: chemohormonotherapy
 Chemoradio: chemoradiotherapy
 Chge: change
 CHOL: 11C choline
 Cholang: cholangio-pancreaticography
 CI: conventional imaging
 Classif: classification
 Clin: clinical
 Clinstge: clinical stage
 Cm: centimeter
 Cnfrm: confirmation
 CNS: central nervous system
 CoDe-PET: coincidence detection PET
 Cognit: cognitive
 Colonosc: colonoscopy
 Compar: comparison
 Concom: concomitant
 Concord: concordance
 Conn: connective
 Cont: continued
 Contrad: contradictory
 Conv: conventional
 Corr: correctly
 Correl: correlation
 CR: complete response
 Craniot: craniotomy
 Crse: course
 CT: computed tomography
 CUP: cancer of unknown primary
 Cur: curative
 CYT-356: capromab pendetide
 Cytol: cytology
 Cytopath: cytopathology



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DAR: differential absorption ratio
 Decis: decision
 Decr: decrease
 DedPET: dedicated PET
 Def: definite
 Degen: degenerative
 Dem: dementia
 Detect: detect
 Diff: different
 Differen: differentiated
 Dimens: dimensions
 Dis: disease
 Discrep: discrepancy
 Dissect: dissection
 Dissem: disseminated
 Dist: distant
 Ds: days
 DUR: dose uptake ratio
 Dwnstge: downstage
 Dx: diagnosis
 Ea: each
 EEG: electroencephalographic
 E.g.: for example
 Elev: elevation
 Endos: endosonography
 Endosc: endoscopic examination
 Entero: enteroclysis
 Epil: epilepsy
 Epilept: epileptogenic
 Equiv: equivocal
 ER+: biopsy-proved estrogen receptor-positive
 Esoph: esophageal
 Eval: evaluate
 Evid: evidence
 Ex: exam
 Excis: excisional
 Exp: experience
 Explor: exploratory
 Ext: extension
 Extrathor: extrathoracic
 Fav: favorable
 Fd: found
 FDG: 2-[F-18]Fluoro-2-Deoxy-D-Glucose
 Fm: from
 FN: false negative
 Fn: function
 FNA: fine-needle aspiration
 FNAB: fine needle aspiration biopsy
 FP: false positive
 Ga: gallium
 Gastro: gastroesophageal
 Gastros: gastroscopy
 GCI: gamma camera coincidence imaging
 Gde: grade
 Gluc: glucose
 Gp: group
 GRD: gross residual disease
 Gynecol: gynecological
 H&N: head and neck
 HCC: hepatocellular carcinoma
 HD: hodgkin's disease
 Hep: hepatic
 Hepatocell: hepatocellular
 Hi: high
 Hippocamp: hippocampal
 Hist: history
 Histol: histology
 Histopath: histopathology
 Horm: hormone
 Hypertherm: hyperthermic
 Hypometab: hypometabolic
 Hypopharyng: hypopharyngeal
 Ident: identify
 Imag: imaging
 IMLN: internal mammary lymph node
 Immed: immediate
 Imprvemt: improvement
 IMT: 123I-Iodo-alpha-methyltyrosine
 Inconclus: inconclusive
 Incorr: incorrectly
 Incr: increased
 Indeterm: indeterminate
 Indic: indicative
 Individ: individual
 Info: information
 Init: initiate; initial
 Insuffic: insufficient
 Interict: interictal
 Intracran: intracranial
 Intract: intractable
 Involv: involvement
 IORT: intraoperative radiation therapy
 Irrad: irradiated
 IS: immunoscintigraphy
 Isol: isolated
 Kn: known
 Lapar: laporatory
 Laparat: laparotomy
 Laryng: laryngeal
 Lateraliz: lateralization
 Les: lesion
 Lft: left
 LN: lymph node
 Lo: low
 Loc: local
 Locat: location
 Locoreg: locoregional
 Lowabd: lower abdomen
 LT: long term
 Lumpect: lumpectomy
 Lymphad: lymphadenectomy
 Majdwn: major downstaging



