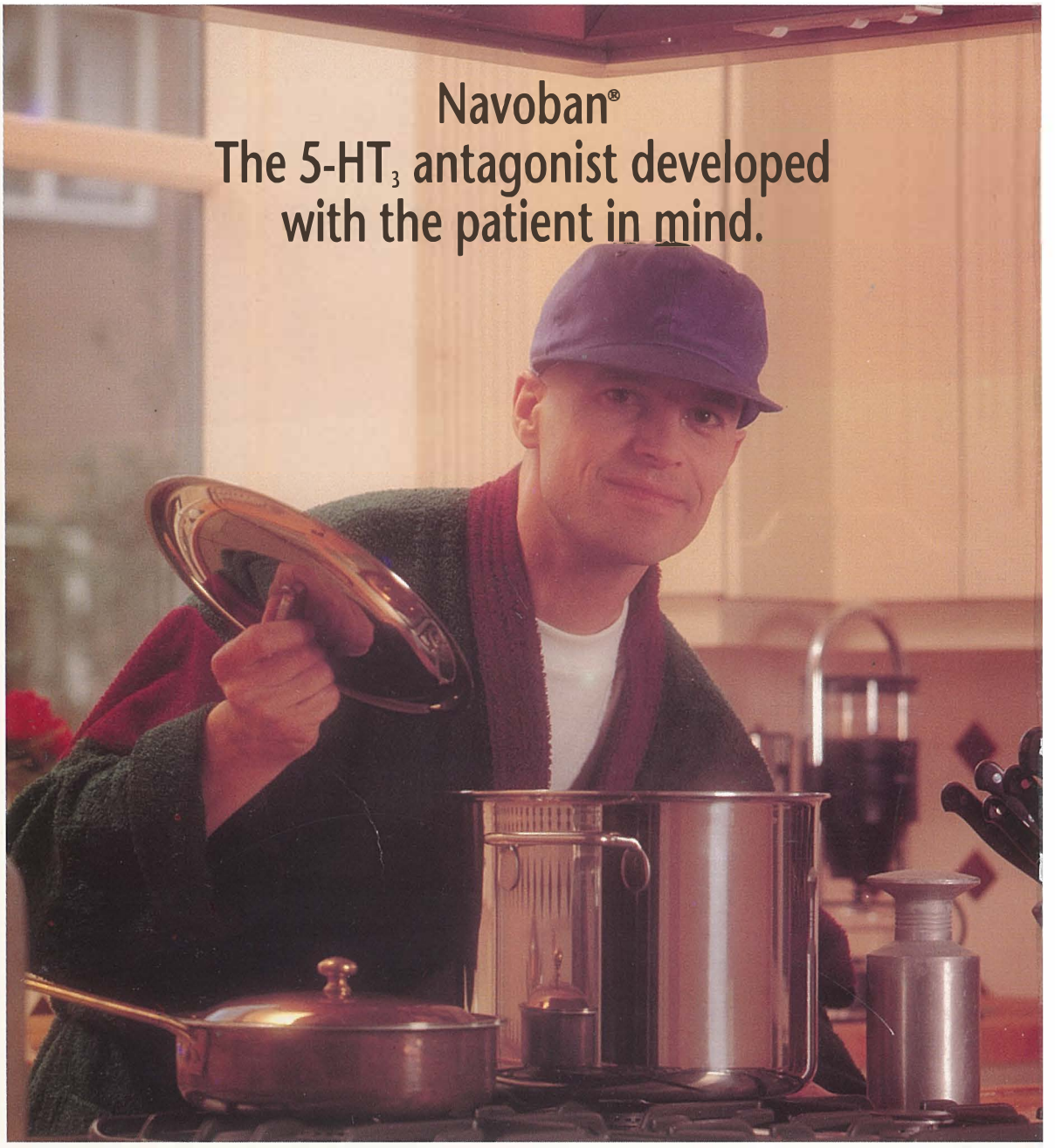
RADIOLOGY AND ONCOLOGY

1994
Vol. 28 No. 1
Ljubljana

ISSN 1318-2099
UDC 616-006



Navoban® (tropisetron)

Always once a day. Always 5 mg.

SANDOZ

Sandoz Pharma Ltd, Pharma Basle Operations, Marketing & Sales, CH 4002 Basle/Switzerland

RADIOLOGY AND ONCOLOGY

Established in 1964 as ***Radiologia Iugoslavica*** *in Ljubljana, Slovenia.* ***Radiology and Oncology*** *is a journal devoted to publication of original contributions in diagnostic and interventional radiology, computerized tomography, ultrasound, magnetic resonance, nuclear medicine, radiotherapy, clinical and experimental oncology, radiophysics and radiation protection.*

Publishers
Slovenian Medical Society – Section of Radiology,
Croatian Medical Association – Croatian Society of Radiology

Affiliated with
Societas Radiologorum Hungarorum
Friuli-Venezia Giulia regional groups of S.I.R.M.
(Italian Society of Medical Radiology)

Correspondence address
Radiology and Oncology
Institute of Oncology
Vrazov trg 4
61000 Ljubljana
Slovenia
Phone: +386 61 1320 068
Fax: +386 61 1314 180

Reader for English
Olga Shrestha

Design
Monika Fink-Serša

Key words und UDC
Eva Klemenčič

Secretaries
Milica Harisch
Betka Savski

Printed by
Tiskarna Tone Tomšič, Ljubljana, Slovenia

Published quarterly

Bank account number 50101 678 48454
Foreign currency account number
50100-620-133-27620-5130/6
LB – Ljubljanska banka d.d. – Ljubljana

Subscription fee for institutions 100 USD, individuals 50 USD.
Single issue for institutions 30 USD, individuals 20 USD.

According to the opinion of the Government of the Republic of Slovenia, Public Relation and Media Office, the journal RADIOLOGY AND ONCOLOGY is a publication of informative value, and as such subject to taxation by 5 % sales tax.

Indexed and abstracted by
BIOMEDICINA SLOVENICA
CHEMICAL ABSTRACTS
EXCERPTA MEDICA/ELECTRONIC PUBLISHING DIVISION

CONTENTS

DIAGNOSTIC AND INTERVENTIONAL RADIOLOGY

ULTRASOUND AND CT

ONCOLOGY

The publication of the journal is subsidized by the Ministry of Science and Technology of the Republic Slovenia.
Contributions of Institutions:
Inštitut za diagnostično in intervencijsko radiologijo, KC Ljubljana; Klinika za otorinolaringologijo in maksilofacialno kirurgijo, KC Ljubljana; Klinički zavod za dijagnostičku interventnu radiologiju, KBC Rebro, Zagreb; Onkološki inštitut, Ljubljana

Radiol Oncol 1994; 28: 5-9.

Radiological presentation of pulmonary hamartomas

Hrvojka Soljačić,[1] Zlata Herceg[2] and Zoran Slobodnjak[3]

[1] Clinic for Lung Diseases Jordanovac, Zagreb, Croatia, [2] Clinic for Lung Diseases Jordanovac, Zagreb, Croatia; Clinical Institute for Thoracic Radiology Clinic for Thoracic Surgery, [3] Clinic for Lung Diseases Jordanovac, Zagreb, Croatia

A review of thirty-seven cases of pulmonary hamartomas, relatively rare benign tumors of the lung, is presented. In all cases hamartomas were discovered radiologically and confirmed cytologically and pathohistologically before and/or after surgery. The prevalence of hamartoma was established in male patients (males vs. females, 2:1) and in older age group. There were no sex and age related differences with respect to the size or location of tumor.

Key words: lungs neoplasms-radiography; hamartoma; human

Introduction

Hamartomas are benign tumors of the lung. These tumors usually consist of all histologic elements that are present in normal mature pulmonal tissue. They are composed of hyaline, cartilage (sometimes with calcifications), lipomatous and fibrous tissue with cleft-like spaces lined with low columnar epithelium. The tumors are often surrounded by a layer of compressed alveolar tissue.[1]

The etiology of hamartomas is still unknown. There are opinions that these tumors are congenital by origin, or are a result of hyperplasia of normal structures with resultant tumor formation.

Pulmonary hamartomas are very rare, and most series in the literature involve small numbers of patients seen over a period of years or decades. E. g., McDonald et al.[2] found only 20 cases in 7970 necropsies; Rubin and Berckman[3] reported 28 cases in the total of 8000 postmortem examinations; Blair and McElvein reported 25 patients with pulmonary hamartoma during 36-year period. The same authors also mentioned that in all English medical literature up to 1963, a total od 200 hamartomas were reported. Toomes et al.[5] reviewed 74 cases in ten year period; Deodato et al.[6] observed 11 cases of these pulmonary tumors during the last ten years; Hansen et al.[7] diagnosed 89 cases during 25 years.

According to most previously citated authors, hamartoma in the lung occurs principally in the pulmonary parenchyma in the bronchi or epipleurally.[8]

The majority of hamartomas are asymptomatic and are found incidentally on chest X-ray examination, with typical coin lesion appearance. These are typical solitary pulmonary nodules

Correspondence to: Hrvojka Soljačić, MD, Clinic for Lung Diseases Jordanovac, Jordanovac 104, 41000 Zagreb, Croatia.

UDC: 616.24-006-073.75

that appear like a single opacity in the lung on radiography, with margins that are distinct enough to permit the measurement of diameter.

The main problem is to distinguish hamartomas from other coin lesions of the lung, specially those of malignant origin. Recently, the rapid progress of radiological diagnosis by transthoracic needle-biopsy has improved the possibility of correct diagnosis of hamartoma.[7, 9]

This is of great help to a surgeon. Namely, even where a lung nodule has been identified as a hamartoma, surgery is the treatment of choice for two reasons: Firstly, because of the possibility of nodular growth,[8] and secondly, because of possible, although rare, malignant alteration.[10, 11]

The purpose of this study was to point out the diagnostic value of hamartoma detection by radiological methods, as well as the possibility of diagnostic verification by percutaneous needle biopsies. Major characteristics of hamartomas are also presented.

Material and methods

The medical records and radiographs of all patients with pulmonary hamartomas, diagnosed at the Clinic for Lung Diseases Jordanovac from the beginning of 1983 to the end of 1992, were reviewied. There were in total thirty-seven of those patients, with age range from 18 do 68 years.

Diagnosis of hamartoma was based on radiologic examinations. All patients were submitted to radiography in two projections and to tomography of the lungs, both in upright position. Special attention was paid to the form, size, localization and structure of the tumor, as well as to its relation to the surrounding structures. By means of radiography in two projections it was possible to establish the form, size and localization of the tumor. In addition, the contours of the tumor in contrast to the surrounding structures as well as the shape of the tumor were presented by tomography.

In our own series, all hamartomas were distinctly delineated from the surrounding structures. Owing to the distinct border of the hamartomas in relation to its surrounding structures, primary pulmonal neoplasmas can be excluded. However, this remarkable property still allows for some other lesions, such as tuberculomas, echinococcal cysts, metastatic tumors and some other benign tumors in differential diagnosis.

The calcifications within tumors, which were found in some of our cases were centrally located. This localization does not imply the precise diagnosis of hamartoma, but significantly differs from irregularly arranged crumbly calcifications that are usually found within tuberculomas.

Therefore, after determining the precise localization of a solid, well circumscribed coin lesion with or without central calcification, the percutaneous needle biopsy was performed to establish a precise diagnosis of hamartoma.

Al patients were operated and the postoperative pathohistological examinations of resection specimens were done.

Results are presented as mean ± s.e.m.

Results

Pulmonary hamartomas were found in 24 (65 %) male (age range 21–65), and 13 (35 %) female (age range 18–68) patients.

As evident from Figure 1, hamartomas are more common in males and in older age groups.

The location and the size of hamartomas were identified on the basis of chest X-ray images. The majority of our patients' tumors were located in the pulmonary parenchyma, with no predilection for any one lobe. There were 9 hamartomas located in the right upper

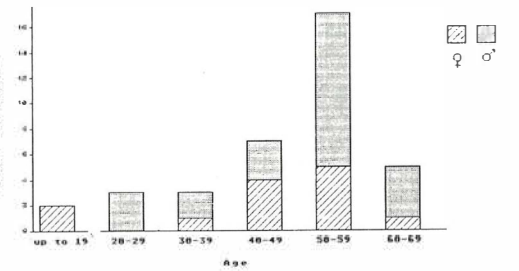


Figure 1. Age and sex distribution of pulmonary hamartomas in 37 patients.

lobe, 2 in the right middle lobe, 9 in the right lower lobe, 7 in the left upper lobe, 9 in the left lower lobe, and in one patient hamartoma was spread in the right upper and the right middle lobes.

On X-ray films, a hamartoma is usually imaged as a sharply demarcated coin lesion of ovoid shape (Figure 2 and 3).

In hamartomas with non-homogeneous structure calcifications are characteristic (Figure 4 and 5).

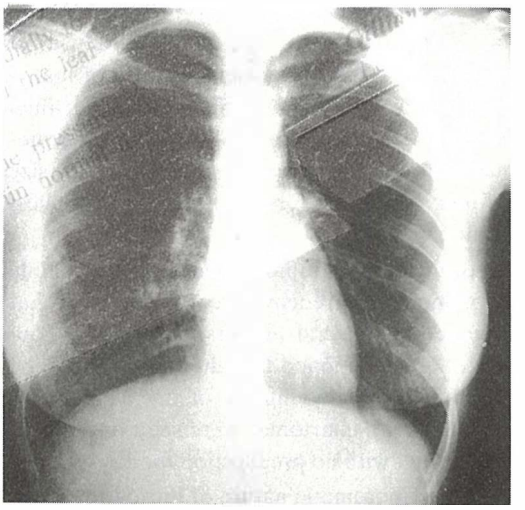


Figure 2. A PA chest view. Hamartoma of the right lung. (Arrow)

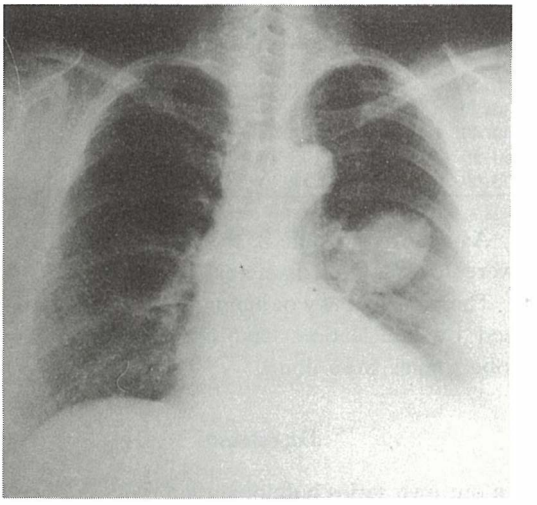


Figure 4. A PA chest view. Hamartoma of the upper left lobe.

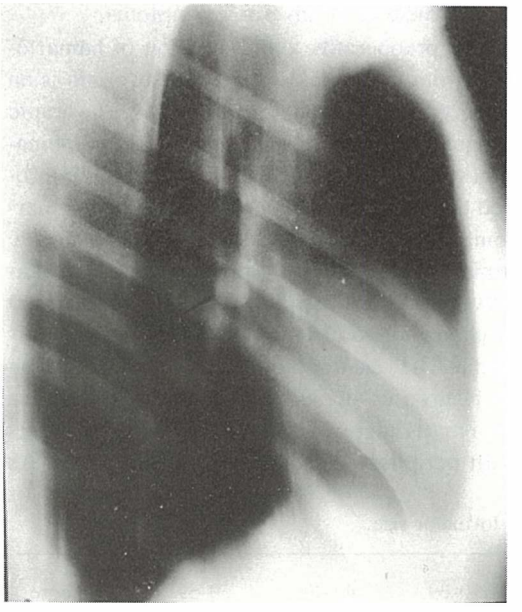


Figure 3. A lateral tomographic view. The same case as in Figure 2.

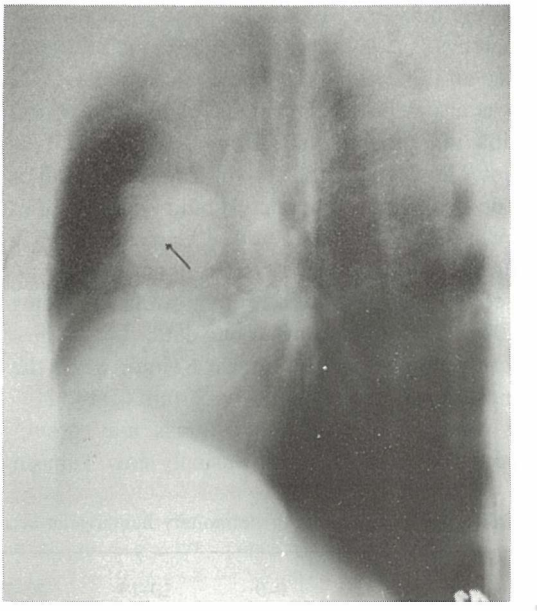


Figure 5. A lateral tomographic view. The same case as in Figure 4. Note a characteristic calcification in hamartoma. (Arrow)

Of two diameters, transverse diameter was taken into account, as is usual in literature.

From the results shown on Table 1, there was no correlation between the size of tumor and patients' age.

Table 1. The size ($\bar{x} \pm$ s.e.m.) of pulmonary hamartomas in 37 patients in relation to age.

Age	No. of patients	Tumor diameter (mm)
up to 19	2 (5.40%)	51.00±31.00
20–29	3 (8.11%)	22.66±4.81
30–39	3 (8.11%)	16.67±3.28
40–49	7 (18.92%)	21.14±2.17
50–59	17 (45.95%)	25.05±3.41
60–69	5 (13.51%)	26.60±9.62

As shown on Table 2, the most hamartomas were 10–29 mm in diameter.

The vast majority of hamartomas were removed by enucleation, and only in two cases lobectomies were done.

Discussion

In our own series pulmonary hamartomas were mainly diagnosed in patients between the fourth and seventh decades (Figure 1). These data are in accordance with many others.[6,7,12] As in our study, only few cases were reported in younger persons.[13] A greater prevalence in male patients shown in our material (males vs. females, 2:1) was also reported by others,[4,7,14] with a variation in preponderance from 2:1 to 3:1. It is not known whether this difference is real or due to a higher frequency of pulmonary diseases in males, with a discovery of hamartoma being incidental.[7] The usual size of hamartomas ranged between 20 and 40 mm,[6,7] rarely exceeding 100 mm.[6,8] According to our data, the smallest lesion was 3 mm, the largest 82 mm, while the most usual size was about 10–30 mm (Table 2).

Although benign, hamartomas may grow,[8] but the growth of tumor is usually slow. Hansen et al.[7] recorded growth in 48% of follow up cases.

Although in our cases a correlation between the age and size of tumor was not recorded (Table 1), a positive correlation between these two parameters has been shown by Hansen et al.[7]

Studying some differences between benign and malign lesions of the lung, Zwirewitz et al.[9] reported that the mean diameter of malignant lesions is usually significantly greater than that of benign nodules. So, large cell carcinomas showed the greatest overall mean size (57 mm ± 23) and hamartomas the smallest (17 mm ± 11).

Localization of tumor in our patients mainly in pulmonary parenchyma corresponds to that observed by several authors.[5,7,15] While Toomes et al.[5] mentioned that the right upper lobe was the most frequent site of involvement, in our patients hamartomas were seen in all parts of the lung, with no predilection for any lobe.

The asymptomatic nature of these tumors, as registered in our patients, was not surprising because – owing to their localization and size – they could rarely produce a compression of vital structures to give any symptoms.

The preoperative differentiation of hamartomas from the nodules of malignant origin is an important task for a radiologist. The rapid progress of radiological diagnosis in combination with transthoracic needle biopsy has improved the possibility of proper diagnosis of hamartoma.[7,16] In the past decades thoracic needle aspiration biopsy, as a specific and highly sensitive procedure, has been considered a method of choice for confirming the diagnosis of hamartoma. The accuracy of this method according to Hansen et al.[7] was 85 percent, while in our own material it was about 80 percent in patients with peripheral localization of hamartoma. As

Table 2. The incidence of pulmonary hamartoma according to tumor size.