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Majmgmt: major management	Otolaryn: otolaryngologic
Majupstg: major upstaging	Ovar: ovarian
Malig: malignancy	Overstgd: overstaged
MALT: mucosa-associated lymphoid tissue	P53: p53 protein expression of HCC
Mar: marrow	Pancr: pancreatic
Mastect: mastectomy	Papill: papillary
Meas: measurable	Paramalig: paramalignant
Mediast: mediastinal	Part: partial
Mediastinos: mediastinoscopy	Pathol: pathology
Melan: melanoma	Pblms: problems
MET: 11-Cmethionine	Pca: prostate cancer
Met: metastatic	PCS: positron coincidence scintigraphy
Metab: metabolism	PD: progressed disease
Mets: metastases	Pelv: pelvic
Mgmt: management	Perfsurg: perform surgery
MH: M. Hodgkin	Perfus: perfusion
MIBG: metaiodo-benzylguanidine	Persist: persistent
MIBI: 99mTc-Methoxyisobutylisonitrile	PET: positron emission tomography
MI-CPS: medically intractable complex partial seizures	Phys: physical
Mindwn: minor downstaging	Polychemo: polychemotherapy
Minmgmt: minor management	Poor: poorly
Minupstg: minor upstaging	Potent: potentially
Misc: miscellaneous	PPV: positive predictive value
MM: mammography	PR: partial response
Mo: month	Preop: preoperative
Mod: moderately	Prim: primary
Modals: modalities	Prob: probably
Mon: monitor	Proced: procedure
Morphol: morphologic	Progress: progressing metastases; progression
MR: magnetic resonance	Prolif: proliferative
MRD: minimal residual disease	Prost: prostate
MRI: magnetic resonance imaging	PSA: prostate specific antigen
MTC: medullary thyroid cancer	Pt: patient
N: total number of patient studies or lesions	Pt gp: patient group
N staging: nodal staging	Pts: patients
NC: no change	Pulm: pulmonary
ND: new disease	RA: research article
Necros: necrosis	Rad: radiation
NED: no evidence of disease	Rad lymphad: radical lymphadenectomy
Neg: negative	Radiog: radiography; radiographic
Neurodeg: neurodegenerative	Radiol: radiological
NHL: non-Hodgkin's lymphoma	Recur: recurring
Nk: neck	Recurr: recurrence
No: number	Ref: reference
Nochge: no change	Refrac: refractory
Noneupstg: none upstaged	Relap: relapsed
NPV: negative predictive value	Resect: resectable
Nr: near	Resid: residual
Nsclc: non-small cell lung cancer	Resis: resistant
Nt: not	Restage: restaging
Observ: observation	Retroper: retroperitoneal
Occ: occupying	Retrosp: retrospective
Oper: operable	Rev: review
OPET: other PET	RIT: radioimmunotherapy
Ophthal: ophthalmologic	RNB: radionuclide bone scintigraphy
Osteo: osteomyelitis	Rpts: reports



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Rspnse: response
Scint: scintigraphy
Sclc: small cell lung cancer
Scn: scan
SEEG: stereo-EEG
Sens: sensitivity
Ser: serial
Signif: significantly
Simult: simultaneously
Sites: lesion sites
Sl: slight
Smplg: sampling
SNB: sentinel node biopsy
Sonog: sonography
Sp: space
Spdsht: spreadsheet
Spec: specificity
SPECT: single-photon emission CT
SPET: single photon emission tomography
SPN: solitary pulmonary nodule
S.t.: short-term
Std: standard
Stereo biop: stereotactic biopsy
Stg: stage
Strat: strategy
STS: soft tissue sarcomas
Subclin: subclinical
Subgps: subgroups
SUR: standardized uptake ratio
Surf: surface
Surg: surgery
Susp: suspected
Suspici: suspicious
SUV: standardized uptake values
Symp: symptom
Sys: system
Sz: seizure
TCB: trucut biopsy
Temp: temporal
Tg: thyroglobulin
Theor: theoretical
Ther: therapy
Therap: therapeutic
Thor: thoracic
Thorac: thoracotomy
Thyrd: thyroid
Thyroglob: thyroglobulin
Tiss: tissue
T: L ratio:tumor-to-normal liver ratio
TLE: temporal lobe epilepsy
TN: true negative
Tot: total
TP: true positive
Trtmt: treatment
TTNA: transthoracic needle aspiration
Tum: tumor

Ultim: ultimately
Understgd: understaged
Unil: unilateral
Unk Prim: unknown primary
Unnecess: unnecessary
Upstge: upstage
Uptke: uptake
US: ultrasonography
Uter: uterine
UTH: unilateral temporal hypometabolism
Util: utilization
Vasc: vascular
Vert: vertebral
Vs: versus
W: with
WB: whole body
WBS: whole-body scintigraphy
Wd: would
With: withdrawal
Wks: weeks
Wt: weight
WW: watchful waiting
Y: year
131-I: 131-Iodine
2x2: two by two table of true/false positive values and true/false negative values
18F-FDG: 18F-fluorodeoxyglucose
201TI SPET: 201TI single photon emission tomography
201TI SPECT: 201 TI chloride single-photon emission CT
#: number
+: positive
-: negative
<: less than
>: greater than

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