Transverse diameter (mm)	0–9	10–19	20–29	30–39	40–49	50–59	60 or more
No. of patients	1	15	11	6	0	2	2
%	2.69	40.54	29.73	16.22	0	5.41	5.41

mentioned above, the diagnosis of hamartoma in all our cases was proved by postoperative histological analysis.

A precise preoperative radiological diagnosis with microscopic verification enables the surgeon to choose an adequate type of resection. If the tumor is well circumscribed, the surgeon will attempt to use local resection or excision. This approach was mainly used in our patients. However, if that was not the case, the method of choice would be lobectomy (Figure 4 and 5).

According to some opinions, hamartoma, although benign tumor, must be operated immediately after radiologist's diagnosis to reduce time delay and diagnostic errors to minimum.[17] Another reason is to minimize the possibility of tumor growth or its malignant alteration.[5,10,18]

On the contrary, according to Hansen et al.,[7] since most hamartoma can be diagnosed radiologically with high accuracy, surgery is required only in the cases of continuous expansion of the tumor, pulmonary symptoms and when malignancy cannot be excluded. If no tumor growth has been observed, surgical treatment of hamartoma is not necessary, although there is a need for continuous follow up of those patients.

References

1. Robbins SL. Neoplazme. In: *Patologijske osnove bolesti*. Zagreb: Školska knjiga, 1987: 118–61.
2. McDonald JR, Harrington SW, Clagett OT. Hamartoma (often called chondroma) of the lungs. *J Thorac Surg* 1945; **14:** 128–43.
3. Rubin M, Berckman J. Chondromatous hamartoma of the lung. *J Thorac Surg* 1952; **23:** 393.
4. Blair TC, McElvein RB. Hamartoma of the lung. *Dis Chest* 1963; **44:** 296–303.
5. Toomes H, Delphendahl A, Manke HG, Vogt-Moykopf I. The coin lesions of the lung. *Cancer* 1983; **51:** 534–37.
6. Deodato G, Messina S, Chisari A, Nicolasi M, Piccirillo P, Compagnone S, Tornambene F, Torre T. Hamartochondroma of the lung. Apropos 11 cases. *Minerva Chir* 1991; **46** (19): 1019–25.
7. Hansen PC, Holtveg H, Francis D, Rasch L, Bertelsen S. Pulmonary hamartoma. *J Thorac Cardiovasc Surg* 1992; **104:** 674–8.
8. Stulz P, Dalquen P. Unusual localization of a pulmonary hamartoma. Case Report. *Thorac cardiovasc Surgeon* 1991; **39:** 55–7.
9. Zwirewich CV, Vedal S, Miller R, Muller LN. Solitary pulmonary nodule: High resolution CT and radiologic-pathologic correlation. *Radiology* 1991; **179:** 469–76.
10. Hayward RH, Carabasi J. Malignant hamartoma of the lung. *J Thorac Cardiovasc Surg* 1967; **51:** 457–66.
11. Cavin E, Masters JH, Moody J. Hamartoma of the lung. *J Thorac Surg* 1958; **35:** 816–20.
12. Bateson EM. So-called hamartoma of the lung: a true neoplasm of fibrous connective tissue of the bronchi. *Cancer* 1973; **31:** 1458–67.
13. Oldham HN, Young WG, Scaly WC. Hamartoma of the lung. *J Thorac Cardiovasc Surg* 1967; **53:** 735–49.
14. Koutras P, Urschel HC, Paulson DL. Hamartoma of the lung. *J Thorac Cardiovasc Surg* 1971; **61:** 768–76.
15. Fudge TL, Ochsner JL, Mills NL. Clinical spectrum of pulmonary hamartomas. *Ann Thorac Surg* 1980; **30:** 36–9.
16. Hamper UM, Khouri NF, Stitik FP, Siegelman SS. Pulmonary hamartoma: diagnosis by transthoracic needle-aspiration biopsy. *Radiology* 1985; **155:** 15–8.
17. Vrančš N, Slobodnjak Z. Kirurško liječenje hamartoma pluća. *Liječ Vjesn* 1989; **111:** 461–2.
18. Karasik A, Modan M, Jacob CO. Increased risk of lung cancer in patients with chondromatous hamartoma. *J Thorac Cardiovasc Surg* 1980; **80:** 217–20.

Radiol Oncol 1994; 28: 10-4.

Side-effects and complications of percutaneous transluminal angioplasty of the subclavian artery: Analysis of 55 cases

Andrija Hebrang,[1] Josip Mašković,[2] Ivan Drinković[1]

[1] *Clinical Hospital »Merkur«, Zagreb, Croatia*
[2] *Clinical Hospital Split, Split, Croatia*

The complications and side-effects of percutaneous transluminal angiplasty (PTA) of the subclavian artery are presented. In the group of 55 patients 46 (83.6 %) had stenosis and nine (16.4 %) had the obliteration of the subclavian artery. The pathological changes were established by clinical examination and angiography of the supraaortal vessels. The patients were 38–68 years old (mean age 57 years). The first group comprising 30 patients did not receive heparin, while the other 25 patients received 5.000 units of heparin by catheter immediately before dilatation. The procedure was successful in 48 out of 55 patients (87.3 %). The complications and side-effects were registered in 17 patients (30.9 %). Among the most common were: transient thoracic pain (6 patients, 10.9 %), transitory ischemia of the fingers in 3 (5.5 %), and one groin hematoma (1.8 %). The symptoms of transitory vertebrobasilar insufficiency were registered in seven patients (12.8 %), in four of them vertigo and in three headache. There were no signifficant differences in the number of complications between the group treated with heparin (7 patients, 28 %) and the group without heparin treatment (10 patients, 33,4 %). We conclude that the complications and sideeffects of PTA of the subclavian artery are less common then in the operative treatment, and point out PTA as the method of choice in the treatment of the subclavian artery stenosis or obliteration. The use of heparin does not influence the incidence of complications.

Key words: Subclavian artery; angioplasty, PTA; complications, balloon – adverse effects; heparin use

Introduction

Percutaneous transluminal angioplasty (PTA) of the subclavian artery is established as a method of choice for the treatment of stenoses proximal or distal to the origin of the vertebral artery. Among other authors, we have reported on very good long-term results during a period of 6 to 48 months in 52 patients.[1] Until now, many details about the technique have been published.[2–4] Meanwhile, the risk of complications during the dilating of stenoses near the

Correspondence to: Prof. Andrija Hebrang, MD, PhD, Department of Radiology, Clinical Hospital »Merkur«, Zajčeva 19, 41000 Zagreb, Croatia.

UDC: 616.134-007.271-089

origin of the vertebral artery exists. Although the problem of crossing the origin of the vertebral artery by a balloon catheter has been described,[5] the vertebral artery can be obstructed and the symptoms of vertebrobasilar insufficiency can appear. Also, the dilatation of proximaly sited plaques can be the source of distal embolism, including the embolism of the brain through vertebral artery. There are no analyses of complications in great series reported in the literature. Here we present an analysis of the side effects and complications after PTA of the subclavian artery in 55 patients.

Material and methods

For this study we analyses PTA of subclavian artery in 55 patients, performed in two different centers. In all cases the same indications, technique and follow-up procedure were used. The patients were 38–68 years old (mean age, 57 years).

The indication for PTA was stenosis of the subclavian artery in 46 patients (83.6%), and an occlusion in nine patients (16.4%). The main symptom registered before treatment was brachial ischemia in all patients. Thirty nine patients (70.9%) had symptoms of the vertebrobasilar insufficiency like dizziness, ataxia, blurred vision or a combination of these symptoms. Clinical investigations included blood pressure measurement on both arms, Doppler sonography and/or oscilography. As a definitive diagnostic method angiography of the supraaortic vessel was performed. The patients were examined by conventional transfemoral angiography or by transbrachial DSA. Initially, an aortic arch angiography was made to establish the location of stenosis. After the stenosis had been presented, selective studies of both carotid and vertebral arteries were carried out. The aim of selective angiographic studies was to present the intracranial circulation and the role of a stenosed subclavian artery in brain irrigation. Accepting the rule that only the vertebral artery should not be obliterated by a balloon catheter, one patient with stenosis of the subclavian artery of near vertebral origin and obliteration of the contralateral vertebral artery were excluded from further procedure. In the analyzed group, the stenosis of the subclavian artery was located proximally to the origin of the vertebral artery in 27 patients (49.1%), causing the subclavian steal syndrome with retrograde blood flow in the ipsilateral vertebral artery. Stenoses in another 19 patients (34.5%) were located distally to the origin of the vertebral artery. Retrograde vertebral flow was found in all nine patient with obliteration of the subclavian artery.

We used the PTA technique described in the literature.[1,2,4] In 52 out of 55 patient the transfemoral approach was performed. In three patients with occluded femoral arteries PTA was conducted via the axillary artery. After passing the stenosis or occlusion with a 160-centimeter long guide wire, the balloon catheter was introduced. The width of the balloon, ranged between 8 and 10 millimeters, depending on the diameter of the non-affected part of the subclavian artery. The length of the balloon was 2 to 4 centimeters and the pressure of 5 to 7 amtpospheres was used. A 160-centimeter guide wire was left in the angioplasty catheter during the procedure to prevent the balloon from mooving. The first group of 30 patients did not receive heparin. The other 25 patients were given 5.000 units of heparin throught angioplasty catheter before dilatation. After dilatation procedure and balloon emptying, the catheter was withdrawn and follow-up angiography performed. All side-effects and complications were registered during 24 hours after procedure.

Results

Technical success immediately after procedure was established by follow-up angiography and by the measurement of blood pressure on the affected side and on the contralateral hand. A blood pressure gradient between both arms under 10% was considered as a good result. The follow up angiography of successfully dilated arteries immediately after the dilatation

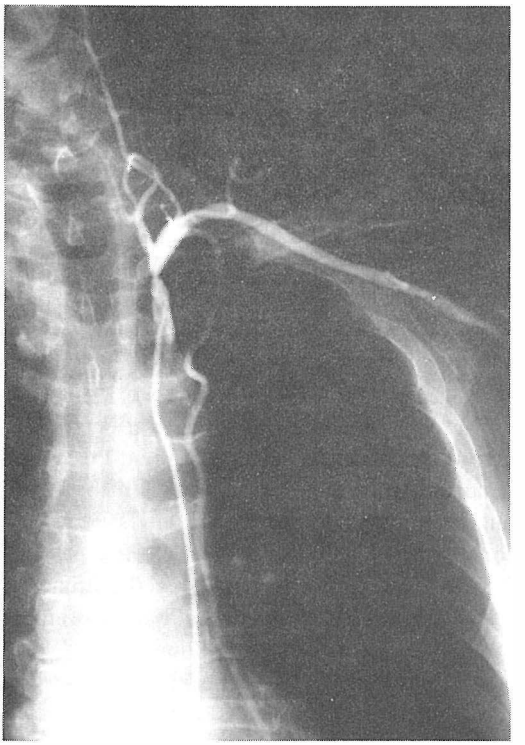


Figure 1a. Significant stenosis of the left subclavin artery proximal to the vertebral origin.

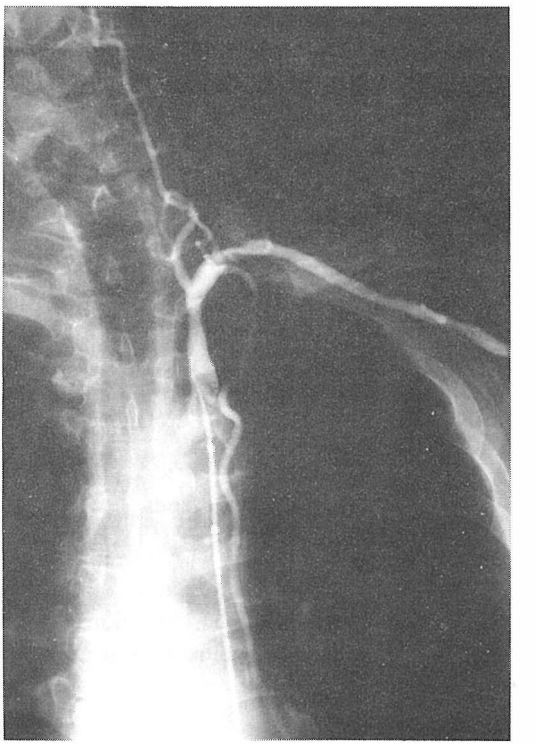


Figure 1b. After PTA normal lumen of the subclavian artery is presented.

showed the normal lumen or the remaining narrowing not greater than 30% of the lumen (Figure 1a, b). Using these criteria, we found satisfactory technical result in 43 out of 46 patients with stenosed (93.5%) and in five out of nine patients with occluded subclavian arteries (55.6%).

Side-effects and complications in the examined group were registered in 17 out of 55 patients (30.9%). Seven out of seventeen side-effects and complications occurred in the patients treated with heparin and ten in the patients without this treatment (Table 1).

Several different side-effects and complications are presented in our results. Local pain in the site of stenosis during dilatation occurred in six patients (10.9%). The pain was of short duration and did not require therapy. A transitory headache and vertigo occurred in three and four patients, respectively. The distal brac-

Table 1. Types and number of side-effects and complications in heparin treated and untreated groups during PTA of the subclavian artery.

Side-effects & complications	Heparin treated (25 patients)		Heparin untreated (30 patients)		Total	
	N	%	N	%	N	%
Thoracic pain	2	8.0%	4	13.3%	6	10.9%
Headache	1	4.0%	2	6.7%	3	5.5%
Vertigo	2	8.0%	2	6.7%	4	7.3%
Hand Ischemia	1	4.0%	2	6.7%	3	5.5%
Groin Hematoma	1	4.0%	0	–	1	1.8%
Total	7	28.0%	10	33.4%	17	30.9%

hial ischemia in three of our cases appeared as transitory pain in the fingers (duration half to one hour). The pain disappeared without treatment. One groin hematoma was registered 24 hours after treatment in a patient who received heparin during PTA procedure. The hematoma had eight centimeters in diameter. After symptomatic therapy, hematoma disappeared in ten days. In total, 17 out of 55 patients showed one of the side effects or complications all of which were transitory. Taking in to account all compolications, there were no significant differences between the heparin-treated and untreated groups of patients ($p < 0.001$).

Discussion

PTA of the subclavian artery is a well established procedure which carries a low risk of complications in comparison with surgery.[6–8] The incidence of complications during and after surgical treatment of subclavian stenosis includes pneumothorax, thrombosis, chylothorax, pleural effusion or infection in 23% of patients.[9] In the recent history the complications of PTA has not been clearly defined. For a long time the radiologists have been hesitant to use PTA to treat the great supraaortal arteries for the fear of possible embolisation of the brain and cerebral ischemia during balloon inflation.[3,4] Especially the risk associated with the treatment of lesions proximal to the vertebral artery origin has been pointed out. In the beginning indications for PTA included stenoses of the subclavian artery located distally to the vertebral artery, or stenoses in patients with reverse blood flow in the vertebral artery, which prevents distal vertebral embolisation. Later reports indicated that after PTA of the subclavian stenosis antegrade flow in vertebral artery is delayed for several seconds to several minutes, which functions as a protective mechanism against distal embolisation in proximal situated subclavian stenoses.[10] The next problem was transitory occlusion of the vertebral artery during balloon inflation. Vitek showed that such occlusion had no influence on the vertebral circulation.[5]

Our results show three kinds of complications. The first one, transitory thoracic pain in the dilatation site is well known during the treatment of other arteries. The second group of complications included headache and vertigo as the sings of transitory vertebrobasilar ischemia. These symptoms occurred in seven patients, and all of them had stenoses near the vertebral origin. Probably, the symptoms were caused by the inflated balloon over the vertebral origin during the procedure. Although the symptoms of vertebrobasilar insufficiency were transient, we emphasize the importance of angiographic analysis of all supraaortal vessels before therapeutic angioplasty. The obliteration of the opposite vertebral artery may cause significant vertebral ischemia due to temporary occlusion of the only vertebral artery. The possible damage of the plaque near the vertebral origin, or rupture of the wessel, must be avoided by the long guide sited deeply in the axilar artery during the dilatation procedure (Figure 2).

The third group of complications included distal ischemia of the fingers and groin hematoma. The general opinion is that the embolisation of distal arteries can be avoided using a systemic heparinisation. On the other hand, heparin can cause a higher incidence of groin hematoma. Taking into account the possible risks of systemic anticoagulation, especially in patients with liver disease and thrombocytope-

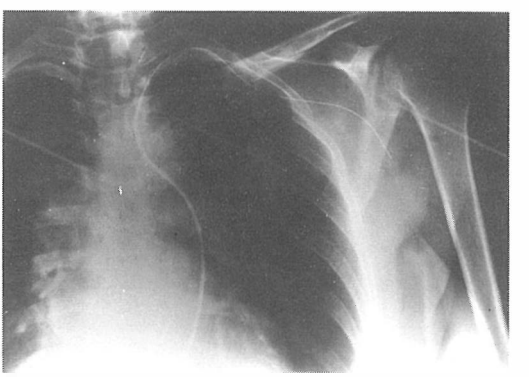


Figure 2. During catheterisation a long safety guide is introduced deeply into the axillary aftery.

nia,[11] some authors did not use heparin.[4,12] Our results show no difference between the group of heparin treated and the group of untreated potients. The only side-effect related to the use of heparin was a case of groin hematoma.

In counclusion, we suggest PTA of the subclavian artery as a method of choice for the treatment of stenoses located proximally, or distally to the origin of the vertebral artery. Because of the transitory signs of the vertebrobasiler insufficiency, the circulation in all supraaortal arteries has to be analysed before angioplasty. We did not have any patients with stenoses located in the origin of the vertebral artery, but some authors showed that the fracture of the plaque in that site may cause the obliteration of the vertebral artery.[13] Finally, according to our results, the severity or incidence of complications in the group of heparin treated and the group of untreated patients are the same.

References

1. Hebrang A, Mašković J, and Tomac B. Percutaneous transluminal angioplasty of the subclavian arteries: Long-term results in 52 patients. *AJR* 1991; **156**: 1091–4.
2. Motarjame A, Keifer JW, and Zuska AJ. Percutaneous transluminal angioplasty of the brachiocephalic arteries. *AJR* 1982; **138**: 457–62.
3. Gordon RL, Haskell L, Hirsch M, Shifrin E, Weinman E, and Romanoff H. Transluminal dilatation of the subclavian artery. *Cardiovasc Intervent Radiol* 1987; **8**: 14–9.
4. Vitek JJ, Keller FS, Duvall ER, Gupta KL, and Chandra-Sekar B. Brachiocephalic artery dilatation by percutaneous transluminal angioplasty. *Radiology* 1986; **158:** 779–85.
5. Vitek JJ. Subclavian artery angioplasty and the origin of the vertebral artery. *Radiology* 1989; **170**: 407–9.
6. Galichia JP, Bajaj AK, Vine DL, and Roberts RW. Subclavian artery stenosis treated by transluminal angioplasty: six cases. *Cardiovasc Intervent Radiol* 1983; **6**: 78–81.
7. Ringelstein EB and Zeumer H. Delayed reversal of vertebral artery blood flow following percutaneous transluminal angioplasty for subclavian steal syndrome. *Neuroradiology* 1984; **26**: 189–98.
8. Burke DR, Gordon RL, Mishkin JD, McLean GK, and Meranze SG. Percutaneous transluminal angioplasty of the subclavian arteries. *Radiology* 1987; **164**: 699–704.
9. Beebe HG, Stark R, Johnson JL, Jolly PC, and Hill LD. Choises of operation for subclavian–vertebral arterial disease. *Am J Surg* 1980; **139**: 616–23.
10. Damuth HD, Diamon AB, Rappaport AS, and Renner JW. Angioplasty of subclavian artery stenosis proximal to the vertebral origin. *AJNR* 1983; **4**: 1239–42.
11. Robertson HJ. Blood clot formation in angioplastic syringes containing nonionic contrast media. *Radiology* 1987; **163**: 621–2.
12. Motarjame A, Keifer JW, and Zuska AJ. Percutaneous transluminal angioplasty of the vertebral arteries. *Radiology* 1981; **139**: 715–7.
13. Losordo D, Rosenfield AK, Pieczek A, Baker K, Harding LM, and Isner J. How does angioplasty work? Serial analysis of human iliac arteries using intravascular ultrasound. *Circulation* 1992; **86**: 1845–58.

Radiol Oncol 1994; 28: 15-8.

The scimitar syndrome and partial anomalous pulmonary venous drainage to the azygos vein

Zdravko Borković,[1] Filip Kovačević,[1] Vjekoslava Raos,[2] Maja Strozzi,[2] Branko Radanović[3]

Nova bolnica Zagreb, Department of Radiology,[1] Department of Cardiology,[2] KBC Rebro, Zagreb,[3] Department of Radiology, Croatia

Scimitar syndrome and partial venous inflow to the azygos vein are very rare malformations of pulmonary venous return. Beside the anomalous pulmonary venous drainage to the inferior vena cava, other associated developmental anomalies of the lungs and pulmonary circulation often can be seen. The most frequent are the hypoplasia of the right pulmonary artery, abnormal pulmonary lobation and bronchial distribution, and altered pulmonary architecture. The diagnosis of the scimitar syndrome is made by the help of chest x-ray, CT scanning and catheterisation, and digital subtraction angiography.

Key words: scimitar syndrome; pulmonary veins; azygos vein

Introduction

Scimitar syndrome is a very rare congenital malformation of the pulmonary venous return to inferior vena cava.

Scimitar syndrome appears very rarely as an isolated pulmonary malformation. The most frequent associated anomalies are: hypoplasia of the right pulmonary artery, anomalous pulmonary lobation, alteration of the pulmonary structure and anomalies of the tracheobronchial system. Beside the anomalous drainage of the right pulmonary vein to the inferior v. cava, other variants of the pulmonary venous drainage can be seen.[1, 2]

Correspondence to: Zdravko Borković, M. D., Nova bolnica Zagreb, Department of Radiology, Aleja izvidača bb, 41000 Zagreb, Croatia.

UDC: 616.141-007.2

The anomaly is regularly detected on patient examination for another cause, or during a systemic examination. The patients have none or very few clinical symptoms.[1-4]

During the examination the mentioned anomaly could be suspected after careful inspection of chest x-ray and the completion of clinical and laboratory results.

The CT scanning of the chest is very helpful in completing the work-up. A definitive diagnosis and the most useful clinical information are obtained by means of digital subtraction angiography and catheterisation with blood gas analysis.

Case report

A 31-year old male was admitted to hospital for pains in the stomach, retrosternal oppres-

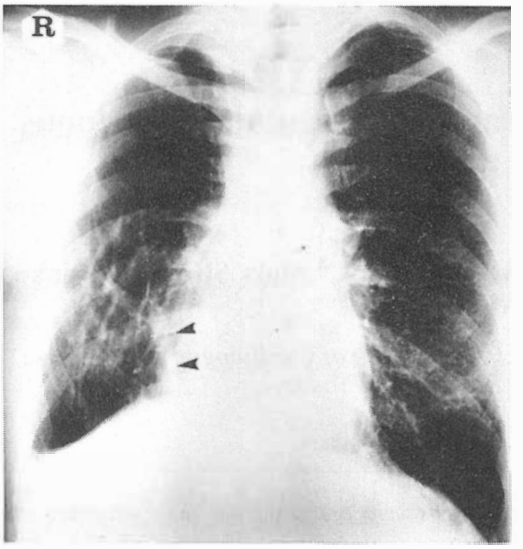


Figure 1. Anomalous venous drainage of the right lung (arrows).

sion and tachyarrhythmia until now healthy. The disturbances began with generalized weakness, retrosternal sensations, feeling of sickness, and pains in the epigastric region.

Cardiac techyarrhythmia is objectivised.

The laboratory findings were normal.

ECG: Atrial fibrillation with absolute arrhythmia 137 BMP, incomplete right fascicular block.

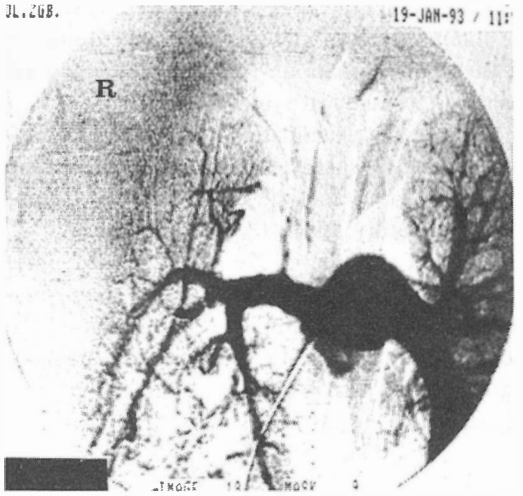


Figure 2. DSA of the pulmonary artery. Hypoplasia of the right pulmonary artery and irregular intrapulmonary arborisation.

After intravenous administration of 280 mg of Propafenon (Rytmonorm) a stabile conversion to sinus rhythm is achieved.

Chest x-ray: Asymmetrical chest due to smaller right side and with narrowing of the intercostal spaces of the same side. The right hilus is smaller. The pulmonary vascular ramification is rarified and irregular in the lower half of the right lung. A vascular shadow originating in the middle pulmonary field on the right side directed medially and distally reaches the diaphragm. Normal finding in the left lung. The configuration of the heart and aorta is also normal (Figure 1).

Cardiac echosonography: Normal valvular configuration, and right pulmonary regurgitation. The contractility and thickness of the myocard and the measures of cardiac cavities were normal. Paracardially on the right, a vascular structure was identified, indicative of a possible pathological communication.

No signs of possible intracardial shunt.

DSA: By means of digital subtraction angiography the pulmonary artery is visualized including its tributaries with normal arborisation on the left. Right pulmonary artery hypoplasia is found with a few irregular intrapulmonary tributaries, with accentuation in the proximal part (Figure 2).

In the venous phase on the left, a normal pulmonary vein inflow into the left atrium is seen. On the right side a wide abnormal vein draining the middle and inferior lung portions into subdiaphragmatic part on the inferior v. cava is seen (Figure 3).

CT: An altered pulmonary structure and an abnormal pulmonary lobation are found on the right. The right hilum is small in size. An anomalous pulmonary venous inflow into azygos vein is seen on the right side.

A wide anomalous pulmonary veins is seen passing between upper and lower pulmonary lobe extrapleuraly, and is directed medially to the inferior v. cava. The structure of the leaf lung is normal (Figure 4).

Right cardiac catheterisation: The pressures in the pulmonary circulaare within normal limits.

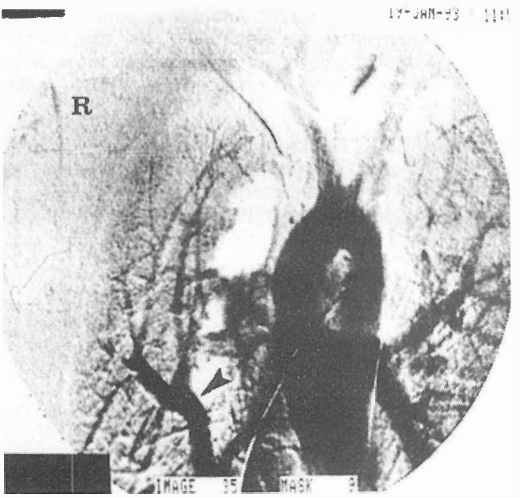


Figure 3. DSA of the lung – venous phase – Anomalous pulmonary venous drainage into the inferior v. cava (arrow).

Oxymetry: Elevation of the blood oxygen saturation in the inferior v. cava of 1,56 vol % which persists, although slightly decreased, in the right ventricle and atrium and in the pulmonaty artery. Systemic blood volume per minute was 8,600 L, pulmonary blood per minute was 13.5 L – a surplus of 4.9 L of blood per minute can be deduced, i. e. a relation of 1.5 + 1 in benefit to pulmonary v. systemic circulation is established.

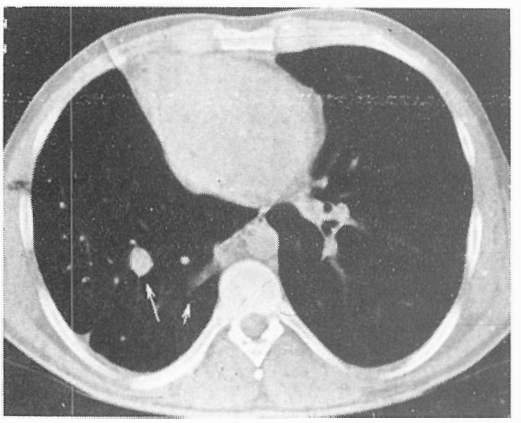


Figure 4. CT scan showing an anomalous vein (long arrow) and an anomalous vein draining into v. azygos (short arrow).

Discussion

Scimitar syndrome is a congenital anomaly of the pulmonary venous circulation in which an anomalous pulmonary vein drains to the inferior caval vein below the diaphragm.

This malformation is accompanied by right pulmonary arterial hypoplasia including its intrapulmonary tributaries, with anomalous pulmonary structure and lobation, including the alterations of bronchial arborisation.[1–7] In our case, we found scimitar syndrome associated with abnormal venous drainage of the right upper pulmonary lobe into the azygos vein. The lower pulmonary lobe is drained into the inferior v. cava. This anomaly features 0.4–0.7 % of all congenital heart anomalies.[1–3]

The anomaly is usually discovered incidentally on routine chest x-rays; in patients with dyspnoic symptomatology it is discovered during diagnostic work-up.

Definitive diagnosis is estabilished by means of chest x-ray and CT. An angiographic examination with presentation of thoracic arteries and veins with a typical finding of right pulmonary artery hypoplasia also including its tributaries, and the presentation of an anomalous vein definitely confirms the diagnosis.

Surgical correction of this anomaly which corresponds to an ASD with left-to-right shunt is usually not neccessary. In case of more prominent shunting, the anomalous vein is surgicaly implanted in the left atrium.

In our case, the consulting heart surgeon declined the neccessity of surgical correction of the anomaly, and suggested further periodical follow-up examinations. Surgical correction should be reestimated in the case of worsenned findings.

References

1. Godvin JD, Tarver RD. Scimitar syndrome: Four new cases examined with CT. *Radiology* 1986; **159:** 15–20.

2. Olson MA, Becker GJ. The scimitar syndrome: CT findings in partial anomalous pulmonary venous return. *Radiology* 1986; **159:** 25–6.

3. Schatz SL, Ryvicker MJ, Deutsch AM, Cohen HR. Partial anomalous pulmonary venous drainage of the right lover lobe shown by CT scans. *Radiology* 1986; **159:** 21–2.

4. Pennes DR, Ellis JH. Anomalous pulmonary venous drainage of the left upper lobe shown by CT scans. *Radiology* 1986; **159:** 23–4.

5. Julsrud P, Fellows KE. Anomalous pulmonary venous connection. In: Abrams HL ed. Abrams HL ed. *Abrams angiography: vascular and interventional radiology*. 3ed. Boston: Little Brown, 1983: 869–94.

6. Thorsen MK, Erickson SJ, Mewissen MW, Youker JE. CT and MR imaging of partial anomalous pulmonary venous return to the azygos vain. *J Comput Assist Tomogr* 1990; **14:** 1007–9.

Radiol Oncol 1994; 28: 19-22.

Ultrasonogram of gallbladder perforation

Nikola Ivaniš, Ivica Kraus, Mira Sever-Prebilić, Nada Brnčić-Dabo, Relja Perić, Aleksandar Včev

Department of Internal Medicine, University of Rijeka, Croatia

During the hospitalization of a female patient suffering from acute calculous cholecystitis the diagnosis of gallbladder perforation had been established. The perforation was suspected on the basis of clinical observation and laboratory tests, and the diagnosis established by conventional ultrasound examination. Emergency surgery confirmed the diagnosis of gallbladder perforation. Eight days after surgery, the patient was discharged from the hospital. Six months later, she was found to be symptom free. The conventional ultrasound proved to be an excellent method for following the course of severe acute calculous cholecystitis, which also enables early diagnosis of perforation. "Hole sign" in the gallbladder wall is important sign of perforation.

Key words: cholelithiasis – complications; gallbladder-ultrasonography; perforation; gallbladder perforation-gallstone-ultrasonic "hole sign"

Introduction

Acute and chronic cholecystitis are distinguishable both clinically and by the use of ultrasonography.

Acute cholecystitis is a disease of middle-aged and elderly individuals. Obese persons and diabetics are especially prone to contract the disease. It is a disease of very frequent occurrence, and under certain conditions and accompanied by the risk factors of lithogenesis it is on the increase, so that today it represents 10 % of all gallbladder diseases. In 90 % of the patients it occurs in connection and as a consequence of gallstones, only in whereas 10 % of the patients the inflammation is not associated with gallstones.

Correspondence to: Nikola Ivaniš, MD, V. M. Lenca 48, 51000 Rijeka, Croatia.

UDC: 616.366-003.217.7-06:616.366-007.43

Chronic cholecystitis is etiologically (in more than 60 % of the patients) most frequently associated with gallstones. It only seldom occurs as a progression of the acute into the chronic form. A frequent cause of the acalculous form of chronic inflammation is sepsis, cholesterolosis, adenomyomatosis and congenital anomaly of the gallbladder.

The sonographic signs of acute cholecystitis are: sonographic Murphy's sign, thickened gallbladder wall, and intraluminal echoes. Other signs, although less dependable, are: distended gallbladder, sonolucency of the wall, and irregularities of the outer margin of the gallbladder.

The calculous cholecystitis is characterized, besides other ultrasound signs, by solitary or multiple stones with an acoustic shadow, forming – with other changes – the characteristic WES trias (wall, echo, shadow).[1]

An important ultrasonographic sign of the gallbladder perforation is the pericholecystic fluid accumulation.[2-7] This finding, however, is not specific.[3,5] In recent times the so-called "hole sign" in the gallbadder wall of the patient with gallbladder perforation has been described.[8,9] "Hole sign" is sonographic finding of a hole in gallbladder wall.

Case report

This is a report on a 58-year-old female diabetic hospitalized at out Medical Clinic with right upper quadrant pains, nausea, vomiting, fits of shivering and fever up to 39 °C. The complaints had begun two to three days prior to hospitalization, after a meal of rich food. The patient had a two-year history of gallstone disease. The night after admission she experienced pain in the right shoulder, and on the following morning signs of localized peritoneal irritation in the right upper quadrant appeared, with febrility up to 39.6 °C. Laboratory tests showed accelerated SE 62/90, leukocytosis of 16.9 with deflection to the left, and blood sugar 8.2. All other findings were normal. On the emergency ultrasonogram of the abdomen a small quantity of free fluid was seen along the right lateral cavity. All this was interpreted as an acute cholecystitis with local peritoneal irritation. During the following three days the patient continued to feel pain in the right upper quadrant with febrility. An emergency scan of the peritoneal cavity was made. Intensive antibiotic therapy was applied. On the eighth day of hospitalization the patient suffered sudden intensive pain in the right upper quadrant and right shoulder with evident local rigidity, but this time also accompanied by pain in the right lower abdomen, and very poor peristalsis. Perforation of the gallbladder was suspected.

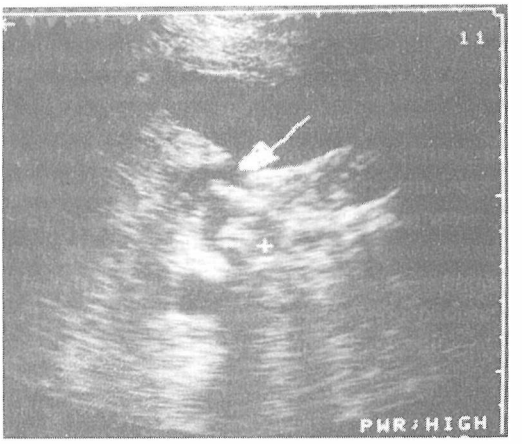


Figure 1. Site of gallbladder perforation ("hole sign").

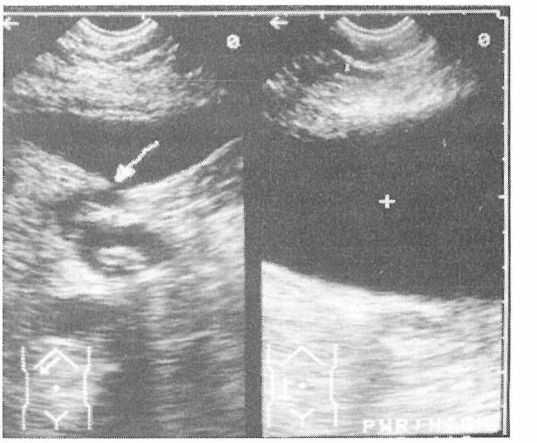


Figure 2. Site of gallbladder perforation (left), and unrestricted bile in the right lateral cavity (right).

An emergency ultrasonogram of the abdomen was made, which revealed free fluid along the right lateral cavity. The gallbladder was shown with thickened walls and stones, and a "hole sign" in the wall of the gallbladder corresponding to gallbladder perforation. In this way the ultrasound examination revealed the perforation of the gallbladder which required emergency surgery.

On opening the peritoneum, about two litres of bile mixed with fibrin platelets were removed. A hole disrupting the fundus of the gallbladder was found, from wich bile was leaking. In the gallbladder concrements were palpable, and the wall was extremely thickened. After preparation, antegrade cholecystectomy was performed. The content subdiaphragmatically right and paracollically right was evacuated, lavage of the peritoneal cavity made, drainage implanted and abdomen closed.

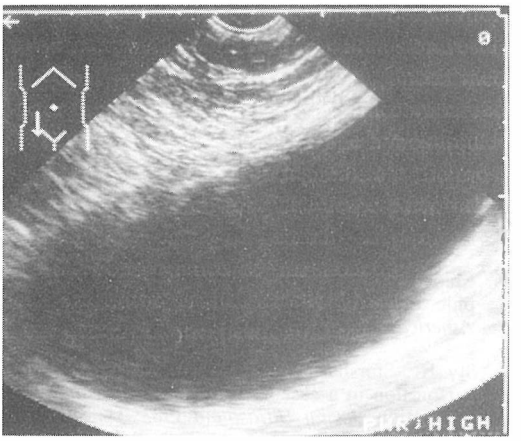


Figure 3. Unrestricted bile in the right lateral cavity.

The pathohistological finding was: gallbladder size 10×7 cm, wall thickness 1.5 cm, necrotic mucous mambranes, with one fistula seen at the fundus.

The postoperative course under the protection of antibiotics was uneventful, and the patient was discharged on the eighth day.

Discussion

In gastroenterology acute cholecystitis is a common disease. In most cases an adequate therapy will be sufficient to cure the disease without any complications. In some instances complications such as perforation of the gallbladder, pericholecystitic abscess and fistula in adjacent organs may develop. All these complications are the consequence of ischemia and necrosis of the gallbladder will.

The perforation of the gallbladder may be localized or unconfined. Localized perforation is confined by the omentum or adhesion resulting from previous inflammations. Unconfined perforation is not so common, but accompanied by a mortality of up to 30 %. The hypovascularity of the gallbladder fundus makes it the most likely site of perforation. The acute perforation is usually the cause of generalized peritonitis. A usual consequence of the subacute gallbladder perforation is the pericholecystitic abscess separated from the peritoneal cavity by adhesion. The chronic perforation into adjacent organs results in the creation of an internal biliary fistula. Fistulation occurs as a consequence of the spread of the inflammation to the adjacent tissue and organs. The most frequent is the fistula of the gallbladder with the duodenum, and less frequently with the hepatal flexure of the colon, stomach, jejunum, and the frontal gastric wall. With 5 % of the patients a clinically asymptomatic biliodigestive fistula can be found on cholecystectomy in the case of chronic inflammation of the gallbladder.

Ileus is the consequence of the migration of concrements through the duodenum or through the fistula into the duodenum. The impaction usually occurs at the ileocaecal valvula.

Through the routine use of ultrasound in detecting the etiology of pains in the right upper quadrant, it is possible in particular cases, to detect the complications of acute cholecystitis. As stated in the introduction, the ultrasound finding of pericholecystitic fluid is an important sign in the diagnosis of gallbladder perforation.[2-7] This sign, however, is not specific enough.[3,5] The most important sign is the "hole sign" in the gallbladder wall.[8,9]

When nonpulsatile, anechois tubular lesions are seen along the perforated gallbladder, differential diagnostics will suggest the consideration of a cyst, abscess, ascites and haematoma. The abscess will usually have fine irregular margins with some internal echoes.[10] The cyst, ascites and haematoma will have no connection with the gallbladder and adjacent organs. A standard sign of a biliary-enteric fistula is usually pneumobilia. The echogenicity of the fistula can be identified because of its reflexive lumen or content.

The diagnosis of the gallbladder perforation may present some difficuity as its symptoms and signs are hardly distinguishable from those of the acute cholecystitis without perforation. There are no laboratory findings indicating gallbladder perforation.

The methods of conventional radiology seldom yield valuable data on gallbladder perforation. In recent years ultrasonography has pro-

ved very useful in the preoperative diagnosis of gallbladder perforation.[8]

Helpful signs have been found to be the accummulation of pericholecystic fluid,[2-7] and the most useful "hole sign" which is very specific.[11] The presence of the pericholecystic fluid alone is not specific enough, as it may accompany also the inflammation of adjacent organs such as the peptic ulcer, pancreatitis, abscess and peritonitis.[3,5]

The case presented is a female patient whose gallbladder perforation had been detected by means of the ultrasonographic examination alone in the course of an acute calculous cholecystitis; the diagnosis was confirmed by consequent surgery.

Ultrasound examination is considered very useful in the cases of suspected gallbladder perforation.

Especially the "hole sign" is the most helpful ultrasonographic sign, because in combination with other ultrasound signs of acute cholecystitis it indicates subacute and chronic gallbladder perforation.

References

1. MacDonald FR, Cooperberg PL, Cohen MM. The "WES" triade specific sonographis sign in gallstones. Gastrointestinal *Radiology* 1981; **6:** 39–41.
2. Berman AB, Neiman HL, Kraut B. Ultrasonographic evaluation of pericholecystic abscess. *American Journal of Radiology* 1979; **132:** 201–4.
3. Crade M, Taylor KJN, Rosenfield AT et al. Ultrasonic imaging of pericholecystic inflammation. *JAMA* 1980; **244:** 708–10.
4. Deitch EA, Engel JM. Ultrasonic detection of acute cholecystitis with pericholecystic abscess. *Am Surg* 1981; **47:** 211–4.
5. Madrazo BL, Francis L, Hricak H et al. Sonographic findings in perforatio of the gallbladder. *American Journal of Radiology* 1982; **139:** 491–5.
6. Sty JR, Starshak RJ, Gorenstein L. Gallbladder perforation in a case of Kawasaki disease: Image correlation. *J Clin Ultrasound* 1983; **11:** 381–5.
7. Takada T, Yasuda H, Uchiyma K. Pericholecystic abscess: Classification of US findings to determine the proper therapy. *Radiology* 1989; **172:** 693–5.
8. Chau WK, Na AT, Feng TT et al. Ultrasound diagnosis of perforation of the gallbladder: Real time application and the demonstration of a new sonographic sign. *J Clin Ultrasound* 1988; **16:** 358–60.
9. Chen JJ, Lin HH, Chiu CT et al. Gallbladder perforation with intrahepatic abscess formation. *J Clin Ultrasound* 1990; **18:** 43–5.
10. Doust BD, Quiroz F, Stewart JM. Ultrasonic distinction of abscesses from other intraabdominal fluid collections. *Radiology* 1977; **125:** 213–5.
11. Chau WK, Wong KB, Chan SC, Na AT, Chan S, Wang JS, Wong CM. Ultrasonic "Hole Sign": A Reliable Sign of Perforation of the Gallbladder. *J Clin Ultrasound* 1992; **20:** 294–9.

Radiol Oncol 1994; 28: 23-5.

Calcaneal fracture and peroneal tendons injuries: CT analysis – case report

Krešimir Čavka,[1] Walter Cereda,[1] Lorenzo Imburgia,[1] Luigi Taddei[2]

[1] Department of radiology, [2] Department of surgery, Regional Hospital Beata Vergine, Mendrisio, Switzerland

A case of a 57 year old patient with labor accident is presented. CT examination of the foot was done immediatly after routine radiographs. CT showed fractures of the calcaneus with lateral subluxation of the left peroneal tendons. The findings were confirmed by surgery.

Key words: tomography, calcaneus, fracture, peroneal tendons, luxation, ankle, injuries

Introduction

Fractures of the talus and calcaneus may be difficult to evaluate using conventional imaging modalities.

Since 1980, computed tomography (CT) has been increasingly used for examination of the musculoskeletal system.[1–4] CT of the ankle is introduced as an effective technique in the evaluation of calcaneal fractures.

CT provided more information on acutely injured patients, and improved preoperative planning, postoperative follow-up, and detailed analysis of causes for chronic residual pain. CT further identified significant soft tissue injuries such as peroneal tendons displacement which cannot be delineated on plain films.[5,6,7]

Correspondence to: Krešimir Čavka, MD, PhD, Ospedale Regionale Beata Vergine, Department of Radiology, Via Turconi 23, 6850 Mendrisio, Switzerland.

UDC: 616.718.726-001.516-073.756.8

Case report

A 57 year old patient was admitted following a labor accident. He fell from the height of 5 m. Routine radiographs showed subluxation of the calcaneocuboid articulation and fracture of the calcaneus (Figure 1).

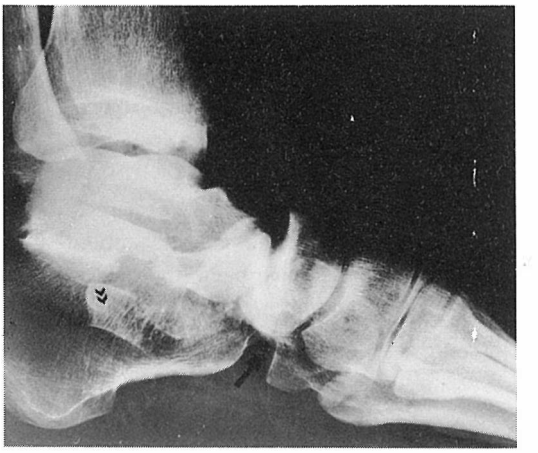


Figure 1. Lateral radiogram indicates subluxation of the calcaneocuboid (arrows) and talocalcaneal articulation with fracture of the calcaneus (arrowheads) and loss of the sinus tarsi.

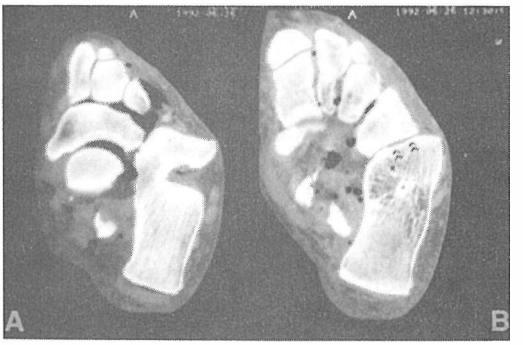


Figure 2a. CT scan in plantar plane shows free air in the cuneonavicular and talonavicular articulation with the sustentacular fragment (arrowheads). The calcaneus is displaced laterally and anteriorly.

Figure 2b. More caudal section shows subluxation of the cuboid bone (arrowheads).

CT examination of the foot was done immediatly after routine radiographs. The patient was studied on a GE-PACE scanner using a 512×512 matrix. The examination was done in supine position of the patient. The scanning plane was parallel to the plantar ankle to the plantar fat pad, with 4 mm spacing.

The CT examination of the left foot evaluated the complete luxation of the talocalcaneal articulation with fracture of the sustentaculum (Figure 2a). More caudal section showed subluxation of the calcaneocuboid articulation (Figure 2b). In addition, the suspicion of subluxation of the peroneal tendons (Figure 3) was made.

The findings were confirmed by surgery. During the operation the surgeon found that the peroneal tendons had been completly dislocated anteriorly of the lateral malleolus.

Reconstruction of the calcaneocuboid and talocalcaneal joints have been done (Figure 4). The peroneal tendons were fixated on the posterior part of the lateral maleollus.

Discussion

The foot and ankle are highly complex anatomic structures: there are at least 26 bones and 38 articulations in each foot.[8]

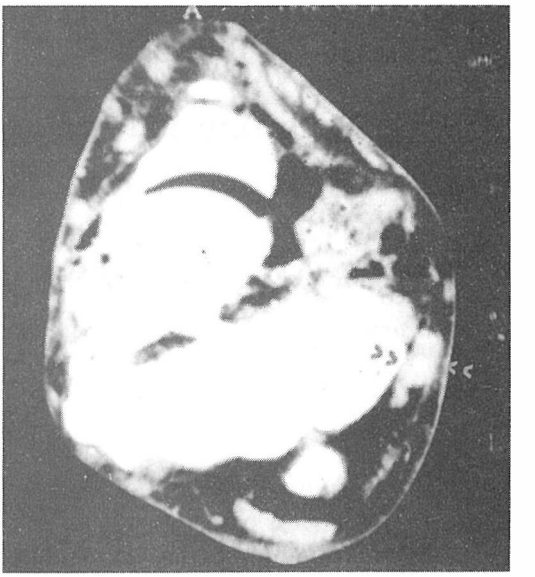


Figure 3. Most cranial section shows the lateral subluxation of the left peroneal tendons (arrowheads).

It is not always possible with conventional radiographic techniques to show foot pathology in the best way.

The main advantages of CT for planning a surgical intervention are a clear assesment of:

a) the size and number of fragments

b) the size and displacement of the sustentacular fragment

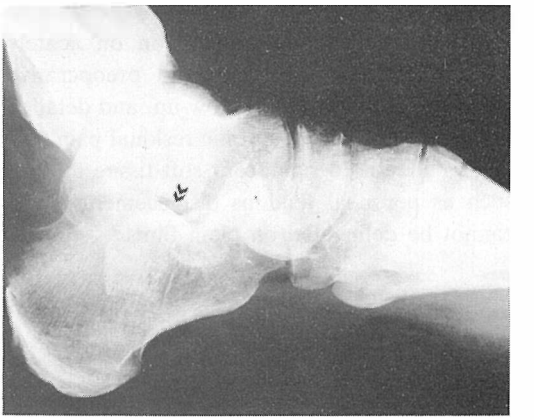


Figure 4. Postoperative lateral radiogram shows minimal subluxation of the calcaneocuboid articulation with normal relationship in talocalcaneal and talonavicular articulations. Normal sinus tarsi (arrowheads).

c) the presence of step and diastasis in the posterior facet.[9]

Injury of the peroneal tendons is one of the major complications of intraarticular calcaneal fractures, and has been difficult to diagnose by conventional radiography. In their retrospective review of 24 intraarticular calcaneal fractures, Rosenberg et al. have found acute peroneal tendon abnormalities in 22 cases (92%).[7]

References

1. Genant HK, Wilson JS, Bovill EG, Brunelle FO, Muray WR, Rodrigo JJ. Computed tomography of the musculosceletal system. *J Bone Joint Surg* (Am) 1980; **62:** 1088–101.
2. Heger L, Wulff K. Computed tomography of the calcaneus: normal anatomy. *AJR* 1985; **145:** 123–29.
3. Hubbard LF, McDermott JH, Garett G. Computed axial tomography in musculosceletal trauma. *J Trauma* 1982; **22:** 388–94.
4. Solomon MA, Gilula LA, Oloff LM, Oloff J, Compton T. CT scanning of the foot and ankle: normal anatomy. *AJR* 1986; **146:** 1192–203.
5. Bradley SA, Davies AM. Computed tomographic assessment of soft tissue abnormalities following calcaneal fractures. *BR J Radiol* 1992; **65:** 105–11.
6. Rosenberg ZS, Feldman F, Singson RD, Price GJ. Peroneal tendon injury associated with calcaneal fractures: CT analysis. *Radiology* 1986; **161:** 743–48.
7. Rosenberg ZS, Feldman F, Singson RD, Price GJ. Peroneal tendon injury associated with calcaneal fractures: CT findings. *AJR* 1987; **149:** 125–29.
8. Solomon MA, Gilula LA, Oloff LM, Oloff J. CT scanning of the foot and ankle: 2. clinical applications and review of the literature. *AJR* 1986; **146:** 1204–14.
9. Heger L, Wulff K, Seddiqi MSA. Computed tomography of calcaneal fractures. *AJR* 1985; **145:** 131–7.

Radiol Oncol 1994; 28: 26-33.

Prognosis of patients with non-Hodgkin's lymphoma (NHL), the influence of Kiel classification on survival

Berta Jereb[1], Janez Stare[2], Gabrijela Petrič-Grabnar[1], Janez Jančar[1]

[1] The Institute of Oncology, Ljubljana, [2] The Institute for Biomedical Information, Medical Faculty, University of Ljubljana, Slovenia

The effect of treatment modalities on the survival of patients with NHL and the prognostic impact of Rappaport's and Kiel classifications was analysed with the multivariate Cox model. Between 1978 and 1986, 482 adult patients received their first treatment for non Hodgkin's lymphoma at the Institute of Oncology in Ljubljana. We compared a group of 317 patients classified according to both Rappaport and Kiel classification (Group K) with a group of 165 patients classified according the Rappaport classification alone (Group R). Group K was divided into subgroups of high grade or low grade lymphomas, which again were analysed separately. The Kiel group patients had 1.5 times better chances for cure than the R- group. Stage was significant for the predicting of outcome. Chemotherapy and radiation significantly improve the survival in the high-grade (K) group but do not seem to have any bearing on the outcome in the low-grade (K) group of patients.

Key words. iymphoma, non-Hodkins; prognosis; Kiel classification

Introduction

In recent decades, considerable progress has been achieved in the management of patients with non Hodgkin's lymphoma (NHL). The biology of this disease with its multitude of biologic variations has become better understood.[1-10] New chemotherapeutic regimens have been introduced, enabling the treatment to be more "individualized"[11-18], i.e. adjusted to the aggressiveness of the disease.[19-26] As a result, the survival has improved, mainly in the group of patients with highly malignant lymphomas.[17] A number of new prognostic factors have been recognized. While the significance of some of these is undisputed (extent of the disease, primary site), there is still some diversity of opinions concerning others: age, sex, histology of the tumor, its cellular proliferation and DNA content.[27, 28]

In an earlier report, we analysed patients with NHL by means of a multivariate model and have found sex, age, stage and some treatment methods to be of significance for the

Correspondence to: Berta Jereb, Ph. D., M.D., Institute of Oncology, Ljubljana, Zaloška 2, Slovenia. Tel.: (+ 386 61) 323-063 (ext. 37-17). Fax: (+ 386 61) 1314 180.

UDC: 61.6-006.441-036

outcome. We could also show the survival to have significantly improved during the long period of time under investigation. In that analysis, however, neither the primary site nor the histological type of the tumor were included among the variables.[29] In this paper we report on the results of our analysis of a more recent group of patients, with these two variables included in the multivariate model. We aimed to find out whether Kiel classification serves us better in daily work with these patients. Therefore, no reclassification was attempted. We also tried to establish the effect of treatment methods on the survival in different groups of patients. The prognostic impact of Rappaport's[30] as well as Kiel classification[31] has been evaluated separately.

Material and methods

Between 1978 and 1986, 482 adult patients had their first treatment for NHL at the Institute of Oncology in Ljubljana. Patients younger than 15 years, those with chronic lymphocytic leukemia (CLL) and those admitted for recurrent disease were not included in the study. The extent of disease was assessed by clinical examination, biochemistry and blood status, chest X-ray, bone marrow aspiration of the iliac crest, and radionuclide scanning of the liver and spleen. After 1980, the investigations also included an abdominal sonogram, or CT and bone marrow biopsy. For histological classification the Rappaport system was used before 1980, and an updated Kiel classification after 1980. The treatment approaches used during these two periods were different: in the earlier period, one drug or a COP combination (Cyclophosphamide, Vincristine, Prednison) was, as a rule, complemented by radiation to the bulky lesions. After 1980, CHOP (including also Adriamycin) combination was most often used for high grade NHL. Patients with low grade histology and stages I and II were treated by radiation, whereas those with advanced stages were often only observed and treated only if symptomatic. Radiation therapy was applied to bulky lesions, and surgery for gastrointestinal tumors was more favoured after 1980 than before. Ann Arbor system was used for clinical staging throughout.[32]

Our aims were:

1. to find out whether the histological classification system did influence the survival and disease free survival presumably owing to better adjustment of treatment methods to the biological behaviour of disease;

2. to find out whether the independent variables significant for survival were the same in Group K (classified according to Kiel) and Group R (classified according to Rappaport);

3. to find out whether the variables significant for the survival of patients with high grade tumors were the same as for those with low grade tumors.

Statistical analysis

For each patient the following data were recorded for statistical analysis and used as independent variables:

– sex:
245 males, 237 females
– age at the time of diagnosis:
between 16 and 89 years
mean 55.6
median 58.0
– stage:

stage I –	117
stage II –	147
stage III –	76
stage IV –	142

429 were classified as A and 53 had B symptoms.
– primary site:

nodal	250
extranodal	217
unknown	15

– with subgroups:

peripheral nodes	185

mediastinum	26
abdominal	32
head and neck	75
skin	16
bone	10
gastrointestinal tract	88
other extranodal (breast 11, testis 6, spleen 7, soft tissue 6, central spinal 3, and ovary, kidney, uterus, parotis one each)	37
unknown	13
– histology: Rappaport's classification only (R-group):	165
nodular	20
diffuse	95
other	50
Kiel classification (K-group):	317
low grade	163
high grade	124
other	30

There were 174 cases classified according to both systems, and 31 could not be histologically classified.

– methods of treatment:
chemotherapy, radiotherapy and surgery.

The methods of first treatment are given in Table 1.

– duration of chemotherapy:
< 6 months
6 months – 1 year
>1 year

The data were statistically evaluated by survival analysis methods, the time from diagnosis until death being the outcome of interest. The survival curves were calculated according to Kaplan-Meier method.[33]

To reduce the number of potential prognostic variables to a manageable level, we first did univariate analysis, subdividing the data by prognostic variables and comparing the survival curves by log-rank test. The variables which proved to be significantly associated with survival, as well as some other variables considered important by clinicians, were then included in Cox proportional hazards model[34] which was used for the following two purposes:

1) to follow other prognostic variables when trying to confirm the connection between survival and histological classification,

Table 1. Treatment, combination of the 3 methods.

Chemotherapy/ /RT	Number of patients percent of patients none	mono	MOPP	COP	CHOP	other	Total
no RT	38	20	0	36	40	21	155
	27	72		37	26	36	32
< 2000 cGy	7	4	0	17	30	11	69
	5	14		18	19	19	14
≥ 2000 cGy	97	4	4	43	84	26	258
	68	14	100	45	55	45	54
Total	142	28	4	96	154	58	482
Surgery/ChT							
none	113	24	3	80	121	44	385
	80	86	75	83	79	76	80
surgery	29	4	1	16	33	14	97
	20	14	25	17	21	24	20
Total	142	28	4	96	154	58	482

Surgery/ /RT	Number of patients percent of patients none	surgery	Total
	136	19	155
	35	20	32
	50	19	69
	13	20	14
	199	59	258
	52	60	54
	385	97	482

Table 2. Survival according to histological calssification.

	STATUS							
Histological classification	Alive no sympt.	Alive with disease	Died of NHL	Died of other causes	Died of treatment	Died of unknown causes	Lost to follow-	Total
Kiel	127	23	112	13	5	28	9	317 66 %
No Kiel	48	2	75	12	3	21	4	165 34 %
Total	175 36 %	25 5 %	187 39 %	25 5 %	8 2 %	49 10 %	13 3 %	482 100 %

2) to identify important prognostic variables in some subgroups. We did this in order to see if the importance of prognostic variables varied between the subgroups.

Results

By the end of the study in December 1990, 200 patients were alive, 269 dead and 13 were lost to follow up (Table 2).

The patients who had their tumors classified according to Kiel system (treated more recently) had significantly better chances for survival than those who had their tumors classified according to Rappaport system (Figure 1). On the whole, patients with extranodal primary sites did not fare better than those with primary nodal disease (Figure 2). However, patients with extranodal primary tumors of the bone, skin, head and neck, and gastrointestinal organs did significantly better than those with some rare "other extranodal" tumors and those with the primary site in the lymph nodes. The patients with primary abdominal lymph node involvement had the worst prognosis while the prognosis of those with primary bone NHL was the best (Figure 3). Neither age nor sex appeared to be prognostically significant factors.

The results of the multivariate analysis are presented in Tables 3- 6; the hazard ratio is the ratio between the hazard of patients in a specific group and the hazard of patients in a reference group; it is of statistical significance according to the log-rang test. The multivariate analysis confirmed the result of the univariate analysis, showing a 1.5 higher hazard ratio (risk value) for Group R than for Group K.

The same prognostic variables were than used in the multivariate analysis for Groups K and R separately. Table 3 shows the values for Group K. The stage of disease, which emerged as a highly significant factor, was followed by the mediastinum as the primary nodal site, and

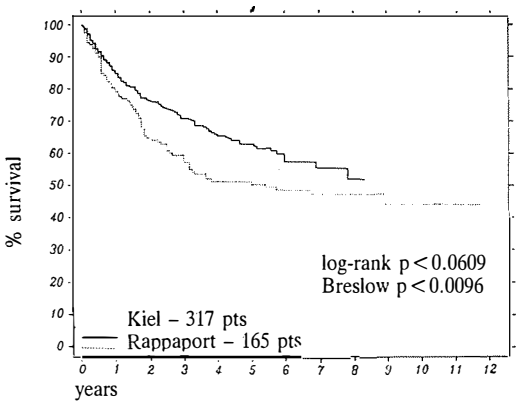


Figure 1. Survival by histologic classification system.

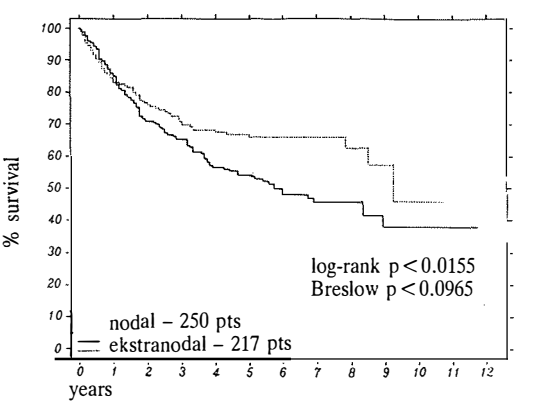


Figure 2. Survival by primary site.

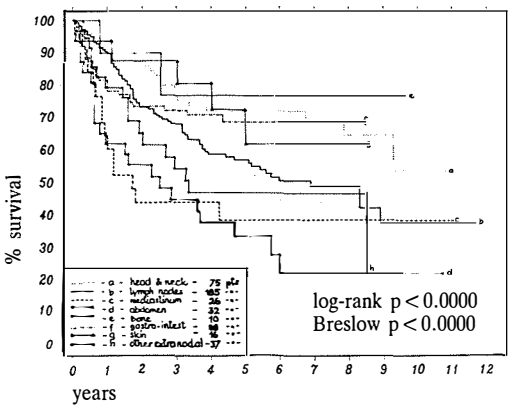


Figure 3. Survival by primary site.

by sex and histological type, respectively. The influence of chemotherapy did not emerge as significant.

The results for Group R are similar, however, radiotherapy appeared among the significant factors while histology did not (Table 4).

As to the survival of Group K patients with low-grade NHL, stage was the only variable which emerged as significant while for Group K patients with high grade NHL the results are quite different, the main difference being the impact of treatment on prognosis, which was significant in patients with high grade tumors but not in those with low grade tumors (Table 5, 6).

Table 3. Results of multivariate analysis for K-group patients.

Predictors	b	hazard ratio	p
low grade	+	1.00	
high grade	0.1985	1.65	0.0472
other	0.6889	1.99	0.0228
sex			
female	+	1.00	
male	0.4450	1.56	0.0238
stage			
A	+	1.00	
B	0.5641	1.76	0.0402
chemotherapy			
yes	+	1.00	
no	0.4785	1.61	0.0977
stage			
I	+	1.00	
II	1.1497	3.16	0.0028
III	1.7490	5.75	0.0000
IV	1.7630	5.83	0.0000
primary site			
hand & neck	+	1.00	
peripheral lymph node	0.2486	1.28	
mediastinum	1.0601	2.89	0.0182
abdomen	0.3884	1.47	
bone	-0.3253	0.72	
gastro	0.3044	1.36	
skin	-0.1448	0.87	
other	0.4467	1.56	

+ = reference group

Table 4. Results of multivariate analysis for R-group patients.

Predictors	b	hazard ration	p
stage			
A	+	1.00	
B	0.9767	2.66	0.0109
radiation			
yes	+	1.00	
no	0.6798	1.97	0.0137
stage			
I	+	1.00	
II	-0.0128	0.96	
III	1.1267	3.09	0.0135
IV	0.3602	1.43	
primary site			
head & neck	+	1.00	
peripheral lymph nodes	0.1124	1.12	
mediastinum	0.8817	2.41	0.1482
abdomen	1.7929	6.01	0.0002
bone	-0.6023	0.55	
gastro	0.4293	1.53	
skin	0.5259	1.69	
other	1.2896	3.63	0.0151

+ = reference group

Discussion

This study confirms our previous report on better results in patients treated more recently. This time, however, the primary site of the tumor and its histological type according to Kiel and Rappaport classifications have been included among the investigated parameters.

Table 5. Results of multivariate analysis for patients with low-grade tumors.

Predictors	b	hazard ratio	p
stage			
I	+	1.00	
II	1.2940	3.65	0.0252
III	1.7150	5.56	0.0252
IV	1.8709	6.49	0.0005

+ = reference group

Table 6. Results of multivariate analysis for patients with high-grade tumors.

Predictors	b		hazard ratio	p
sex				
female	+		1.00	
male		0.8107	2.25	0.0142
chemotherapy				
yes	+		1.00	
no		2.0786	8.00	0.0013
radiation				
no	+		1.00	
yes		0.7647	2.15	0.0457
surgery				
yes	+		1.00	
no		1.2860	3.62	
stage				
I	+		1.00	
II		2.0496	7.77	0.0039
III		2.4621	11.73	0.0008
IV		2.2096	24.77	0.0000
primary site				
head & neck	+		1.00	
peripheral lymph nodes		0.5562	1.74	
mediastinum		1.7580	5.80	0.0127
abdomen		0.0392	1.04	
bone		-0.5180	0.60	
gastro		0.9686	2.63	
skin		-0.0268	0.97	
other		0.4575	1.58	

+ = reference group

The finding that Kiel classification system had favourable impact on the prognosis (the patients of Group K had a 1.5 better chance for survival than those in Group R) suggest that the use of Kiel system enabled better identification of low-risk and high-risk patients, resulting in better adjustment of treatment to the patient and his condition than in Group R (Table 3, 4). However, regarding other factors, the results in this series were not in full agreement with those in the previous one. We could not confirm age or sex as significant for prognosis in the univariate analysis of the whole series of 482 patients nor did the group of patients with all extranodal primary sites have better survival than those with nodal ones. It is possible that the rather high proportion of patients who died of other causes made the evaluation of survival curves in the univariate analysis less accurate, thus rendering the differences less significant. In the univariate analysis, the following factors were found to be prognostically significant: stage of the disease, primary site and the system of histological classification. They were also confirmed by multivariate analysis of the whole group. On separate analysis of Groups K and R, stage and primary site remained significant in both, stressing their independent influence on the prognosis. There have been reports on a correlation of histological subgroups according to Rappaport system, but in our Group R histology did not emerge as a significant predictor of the outcome. In Group K, however, it did, thus confirming the reviews. The finding that males had a worse outcome in Group K but not in Group R is hard to explain.

Chemotherapy as a predictor had only limited influence in Group K, while radiation in Group R was a stronger predictor of the outcome. Radiation was also used more often in the earlier group of patients and was to a lesser extent directed by different histological grading. The results of the separate analysis of the low-risk and the high-grade Group K patients are striking. Only stage I patients in the early stages, treated by radiation were curable, while the treatment had little or no effect on the course of the disease in more advanced stages.

In the high-risk group, stage was a highly significant predictor, however, chemotherapy as well as radiation had a significant impact on the outcome of the disease. It is evident that for the high-risk patients in Group K an effective treatment has been introduced while for the patients with advanced low risk tumors the question how to treat and whether to treat at all remains open.

We are well aware of the fact that treatment has changed over years, and full justification of our results could only be obtained by reclassification of the whole material. This being unfeasible, we nevertheless believe that the change of classification method had an influence on survival. We tried to check the possible effect of "treatment change" by including the year of diagnosis as a confounding variable. The "histological classification" variable remained significant in the multivariate regression model. We should also stress that all the patients diagnosed prior to the year 1978 were

excluded, so that only patients diagnosed between the years 1978 and 1986 were used in the analysis.

Conclusions

There are some conclusions that can be drawn from the analysis of the presented group of patients:

1. The group of patients that was classified according Kiel system and had their treatment adjusted to the histological grade had a 1.5 better chances for cure than the patients with tumors classified according Rappaport system.

2. Nodal abdominal primary site was associated with poor prognosis, while patients with gastrointestinal primary tumors did rather well.

3. Stage is a significant factor for predicting the outcome.

4. Chemotherapy and radiation are significantly improving the survival in the high-risk (K) group but do not seem to have any bearing on the outcome in the low risk (K) group of patients.

Acknowledgement

We are deeply indebted to Drs M. Vovk and M. Jenko for cooperation in the analysis of patients the great majority of whom have been under their care. The work has been supported by a grant from the Ministry for Science and Technology of the Republic Slovenia.

References

1. Bloomfield CD, Goldman A, Dick F, Brunning RD, Kennedy BJ. Multivariate analysis of prognostic factors in the non-Hodgkin's malignant lymphomas. *Cancer* 1974; **33:** 870-9.

2. Leonard RCF, Cuzick J, MacLennan ICM et al. Prognostic factors in non-Hodgkin's lymphoma: the importance of symptomatic stage as an adjunct to the Kiel histopathological classification. *Br J Cancer* 1983; **47:** 91-102.

3. D'Amore F, Christensen BE, Brincker H et al. Clinicopathological features and prognostic factors in extranodal non-Hodgkin lymphomas. *Eur J Cancer Clin Oncol* 1991; **27:** 1201-8.

4. Vose JM, Armitage JO, Weisenburger DD et al. The importance of age in survival of patients treated with chemotherapy for aggressive non-Hodgkin's lymphoma. *J Clin Oncol* 1988; **6:** 1838-44.

5. Nabholtz J-M, Friedman S, Bastien H, Cuisenier J, Horiot J-C, Guerrin J. A clinico-pathological and prognostic analysis of non- Hodgkin lymphoma: a study of 203 patients. *Acta Oncol* 1988; **27:** 489-95.

6. De Wolf-Peeters C, Caillou B, Diebold J et al. Reproductibility and prognostic value of different non-Hodgkin's lymphoma classifications: study based on the clinicopathologic relations found in the EORTC trial (20751). *Eur J Cancer Clin Oncol* 1985; **21:** 579-84.

7. Heinz R, Fortelny A, Schneider B et al. Long term follow-up of 1520 NHL patients classified according to the Kiel classification - experiences of a single institution (meeting abstract). Fourth international conference on malignant lymphoma, Lugano, 1990:59.

8. Burgers VMJ, Somers R, Quasim MM, Glabbekke van M. Report on the EORTC lymphoma trial 20751. *Int J Radiat Oncol Biol Phys* 1983; **9:** 11-5.

9. Fyles A, Brada M, Ashley S, Horwich A. Localized low grade non-Hodgkin's lymphoma (NHL) (meeting abstract). 7th Annual meeting of the European society for therapeutic radiology and oncology. Den Haag, 1988.

10. Joensuu H, Klemi PJ, Soderstrom K-O, Jalkanen S. Comparison of S-phase fraction, working formulation, and Kiel classification in non-Hodgkin's lymphoma. *Cancer* 1991; **68:** 1564-71.

11. Federico M, Gobbi PG, Barbieri F, Silingardi V. Relationship between prognostic factors and therapy in high-grade non-Hodgkin's lymphomas over two decades. *Haematologica* 1989; **74:** 511-9.

12. McMaster M, Greer J, Greco A et al. Analysis of prognostic factors in patients (pts) treated with high dose , brief duration therapy for poor prognosis non-Hodgkin's lymphoma (NHL) (meeting abstract). *Proc Annu Meet Am Soc Clin Oncol 1989*; **8:** A1064.

13. Armitage JO, Cheson BD. Interpretation of clinical trials in diffuse large-cell lymphoma. *J Clin Oncol* 1988; **6:** 1335-47.

14. Epelbaum R, Faraggi D, Ben-Arie Y et al. Survival of diffuse large cell lymphoma: a multivariate analysis including dose intensity variables. *Cancer* 1990; **66:** 1124-9.

15. Stuart NS, Blackledge GRP, Child JA et al. A new approach to the treatment of advanced high-grade non-Hodgkin's lymphoma - intensive two-phase chemotherapy. *Cancer Chemother Pharmacol* 1988; **22:** 141-6.

16. Shimayama M, Ota K, Kikuchi M et al. Chemotherapeutic results and prognostic factors of patients with advanced non-Hodgkin's lymphoma treated with VEPA or VEPA-M. *J Clin Oncol* 1988; **5:** 128- 41.

17. Inwards DJ, Armitage JO. Modern chemotherapeutic regimes in the management of aggressive non-Hodgkin lymphoma: can they be improved. *Eur J Cancer* 1991; **27:** 510-3.

18. Liang R, Todd D, Chan TK. HOAP-Bleo as salvage therapy for diffuse aggressive non-Hodgkin's lymphoma. *Cancer Chemother Pharmacol* 1988; **22:** 169-71.

19. Aviles A, Diaz-Maqueo JC, Sanchez E, Cortes HD, Ayala JR. Long-term results in patients with low-grade nodular non- Hodgkin's lymphoma. *Acta Oncol* 1991; **30:** 329-33.

20. Sutcliffe SB, Gospodarowicz MK, Bush RS et al. Role of radiation therapy in localized non-Hodgkin's lymphoma. *Radiother Oncol* 1985; **4:** 211-23.

21. Heinz R. Long-term follow-up of CHOP-treated non-Hodgkin lymphoma of high-grade malignancy. *Blut* 1990; **60:** 68-75.

22. Hagberg H, Pettersson U, Glimelius B, Sundstrom C. Prognostic factors in non-Hodgkin lymphoma stage I treated with radiotherapy. *Acta Oncol* 1989; **28:** 45-50.

23. Brittinger G, Bartels H, Fulle HH et al. Grundlagen und bisherige Ergebnisse der perspektiven Studie der Kieler Lymphom gruppe uber Non-Hodgkin-Lymphome. In: Stacher A, Hocker P eds. Lymphknotentumoren. Munchen: Urban und Schwarzenberg, 1979: 1931- 200.

24. Scarantino CW, Greven KM, Buss DH. Single high dose-large field irradiation in drug resistant non-Hodgkin's lymphoma. *Int J Radiat Oncol Biol Phys* 1988; **14:** 1001-5.

25. Mackintosh JF, Cowan RA, Jones M, Harris M, Deakin DP, Crowther D. Prognostic factors in stage I and II high and intermediate grade non-Hodgkin's lymphoma. *Eur J Cancer Clin Oncol* 1988; **24:** 1617-22.

26. Cowan RA, Jones M, Harris M et al. Prognostic factors in high and intermediate grade non-Hodgkin's lymphoma. *Br J Cancer* 1989; **59:** 276-82.

27. Cowan RA, Harris M, Jones M, Crowther D. DNA content in high and intermediate grade non-Hodgkin's lymphoma - prognostic significance and clinicopathological correlations. *Br J Cancer* 1989; **60:** 904-10.

Radiol Oncol 1994; 28: 34-9.

Epidemiological features of cervical carcinoma in young women of Slovenia

Vera Pompe Kirn and Neva Volk

Institute of Oncology, Ljubljana, Slovenia

According to the data of the Cancer Registry of Slovenia in the period 1987–1989, cervical cancer represented 27% of all cancers in young women of Slovenia. The age specific incidence was 11/100 000 women, and the cumulative rate (0–34) 0.14/100. With these rates Slovenia was placed in the middle of the rank order of 21 selected European regions and countries. The rates for women aged 25–34 had been increasing till the year 1965, and were rather stable afterwards. An obvious increase has not yet been registered. Before the age of 25 invasive cervical carcinoma has been a very rare phenomenon in Slovenia since 1950 (one or two cases per year). A more detailed analysis of the period 1975–1989 revealed rather stable invasive cancer rates. In the 80's intraepithelial cancer rates have been decreasing, a tendency towards decline in the percentage of the localized stage as well as of the FIGO IA stage was noted. In the case that the described tendency continues, an increase in the invasive carcinoma rates in young women of Slovenia can be expected. The described situation calls for further analysis and action, considering Recomendations of the committee of cancer experts on cervix uteri cancer screening, 6 April 1992.

Key words: cervix neoplasms-epidemiology, adult; Slovenia; cervix uteri cancer, young women, incidence in Slovenia, stages.

Introduction

The Cancer Registry of Slovenia was founded at the Institute of Oncology in Ljubljana in the year 1950 on the initiative of Profesor Ravnihar. It is a special service for collecting, processing and analysing cancer incidence and cancer patient survival data in Slovenia.

Cervical carcinoma has been one of the most frequently analysed primary cancer sites.[1–4]

Correspondence to: prof. Vera Pompe Kirn, MD, PhD, Institute of Oncology, Zaloška 2, 61105 Ljubljana, Slovenia.

UDC: 618.146-036.2

For the purpose of the 8th ESGO congress in Ljubljana an analysis of the incidence of this carcinoma in young women was prepared.

Methods and data

Standard descriptive epidemiological methods were used, crude incidence rate being defined as the rate of all cancer cases per 100 000 population, and cumulative risk as the risk which an individual would have of developing a cancer in question during a certain age-span if no other causes of death were in operation.

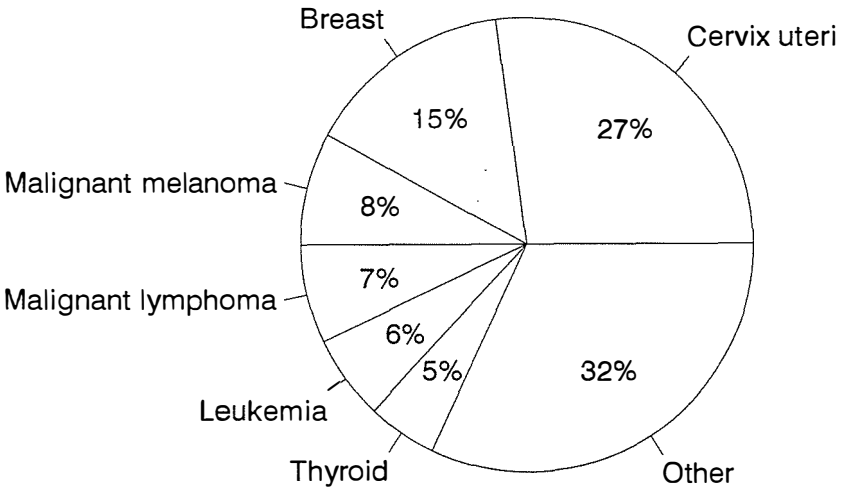


Figure 1. Six leading cancer sites in women aged 20-34 years, Slovenia 1987-89.

The cumulative rate is an approximation to the cumulative risk. It is the sum over each year of age of the age specific incidence rates taken from birth to the age of 34 for 0–34 rate, and to the age of 74 for 0–74 rate.[5]

Data for Slovenia were taken from the computerised data base of the Cancer Registry, and data for other European countries were taken from the last Volume of the publication Cancer Incidence in Five Continents, where only data of high quality cancer registries are published.[6]

Results

In young women, defined by the age 20–34 years in the period 1987–1989, cervical carcinoma was still the most frequent primary cancer site in Slovenia. It represented 27% of all cancers in young women (Figure 1). The age specific incidence rate was 11/100 000 women, and the cumulative rate 0–34 was 0.14/100. These rates placed Slovenia in the middle of the rank order of selected European regions and countries (Figure 2). In fact, its cumulative rate represented the median value.

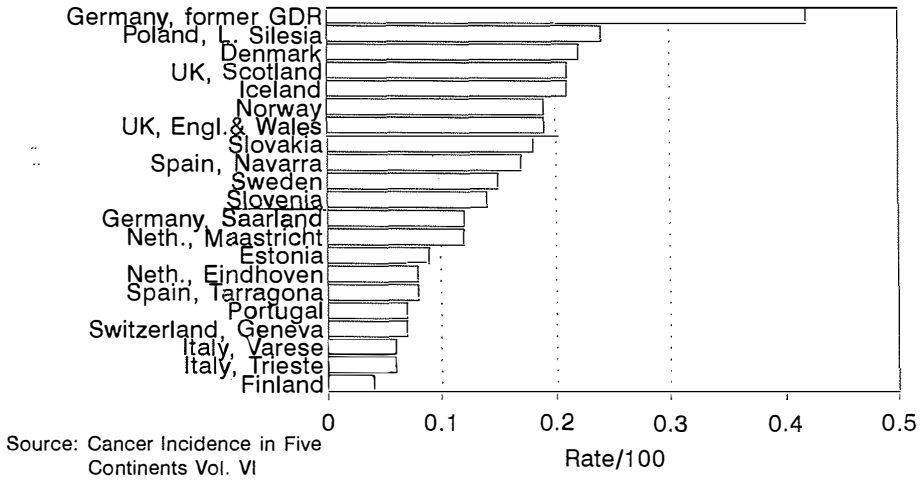


Figure 2. Cumulative cervical cancer incidence rates in young women 0-34, Europe 1983-1987.

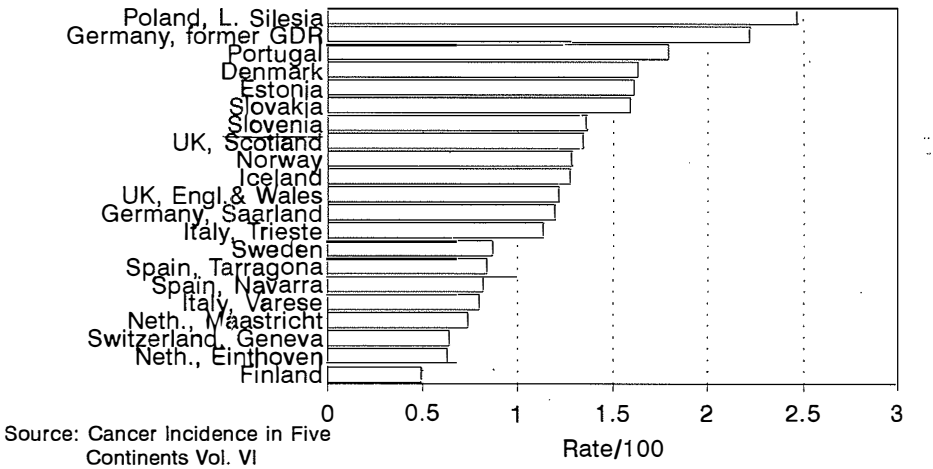


Figure 3. Cumulative cervical cancer incidence rates 0–74, Europe 1983-1987.

The problem of young women does not always reflect the magnitude of the burden of this disease in the studied population. In the rank order of cumulative rates 0–74, Slovenia was in the upper half (Figure 3). The risk of a woman in Slovenia to get cervical cancer till her 75th birthday in the studied period was still 1.4/100.

In Figures 4 and 5 time trends of crude cervical cancer incidence rates for all women and age specific rates for the younger ones are plotted. Invasive and intraepithelial cervical carcinoma rates are given together because at present the incidence in Slovenia, as elsewhere in the world, reflects both exposure to risk factors and level of screening activity. In whole Slovenia an opportunistic screening activity has been going on since the year 1960.[1–4] It was started earlier, i. e. in 1953 in three regions only. These screening activities are mainly reflected in the intraepithelial cervical carcinoma rates. The crude rates of invasive carcinoma had been decreasing till the year 1979, whereas the age specific rates for younger women aged 25–34 had been decreasing till the year 1965 only and were relatively stable afterwards. Be-

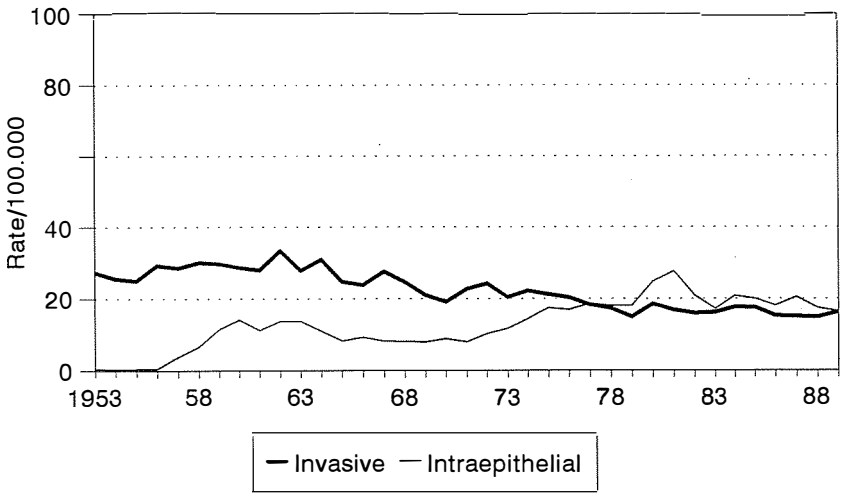


Figure 4. Incidence of invasive and intraepithelial cervical carcinoma, Slovenia 1953-1989.

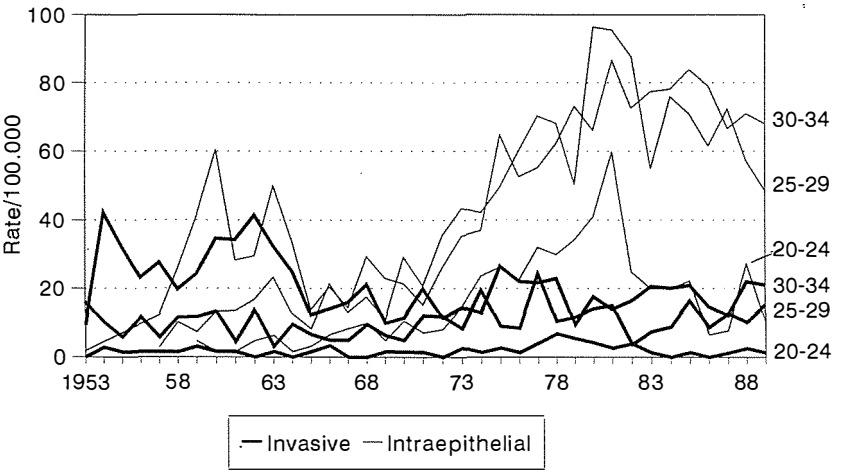


Figure 5. Incidence of invasive and intraepithelial cervical carcinoma in young women, Slovenia 1953-1989.

fore the age of 25 invasive cervical carcinoma has been a very rare phenomenon in Slovenia since 1950; 1 or 2 cases per year have been registered, only.

The incidence in young women in last 15 years was analysed in more detail. Besides rather stable invasive carcinoma rates, the intraepithelial carcinoma rates were greatly varying, with a peak in 1980–1982 for all three age groups 20–24, 25–29, and 30–34yrs. Later a decreasing tendency was noted in all three age groups (Figure 5). The invasive carcinoma rates were rather stable. At some time points only, an increasing tendency was observed.

The stage distribution of all invasive carcinoma cases in the 15 year period with rather stable rates was much more favourable for younger women then for the elderly (Figure 6). Unfortunately, in time trends of the stage distribution an unfavourable tendency was noticed in young women in Slovenia (Figure 7). The percentages of the so called localised stage were slightly decreasing with time, the decrease was not statistically significant as the confidence

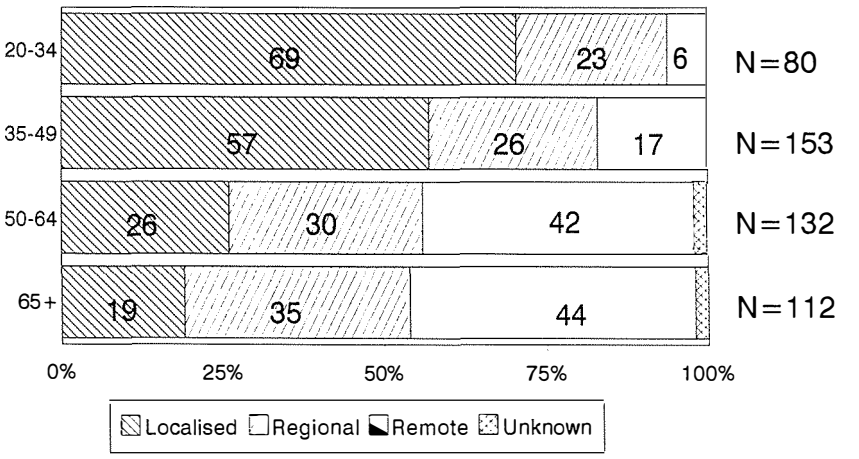


Figure 6. Stage distribution of cervical carcinoma by age, Slovenia 1987-89.

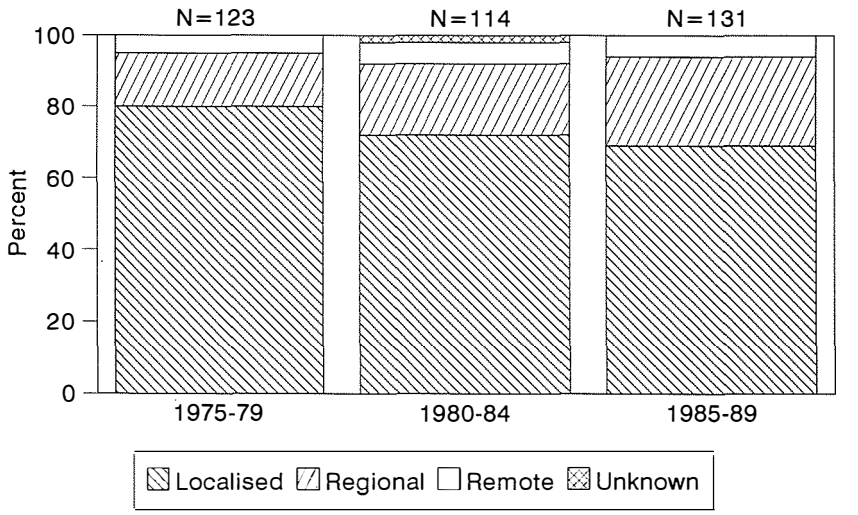


Figure 7. Stage distribution of cervical carcinoma in young women, Slovenia 1975-1989.

intervals were overlapping. In the analysis of FIGO Stage I distribution into A and B stages (Figure 8) a tendency to a less favourable stage distribution was also noticed in the eighties.

Conclusion

In 1987–1989 in young women of Slovenia cervical carcinoma was still the most common cancer site with an age-specific rate of 11/100 000 women. The incidence rates have been rather stable over a long time period despite the opportunistic screening going on in the whole state since the year 1960. Considering different time periods, an obvious increase in the incidence of invasive form could not be confirmed either.

The results of a detailed analysis of the last 15 year period: in the 80's a decrease in the intraepithelial carcinoma rates, a tendency to-

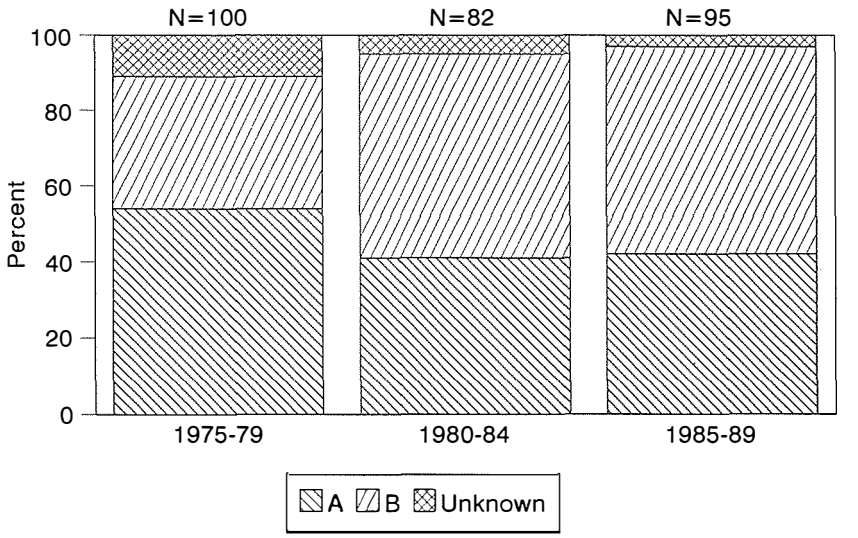


Figure 8. Distribution of A and B FIGO I. stage of cervical carcinoma in young women, Slovenia 1975-1989.

wards a decline in the percentage of the localised stage as well as of the Figo IA stage are a reason for concern, however.

If the described tendency continues, an increase in the invasive carcinoma incidence in young women of Slovenia could be expected as well.

Considering the last European guidelines for Quality Assesment in Cervical Screening[7] the following questions are posed:

Is it the opportunistic screening in Slovenia, as it is, the right way?

What is the quality of taking and reading cervical smears in Slovenia?

Were the registered Stage IB cases the so-called fast growing carcinomas or they occurred to women not assessed by the opportunistic screening practised in Slovenia? In the case they were reached, what was the quality of taking and reading smears?

The situation calls for further analysis and action.

References

1. Kovačič J. *An epidemiologic evaluation of cervical cancer screening* (in slovene). Ljubljana: University of Ljubljana, 1972 (doctor's thesis).
2. Pompe Kirn V, Ravnihar B. Time trends and geographical distribution of uterine cervix cancer and breast cancer incidence in Slovenia (in slovene). *Zdrav. vestn. 1980;* **49:** 149–52.
3. Pompe Kirn V, Vrščaj Uršič M, Kovačič J, Primic Žakelj M. Epidemiological evaluation of early detection of cervical cancer in Slovenia till 1981. *Eur J Gynec Oncol* 1986; **7:** 147–51.
4. Pompe Kirn V, Kovačič J, Primic Žakelj M. Epidemiological evaluation of cervical cancer screening in Slovenia up to 1986. *Eur J Gynaec Oncol* 1992; **13:** 75–82.
5. Cancer registration: principles and methods. *IARC Sci Publ* 1991; 95.
6. Cancer incidence in five continents. Vol 6. *IARC Sci Publ* 1992; 120.
7. Anon. The effectiveness of organised screening programmes. *Eur J Cancer* 1993; **29A** (Suppl 4): S1–S3.

Radiol Oncol 1994; 28: 40-8.

Risk factors connected with the appearance of chronical diseases and cancer in the Republic of Slovenia

Dražigost Pokorn

University of Ljubljana, Medical Faculty, Institute of Hygiene, Slovenia

The article shows the most frequent risk factors in the Republic of Slovenia that the author could gather on the basis of available sources in Slovenia. We could conclude that relatively high incidence and prevalence of chronical and degenerative diseases (cardiovascular diseases and cancer) in the Republic of Slovenia or their permanent increase, if compared with western countries, where it is lower and decreasing already for several years, is a consequence of a much too intensive presence of risk factors in the Republic of Slovenia. Only after a change in the policy of nutrition, environmental protection, medical education and a changed medical welfare service in general, it will be possible to decrease the incidency of these diseases in the newly established state of Slovenia.

Key words: neoplasms-epidemiology; chronic disease-epidemiology; risk factors; Slovenia

Introduction

With their endeavours to prolong the life expectancy (Table 1) during the last twenty years the Slovenians stayed considerably behind their neighbouring countries. In Slovenia, the main reasons for premature mortality are the same as in other European countries; undoubtedly Slovenia is one of the countries where its inhabitants loose their lives because of injuries and suicides.[1] Slovene patients with cardiovascular diseases and cancer, representing 69.1 % of all causes of death, are dying earlier as similar patients in the neighbouring countries. Since 1970, mortality from ischaemic heart diseases has been growing in Slovenia similarly as in other countries of Central and Eastern Europe, while the trend in the developed countries of Northern Europe[2] is opposite (Figure 1). During the last few years a slight decrease in premature mortality from heart- and coronary diseases can be observed (Figure 2).[1,3] Cancer incidence (1950–1987) is also growing (Figure 3).[4]

The Slovenians contract these diseases because of worse primary prevention, consequen-

Corespondence to: Prof. Dražigost Pokorn, PhD, MD, Institute of Hygiene, Medical Faculty, Zaloška 4, 61000 Ljubljana, Slovenia.

UDC: 616-006.6-036.2-02

Table 1. Lifge expectancy in 5 European countries, 1970, 1980, 1988.[1]

Country	1970		1980		1988	
	Men	Women	Men	Women	Men	Women
Austria	66.3	73.4	69.0	76.1	72.1	78.7
Germany	67.2	73.6	69.6	76.8	72.3	79.1
Italy	68.6	74.7	71.0	77.6	72.7	79.2
Sweden	72.3	77.4	72.8	79.0	74.2	80.4
Slovenia	65.0	72.3	67.4	75.2	67.6	76.8

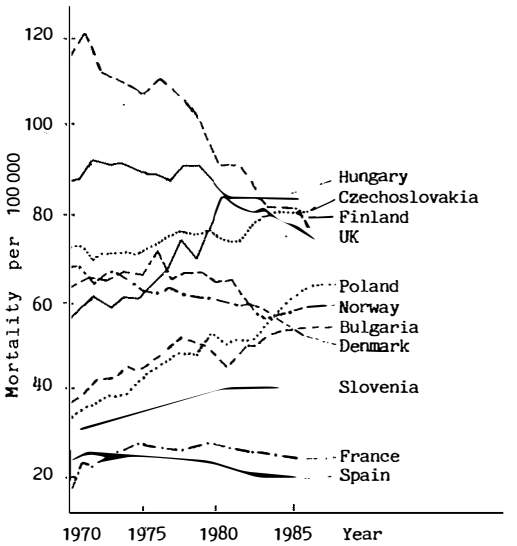


Figure 1. Premature mortality from ishaemic heart diseases. Standard mortality rate for men and women, 0–64 years of age, per 100000.[1, 2]

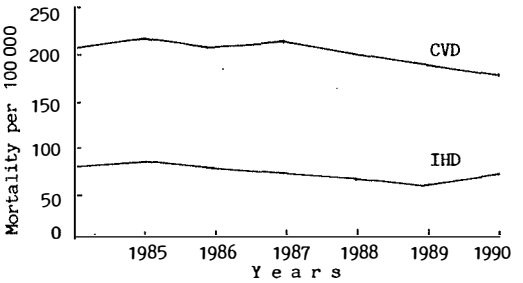


Figure 2. Premature mortality from cardiovascular (CVD) and ishaemic heart diseases (IHD) in Slovenia. Standard mortality rate for men and women, 35–64 years of age, per 100000.[1, 3]

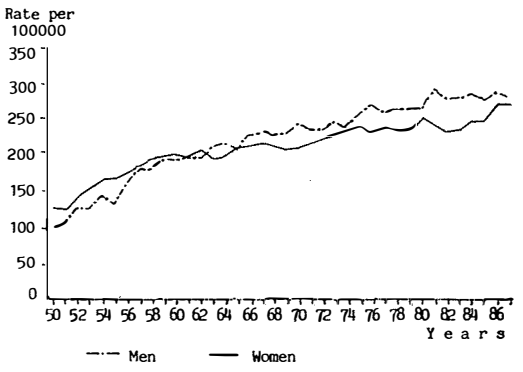


Figure 3. Cancer incidence in Slovenia, 1950–1987.[4]

tly larger prevalence of risk factors, or these illnesses are discovered later and treated less successfully.

The main purpose of this article is to try to show the most frequent risk factors in the Republic of Slovenia on the basis of available sources in Slovenia.

The quoted data can serve only as an orientation for a survey of risk factors associated with the appearance of chronical diseases in a country that has only started with the prevention of cardiovascular diseases and cancer.

Material and method

The data on dead inhabitants acording to the causes of their death, sex and hospital admissions due to the diseases of resident population by international classification of diseases (ICD) are based on the Medical Statistical Annual of Slovenia, 1991, published by the Institute of Health of the Republic of Slovenia,[1] collecting health statistics. From the same source we also took the data on diseases detected in specialized out-patient departments and on gastrointestinal diseases for 1969–1991 and 1977–1989. Data on age-standardized mortality for men and women (for the age 0–64 years per 100.000 inhabitants) were specialy prepared for this survey by the Institute for Health (Personal Report 1992). We incorporated them into the figure showing mortality from ischaemic heart diseases in Central, Eastern and North Europae (E. Helsing).[2] The data on annual cancer incidence, crude incidence rate per 100.000 inhabitants in the Republic of Slovenia, and on annual incidence rates of stomach cancer were obtained from the Central Cancer Register of Slovenia at the Institute of Oncology in Ljubljana.[4] Data on the production, transport and sales of food and on the annual consumption of food and beverages per household member for 1965–1988 and 1970–1990 were derived from the Statistical Annual of the Republic of Slovenia, which is published regularly by the Statistical Office of the Republic of Slovenia.[5]

The energetic value of an average daily meal and the nutrient ratio: protein, fats, carbohy-

drates and dietary fibers in a meal were calculated on the basis of the data for annual consumption of food and beverages per household member in the Republic of Slovenia according to the inquiry on the consumption in households, performed by the Statistical Office of the Republic of Slovenia for 1965–1988 and 1970–1990 every five years,[5] and by the help of plates with nutritional values of the food, Zagreb 1990.[6] In 1988 the pattern of a five-year inquiry included 3.250 households: 56 rural, 811 mixed and 2.383 nonrural households, chosen according to the method of random selection.

The Statistical Office of the Republic of Slovenia was also the source of data on the production of cigarettes. Data on the emission of sulphur dioxide into the air by consumers of fuel and raw materials in the Republic of Slovenia are – on the basis of analyses made by the Institute for Hydrometeorology of the Republic of Slovenia[5] – also gathered by the Statistical Office of Slovenia.

The analyses of samples of drinking water as to their bacteriological and chemical irreproachabilty are regularly performed by regional institutes of hygiene and social medicine in the Republic of Slovenia. Data on irreproachability of drinkable water were obtained from the Report of these microbiological laboratories.[1] The analysis of the magnesium content of drinkable water in Slovenia in 79 at random chosen water sources (of open and closed type) was in 1981 and 1982 performed by the Center for Mineral Water Research in Maribor, Republic of Slovenia.[7]

The data on individual risk factors (smoking, obesity, hypertension, hypercholesterolemia) were obtained from the latest epidemiologic reasearches in Slovenia: Berger et al,[8] Accetto and Javornik;[9] Pokorn;[10] Srebot et al;[11] Fortič[12] and Strgar;[13] Gradišek et al;[14] Jezeršek et al;[15] Radisavljević et al.[16]

The daily nutritional pattern and the content of salt and dietary fibrins in the daily food pattern of the older population was taken from the study of Pokorn et al.[17]

Results with discussion

It is interesting that although chronical diseases affect different organic systems and differ completely also as to their etiopathogenesis, the risk factors for some of them are very similar. For example: the development of arteriosclerosis is advanced by numerous factors, the effects of which should not only be added- up, because they intensify each other.[18] Therefore it is extremly difficult to explain the influence of food on heart and coronary diseases, if important risk factors for the appearance of arteriosclerosis such as physical activity and cigarette smoking of the population are not known.

As important risk factors for the appearance of heart and coronary diseases and cancer among the population of Slovenia, we took into account some nutritional factors, alcohol abuse, and polluted environment, which are systematically collected by the state institutions, and also some other available risk factors – smoking, elevated blood pressure, and plasma concentration of cholesterol.

Among the risk factors for the appearance of chronical diseases it is food that may be one of the most important risk factors.[19, 20] With the average consumption of food and beverages per household member in the Republic of Slovenia (1965–1988) a rise in the consumption of individual groups of food – with the exception of fats – can be observed. The variability of

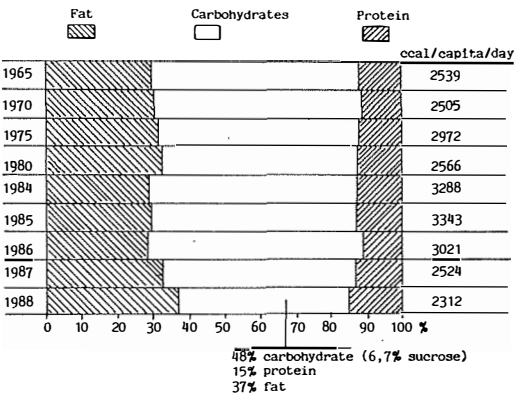


Figure 4. The proportion of fat, carbohydrates and protein in the total energy supply in Slovenia, 1965–1988.[5]

consumption of individual food types is relatively large, which could be attributed to different and insufficiently accurate methods of households inquiring.

After 1965 an obvious fall in the food consumption can be observed, which can be seen also from the average energetic and nutritional value of an average daily meal (Figure 4). From 1965 to 1985 the energetic value of an average daily meal was growing from 2539 to 3343 Kcal. Afterwards it started to fall and in 1988 it ranged at 2312 Kcal/day when an average meal of a Slovenian contained more than 35% of fats and approximately 15% of proteins in respect to the energetic value of the consumed food. The ratio between the animal and vegetable fats was decreased (Table 2). The share of

Table 2. The proportion of animal and vegetable fat in the total fat supply in Slovenia, 1979–1989.[5]

Year	Animal fat (%)	Vegetable fat (%)	Olive oil (5)
1979	21	78.9	0.1
1984	23	76.8	0.2
1989	14	85.8	0.2

olive oil, which is supposed to have also an important protective influence on the appearance of arteriosclerosis,[19] is extremly low. A bigger share of vegetable oils in everyday nutrition can be partly proved also by the increasing production of this food in the Republic of Slovenia. The increased production of eggs, meat, milk and fish during the last twenty years (Figure 5) caused a rise of the percentage of proteins in the daily nutrition and probably also of saturated fats.

The quantity of the consumed table sugar per household member was falling extensively from 1975 to 1988, while the quantitative trade with sugar and candy production are both increasing, which means that the consumption of sugar products, chocolate, cakes, etc. is on the increase. Together with a lower consumption of table sugar there is also a fall in caries incidence (Figure 6). The cause for a lower caries rate in the Republic of Slovenia can not be found only in the lower consumption of table sugar, but also in the improved mouth hygiene and better teeth fluoridation.[21]

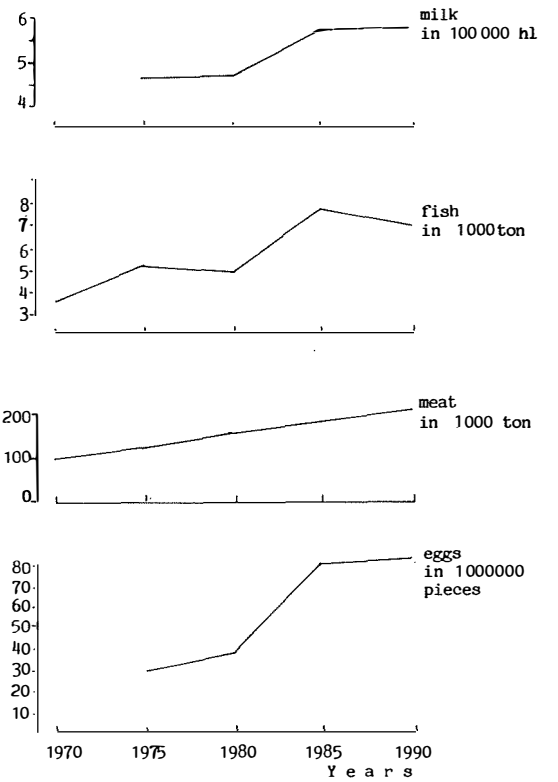


Figure 5. Production of protein food in Slovenia, 1970–1990.[5]

The quantity of the consumed fruit and vegetables has been falling since 1979–1989 (Table 3). Because of the low quantity of daily consumed fruit and vegetables and cereal products, especially wholegrain cereals, the content

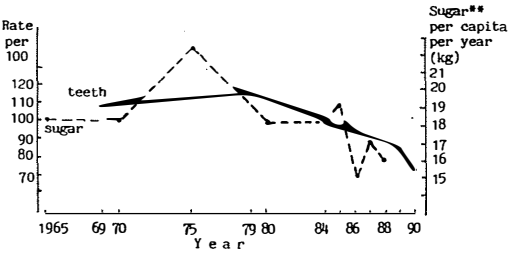


Figure 6. Treatment* in general dental clinics by HC domicile, Slovenia, 1969–1990 and available supply of sugar in Slovenia, 1965–1988.[1, 5]

* Teeth filling with and without treatment, surgical treatment (extracted teeth and other treatment).

** Sugar consumption per persona per year in Slovenia.

Table 3. Per capita consumption of fruit, vegetables and dietary fiber in Slovenia, 1979–1989.[5]

	Unit	1979	1984	1989
Vegetables	kg/day	31.6	16.6	15.7
Fruit	kg/year	56.8	32.7	31.2
Dietary fiber	g/day	16.4	16.0	15.1

of dietary fibers in the daily nutrition has also been decreasing – in the same period it ranged between 16.4 and 15.1 g/day. Together with a low consumption of fruit and vegetable there is also a lower consumption of protective substances and this can be an important risk factor for the appearance of cardio-vascular diseases and cancer.[22-27]

An extremly low value of daily consumed fibers in the daily nutrition of Slovenian population was established also with the analysis of 56 at random chosen patterns of daily meals in the city of Ljubljana. The value of fibrins ranged between 3.5–21.9 g/day (7.7 +/– 3.9 g/day).[17]

A lot of eggs, fats, sugar, meat and milk products and a low quantity of fruit and vegetable (dietary fibrins) can also be a risk factor for the appearance of gallstones,[28, 29] which is also on the increase (Figure 5).

We lack data on salt consumption in households, but we do have data on the quantitative wholesale trade with food (salt). Between 1970 and 1990 the trade with salt increased strongly. An analysis of 56 daily meals showed that the daily salt content ranged between 4.6 and 16.6 g salt/day (8.7 +/– 2.9 g/day), which exceeds the protective food recommendations.[17, 22]

Serum levels of vitamins A and E, which are also important factors in the prevention of cardiovascular diseases and cancer, were analysed in 189 randomly selected and examined persons, aged over 60 years and living in Ljubljana (Figure 7). We did not observe an obvious lack of these three nutrients, which could be partially proved by a menu pattern containing a relatively varied combination of fruit, vegetables, cereals and milk. Approximately 90% of the examined persons had a standardized plasma vitamin E, and over 30 μM/mM of cholesterol. Only with approx. 41% of all examined

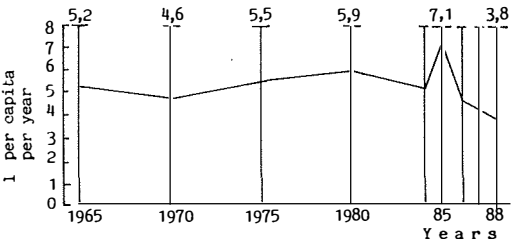


Figure 7. The annual average alcohol (100 vol %) consumption in Slovenia, 1965–1988.[5]

persons the plasma vitamin A exceeded 2.1 M/l, according to Gey et all,[24] this represents the normal preventive concentration of vitamin A and of the standardized vitamin E in the plasma of the examined persons.

Because magnesium content in drinking water can, to a certain degree, also be important for the prevention of cardiovascular diseases,[30] we give a substantial and up till now the only epidemiologic study of magnesium content in drinking water of the Republic of Slovenia. Among 79 randomly chosen samples of drinking water we found 20 samples of water with a low magnesium content.

In the period 1970–1990, an increase was noted also in the field of quantitative trade with alcoholic beverages. In the period from 1965 to 1985, the quantity of consumed pure alcohol, obtained from the consumption of food in households,[5] ranged between 5.2 and 7.1 l per year and person. Afterwards, the consumption of alcohol was steadily decreasing until in 1988 it achieved only 3.8 l per year and person (Figure 7). Lower consumption of alcohol could be

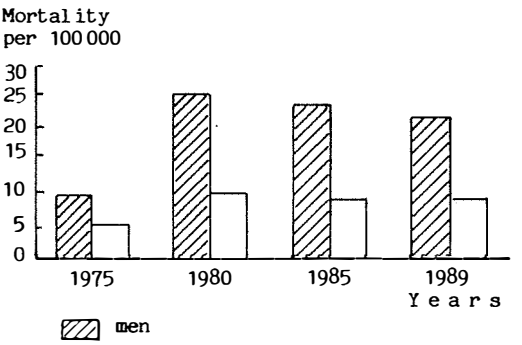


Figure 8. Cirrhosis mortality in Slovenia, 1975–1989.[1]

connected with the lower mortality from liver cirrhosis[31] observed during the last years (Figure 8). This has been proved also by a less frequent appearance and hospitalization of alcoholic liver cirrhosis in the period from 1977 to 1989.

Simultaneously with the increased production of beer we can observe a rise of rectal cancer (Figure 9). Although some epidemiological stu-

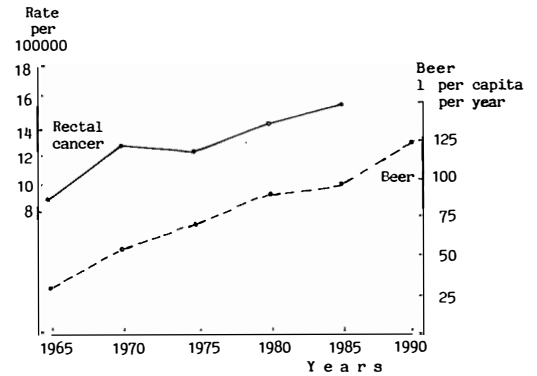


Figure 9. Average annual crude incidence rates of rectal cancer (male) and production of beer in Slovenia, 1965–1990.[4, 5]

dies clearly point out a connection between consumption of beer and rectal cancer, there are others which cautiously deny that.[20, 32-34] Causes for such a high annual incidence of rectal cancer in Slovenia could also be due to inappropriate nutrition, smoking and some other causes.[20, 32-35]

Our survey of risk factors includes some known environmental pollutants, which can also play an important role in the appearance of chronical diseases: polluted air, drinking water, smoking of cigarettes and equipment of individual households with refrigirators, all these are orientational indicators of less spoiled food among the population.

The emission of sulphur dioxide into the air by individual consumers of fuel and raw materials in Slovenia had been steadily increasing until 1987. Afterwards, it obviously began to fall, especially due to stricter legislation measures. This could also be the cause for a less frequent appearance of lung diseases during the last years (Figure 10).[36]

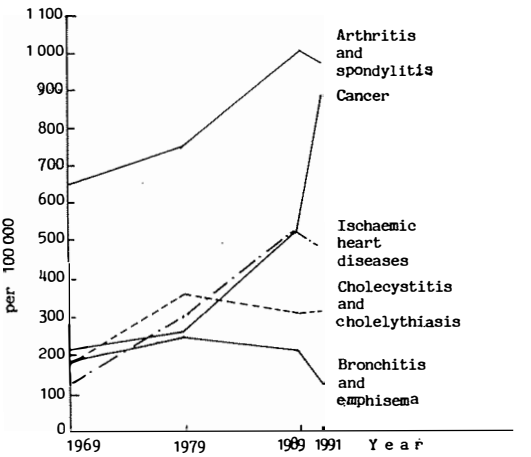


Figure 10. Diagnostic statistics in the out-patient specialist clinics, Slovenia, 1969–1991.[1]

From 1965 till 1984, a steep increase in cigarette production is characteristic. Afterwards, an obvious fall can be observed (Figure 11). Different studies on smoking habits of the Slovenians, performed between 1987 and 1992, including more than 18.000 of inquired persons aged between 15 and 101 years, showed that up to 40 % of men and up to 29 % of adult women within the inquired population were smokers; among the youth aged from 15 to 17 years, there were 17.9–27.8 % of smokers (boys and girls together). In comparison with other countries where smoking is already strongly restricted, having as a consequence a lower

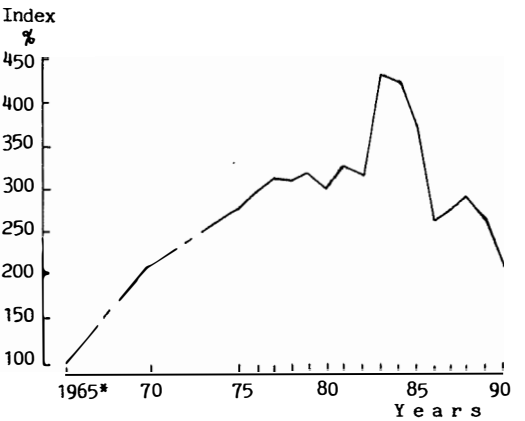


Figure 11. Production of cigarettes in Slovenia, 1965–1991.[5]

incidence of chronical diseases, the percentage of smokers in Slovenia is still very high.

A relatively rough indicator of hygienic irreproachability of food is the availability of refrigirators in households. Food kept in refrigirators is less prone to perishing and has a lower content of nitrites which are an important factor for the appearance of nitrosamines.[37, 38] The availability of refrigerators in households in 1978 was almost 79%, and rose in 1988 to almost 94%. This could be one of the reasons for a lower incidence of stomach cancer in the Republic of Slovenia. Chemical and microbiological pollution of our waters is still very high.

Increased serum cholesterol is an important risk factor for the development of atherosclerosis. Other risk factors (hypertension, smoking, diabetes, obesity, physical non-activity, behaviour pattern) are often connected with the hypercholesterolemy, but play a minor role in the case of its absence.[18] Cholesterol must achieve the value of 5 mmol/l to cause the development of atherosclerosis. Other risk factors do not play such an important role (18). Table 4 shows the rate of persons with total plasma cholesterol exceeding 5.2 mmol/l among the total of 4695 adult subjects from 5 wide epidemiologic studies performed in the Republic of Slovenia. The results show that more than a half of the examined subjects have an important and basic risk factor for the development of atherosclerosis – but these results cannot be generalized for the whole Slovene territory.

Six studies, published in Slovenia between 1987 and 1992, which included 8049 examined subjects (Table 5) showed that the population aged between 25 and 70 years had a relatively different prevalence of hypertension, which is also an important factor for the appearance of cardiovascular and cerebrovascular diseases. Such variability of results can also be a consequence of different methods for blood-pressure measurements.[39-41]

In five epidemiological studies which included 7572 subjects aged from 7 to 101 years we could observe excessive body weight and obesity in 9.6% of the examined men aged between 25 and 64 years, and 22.2–41% of the examined women. Among children aged between 7 and 15 years there were only 2.8–4.7% of boys and 4.8–7.7% of girls with excessive body weight (Table 6). Relatively high body weight of the subjects, although their daily energy consumption is relatively low, can be a consequence of insufficient physical activity and of too high a content of fats in the daily nutrition.[42, 43]

Table 4. Incidence of elevated plasma cholesterol levels in Slovenia (>5.2 mmol/l).

Place of research	% subjects Men	Women	No	Age groups
Zgornja Ščavnica (8)	79.3	76.3	1132	25–64
Brnik (8)	58.6	54.1	743	25–64
Ljubljana (14)	67.0	60.0	1692	25–64
Ljubljana (9)	47.9	67.8	696	60–94
Ljubljana* (10)	17.1	30.7	432	60–101

* institutionalized subjects

Conclusion

We could conclude that a relatively high incidence and prevalence of chronical and degenerative diseases in the Republic of Slovenia, or their constant increase, – if compared with western countries where it is lower and has been decreasing for several years already, – is

Table 5. Incidence of elevated blood pressure (mm Hg) in Slovenia.

Place of research	% subjects Men	Women	Together	No	Age groups
Ljubljana Šiška (15)	–	–	18.9	2965	40–70
Zgornja Ščavnica (8)	17.6	22.9	–	1132	25–64
Brnik (8)	17.0	17.1	–	743	25–64
Ljubljana (14)	47.3	30.9	39.1	1692	25–64
Ljubljana (9)	37.9	51.2	46.1	695	60–94
Ljubljana* (10)	37.1	50.2	47.9	822	60–101

* institutionalized subjects

Table 6. Obesity in men and women in Slovenia.

Place of research	Indices of obesity	% *		No	Age groups
		Men	Women		
Ljubljana (14)	BMI	49.0	41.0	897	25–64
Ljubljana (9)	Q	69.9	61.9	699	60-94
Zgornja Ščavnica (8)	RTM	9.6	22.2	1132	25-64
Brnik (8)	RTM	19.2	23.0	743	25–64
Maribor (16)	RTM	4.7	7.7	1033	7
	RTM	6.3	4.6	1107	11
	RTM	2.8	4.8	928	15

BMI = body mass index (kg/m^2); * > 2.7
RTM = relative body mass: (%); * > 120
Q = Quetelet's index (body mass/body height^2 (g/cm^2)); * > 2.57

a consequence of a much too intensive presence of risk factors in the Republic of Slovenia. Only after a change of the policy concerning nutrition, environmental protection, medical education and a changed medical welfare service in general, it will be possible to lower the incidence of these diseases in Slovenia.

References

1. Health Statistics Annual – Slovenia, 1991. *Zdrav Var* 1992; (Suppl 2): 1 – 488.
2. Helsing E. *Food Policy*. 1991; **16** (5): 371 – 82.
3. Berger DM, Turk J, Florjančič M. Nekaj pomembnih podatkov o boleznih srca in ožilja v Sloveniji. *Zdrav Var* 1992; **31:** 57-61.
4. Kirn PV, Žakelj PM, Ferligoj A, Škrk J. *Atlas of Cancer Inci dence in Slovenia 1978-1987*. Onkološki inštitut, Ljubljana 1992; 3-105.
5. *Statistični letopis R Slovenije 1985,* 1991. Zavod R Slovenije za, Ljubljana 1991; 21-659.
6. Rak KA, Antonić K. *Tablice o sastavu namirnica i pića*. Zavod za zaštitu zdravlja SR Hrvatske, Zagreb 1990; 3-143.
7. Zaveršnik H, Ozim V. Magnezij v naših pitnih vodah. *Zdrav Vestn* 1983; **52:** 179-82.
8. Berger MD, Ravnikar B, Jezeršek P, Lovše B. Razširjenost nekaterih znanih dejavnikov tveganja za bolezni srca in ožilja v zgornji Ščavnici in Braniku. *Zdrav Var* 1992; **31:** 63-70.
9. Accetto B. Zdravstveno stanje starejših ljudi na področju Ljubljane. UKC Medicinska fakulteta. Raziskovalno poročilo, Ljubljana 1987; 1-145.
10. Pokorn D, Accetto B. Nutritional status of the elderly in Ljubljana, 1985-1987. In E. Ancona et all. *Problems in Aging: Epidemiology, Health and Social Care*. Alps-Adria Community Sympo- sium, University of Padua, Padua 1989; 80-93.
11. Srebot RM, Javornik A. Kajenje med srednješolci v Kranju. *Zdrav Vestn* 1989; **58:** 289-90.
12. Fortič B. Razvada kajenja pri slovenskih zdravnikih in njene posledice – preliminarni rezultati študije 3595 zdravnikov z dobo opazovanja od 1972 do 1986. *Zdrav Var* 1988; **27:** 227-34.
13. Strgar E. Razširjenost kajenja med slovenskimi srednješolci. *Zdrav Var* 1991; **30:** 67-70.
14. Gradišek A, Šoln D, Tršan V, Zakotnik J. Študija dejavnikov tveganja za nastanek kroničnih nalezljivih bolezni v Ljubljani. *Zdrav Var* 1992; **31:** 71-7.
15. Jezeršek P, Dolenc P, Jezeršek B. Epidemiologija arterijske hipertenzije. *Zdrav Vestn* 1990; **59:** 153-7.
16. Radisavljević T, Turk MD, Nikolić T. Epidemiološka študija debelosti šolskih otrok in mladostnikov v Mariboru. *Zdrav Vestn* 1992; **61:** 621-3.
17. Pokorn D, Gregorič B, Poklar T, Eržen N. Ocena prehrane v domovih za starejše občane v Ljubljani. *Zbornik Biotehniške fakultete v Ljubljani* 1991; **57:** 259-71.
18. Carleton RA. Report of the Expert Panel on Population Strategies for Blood Cholesterol Reduction. A Statement from the National Cholesterol Education Program, NHLBI, NIH. Special Report. *Circulation* 1991; **83:** 2154-232.
19. Ulbricht TL, Southgate DAT. Coronary heart disease: seven factors. *Lancet* 1991; **338:** 985-92.
20. Rogers AE, Longnecker MP. Biology of Disease. Dietary and Nutritional Influences on Cancer: A Review of Epidemiologic and Experimental Data. *Laboratory investigation* 1988; **59:** 729-61.
21. Vrbič V, Premik M. Prevalence of Dental Caries in the Population of Slovenia. 10. Conferenza Di Aggiornamento Dell Ambito Della Comunita "Alpe-Adria", Portorož 1993; **16:** (Abstracts).
22. WHO. *Healthy Nutrition: Preventing Nutrition – Related Diseases in Europe*. Nutrition Unit Copenhagen, July 1986.
23. Ziegler RG. Vegetables, fruits, and carotenoids and the risk of cancer. *Am J Clin Nutr* 1991; **53:** 2518-98.

24. Gey KF, Brubacher BG, Stahelin HB. Plasma levels of antioxidant, vitamins in relation to ischemic heart disease and cancer. *Am J Clin Nutr* 1987; **45:** 1368-77.

25. Trout DL. Vitamin C and cardiovascular risk factors. *Am J Clin Nutr* 1991; **53:** 3228-58.

26. Block G. Vitamin C and cancer prevention: the epidemiologic evidence. *Am J Clin Nutr* 1991; **53:** 2708-828.

27. Fraser GE, Beeson WL, Philips RL. Diet and Lung Cancer in California Seventh-day Adventists. *Am J Epidemiol* 1991; **133:** 683-93.

28. Heaton KW. The sweet road to gall stones. *BMJ* 1984; 1103-4.

29. Thijs C, Knipschild P, Leffers P. Is Gallstone Disease Caused by Obesity or by Dieting? *Am J Epidemiol* 1992; **135:** 274-80.

30. Schroeder HA, Brattleboro W. Relation between mortality from cardiovascular disease and treated water supplies. *JAMA* 1960; **172:** 1902-8.

31. Mezey E. Alcoholoc liver disease: roles of alcohol and malnutrition. *Am J Clin Nutr* 1980; **33:** 2709-18.

32. Breslow NE, Enstrom JE. Geographic Correlations Between Cancer Mortality Rates and Alcohol-Tobaco Consumption in the United States. *J Nate Cancer Institute* 1974; **53:** 631-9.

33. Vitale JJ, Gottlieb LS. Alcohol and Alcohol-related Deficiences as Carcinogens. *Cancer Research* 1975; **35:** 3336-8.

34. Hinds MW, Kolonel LN, Lee J, Hirohata T. Associations between cancer incidence and alcohol/cigarette consumption among five ethnic groups in Hawaii. *B. J. Cancer* 1980; **41:** 929-40.

35. Haenszel W, Correa P. Developments in the Epidemiology of Stomach Cancer over the Past Decade. *Cancer Research* 1975; **35:** 3452-9.

36. Sunyer J, Anto JM, Murillo C, Saez M. Effects of Urban Air Pollution on Emergency Room Admissions for Chronic Obstructive Pulmonary Diseasae. *Am J Epidemiol* 1991; **134:** 277-86.

37. Ames BN. Dietary Carcinogens and Anticarcinogens. *Science* 1983; **221:** 1256-63.

38. Tannenbaum SR, Wishnok JS, Leaf CD. Inhibition of nitrosamine formation by ascorbic acid. *Am J Clin Nutr* 1991; **53:** 2478-509.

39. Hypertension and coronary heart disease: classification and criteria for epidemiological studies. First report of the expert committee on cardiovascular diseases and hypertension. Technical report series No. 168. Geneva: World Health Organization, 1959.

40. National high blood pressure education program working group report on risk and high blood pressure. An epidemiological approach to describing risk associated with blood pressure levels. *Hypertension* 1985; **7:** 641-52.

41. Rocella EJ, Bowler AE, Horan M. Epidemiologic considerations in defining hypertension. *Med Clin North Amer* 1987; **71:** 785-802.

42. Lissner L, Levitsky DA, Strupp BJ, Kalkwarf HJ, Rol AD. Dieta- ry fat and regulation of energy intake in human subjects. *Am J Clin Nutr* 1987; **46:** 886-92.

43. Astrad PO.Physical activity and fitness. *Am J Clin Nutr* 1992: 12318-69.

Radiol Oncol 1994; 28: 49-57.

The characteristic angle-β concept in electron arc therapy

Marina Pla, Ervin B. Podgoršak, and Conrado Pla

Department of Medical Physics, Montreal General Hospital, McGill University, Montréal, Québec H3G 1A4, Canada

Electron arc therapy *is a special radiotherapeutic technique in which a rotational electron beam is used to treat superficial tumour volumes which follow curved surfaces. While the technique is well known and accepted as clinically useful in the treatment of certain tumours, it is not widely used because it is relatively complicated and its physical characteristics are poorly understood. Moreover, the dependence of dose distributions on a large number of physical and treatment parameters makes treatment planning in electron arc therapy very difficult even for homogeneous media.*

The excellent clinical results achieved by the few pioneers in this field during the past two decades have certainly stimulated an increased interest in electron arc therapy, both for curative treatments as well as for palliation. In fact, manufacturers of linacs now offer the continuous electron arc therapy mode as one of the standard treatment options. While this option is usually purchased with a new linac since it is relatively inexpensive, it is rarely used clinically because of the technical difficulties involved. However, the number of centres using or planning to use this treatment modality is growing, making the improved understanding of technical and physical aspects of electron arc therapy highly relevant.

The characteristic angle-β concept provides a useful empirical approach to treatment planning in electron arc therapy. The concept is reviewed and its applicability is expanded from homogeneous to heterogeneous phantoms. It is also shown that for homogeneous phantoms depth dose data at a particular electron beam energy can be calculated from a data set measured at some other standard energy.

Key words: neoplasms-radiotherapy; electron arc therapy

Introduction

The particular energy loss characteristics of electrons as they penetrate into tissue make electrons suitable for use in treatment of superficial malignant diseases. Stationary electron beams are now routinely used in radiotherapy and several radiotherapy centres have during the past two decades, in addition to stationary electron beam techniques, developed *moving electron beam* techniques which are ussually referred to as *electron arc therapy*.

Electron arc therapy is well-suited for treatment of large superficial tumour volumes which follow curved surfaces. The treatment is performed either with a continuous beam-on rotation of the electron beam *(continuous arc)* or with

Correspondence to: Marina Pla, M. Sc., Department of Medical Physics, Montreal General Hospital, 1650 avenue Cédar, Montréal, Québec H3G 1A4.

UDC: 616-006.6:615.849

a series of overlapping isocentric stationary electron beams *(pseudo arc)*. Electron arc therapy has proved useful in the treatment of patients with recurrent malignant chest wall disease (e.g., breast cancer) who had failed previous conventional photon beam radiotherapy or patients with extensive superficial skin tumours involving large curved surfaces of the body (e.g., sarcomas, limited mycosis fungoides, extensive basal or squamous cell carcinomas, and limited lymphomas).

In our department we have been using electron arc therapy clinically since 1986. During this time we have treated 43 patients and achieved excellent local palliation of the disease in most of them. The clinical experience with our pseudo arc electron therapy on 24 patients treated between November 1986 and June 1990 has been described in detail elsewhere[1] including information on tumour types, tumour locations, tumour control and morbidity.

The calculation of dose distributions in electron arc therapy is a complicated procedure and usually cannot be performed reliably with algorithms used for standard stationary electron beam treatment planning. The dose distributions in electron arc therapy depend in a complicated and seemingly haphazard way on several treatment parameters, such as the electron field width, depth of isocentre, source-axis distance, electron beam energy, surface curvature of the patient, use of secondary and tertiary collimation, field shape as defined by the secondary collimator, and number of monitor units either per degree in *continuous arcs* or per each stationary beam in *pseudo arcs*.

The difficulties in optimization of the treatment parameters for a particular patient make electron arc therapy very complex and prevent its wider use in standard radiotherapy departments. The few radiotherapy centres which to date have used electron arc therapy clinically[1–11] have developed their own specific solutions to the technical problems related to the treatment itself and to the calculation of the dose distributions inside and outside the targeted volume. In our centre we have developed an empirical method, referred to as the characteristic angle-β concept, for the calculation of dose distributions in electron arc therapy. In this paper we review the angle-β concept and extend it to account for multiple electron energies and tissue heterogeneities.

Materials and methods

Experimental apparatus and techniques.

An isocentric linear accelerator (Clinac-18, Varian Associates, Alto Palo, California), capable of producing electron beams in the energy range between 6 MeV and 18 MeV, has been used as the source of electrons. The virtual source of the stationary electron beam is at 85 cm from the isocenter axis making our nominal source-axis distance in the pseudoarc technique equal to 85 cm. The electron beam depth dose distributions for stationary and arc therapy beams were measured in cylindrical phantoms with thermoluminescent dosimetry (TLD) techniques incorporating a TLD reader (model 2000, Harshaw Chemical Company, Solon, Ohio) and dosimeters in the form of LiF rods (TLD-100, Harshaw Chemical Company, Solon, Ohio) with dimensions of $1 \times 1 \times 6\,mm^3$.

Characteristic angle-β concept.

Before introducing the electron arc therapy clinically, we initiated a study of the basic physics of electron arc therapy and developed the characteristic angle-β concept for an empirical, yet general, description of dose distributions in electron arc therapy. The angle-β concept has been described in detail elsewhere,[12] so that here only a concise description of its main features will be given.

For a given electron arc treatment geometry a characteristic angle β can be uniquely determined by three treatment parameters: nominal field width w, depth of isocentre d_i, and virtual source-axis distance f. As shown in Figure 1, the characteristic angle β for an arbitrary point Q on the patient's surface is measured between the central axes of two rotational electron beams positioned in such a way that at point

Q the frontal edge of one beam crosses the trailing edge of the other beam. The treatment arc angle α is of course much larger than the characteristic angle β.

The relationship among the parameters β, d_i, w, and f is then through straightforward geometry given by:

$$w = \frac{2d_i \sin\left(\frac{\beta}{2}\right)}{1-\left(\frac{d_i}{f}\right)\cos\left(\frac{\beta}{2}\right)} \quad (1)$$

and hence one obtains the following quadratic equation for calculating β:

$$\left(\frac{4d^2}{w^2}-1\right)\tan^2\left(\frac{\beta}{2}\right) + 4\left(\frac{d_i^2}{fw}\right)\tan\left(\frac{\beta}{2}\right) + \left(\frac{d_i^2}{f^2}-1\right) = 0 \quad (2)$$

We found experimentally that, for a constant f, electron beams with combinations of d_i and w which give the same characteristic angle β exhibit very similar radial percantage depth dose characteristics even though they may differ considerably in their individual d_i and w. The radial depth doses for a point of interest Q on the contour surface are measured along a direction perpendicular to the contour. This direction may but does not necessarily coincide with the direction of d_i for the point of interest on the contour surface.

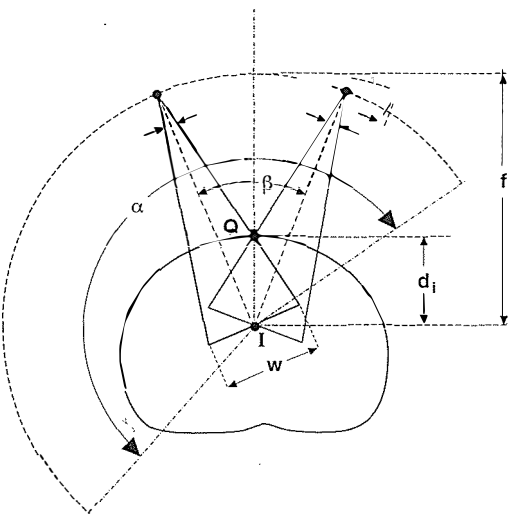


Figure 1. Schematic representation of the electron arc therapy geometry (α = arc therapy angle, β = characteristic angle, I = isocentre, d_i = depth of isocentre, w = field width, f = virtual source-axis distance, Q = point of interest on patient contour).

Isocentre depth

In addition to field size, the isocentre depth generally also has a strong influence on the rotational electron dose distribution. The position of the isocentre, however, cannot be chosen arbitrarily, rather it is fairly rigidly determined by the contour of the patient within the treatment volume. An attempt is usually made to approximate the contour within the treatment volume by an appropriate segment of a circle and this then does not leave much leeway in the choice of the actual position of the isocentre. Since the virtual source-axis distance is fixed for a given therapy machine and the position of the isocentre is determined by a given treatment geometry, the field width becomes the important parameter, the choice of which will strongly influence the dose distribution in the treatment volume and also affect the bremsstrahlung dose outside the treatment volume.

Field width

In the first approximation the field width in electron arc therapy is constant along the field length, i.e., the secondary collimator defines a rectangular field. However, if the patient profile changes drastically along the field length, then electron arc therapy with a constant field width will result in considerable dose inhomogeneities in the treatment volume. It has been shown,[7,13,14] that an appropriate modulation of the field width to compensate for changes in patient axial profiles will improve the dose distribution in the targeted volume. In general, one may thus envisage that the electron field

defined by the secondary collimation will be irregular; a considerable amount of work, however, will have to be done to understand the requirements for the appropriate field shape to accommodate a given clinical situation.

Bremsstrahlung contamination in electron arc therapy

The electron beam field size has a very pronounced effect not only on the treatment time and the dose distribution inside the targeted volume but also on the bremsstrahlung contamination of the moving electron beam. This effect is sometimes overlooked in clinical applications of electron arc therapy and may result in inadvertent, yet serious, radiation induced damage to the patient. The photon contamination is generally aimed in the forward direction along the electron beam central axis. This results in the isocentre axis and its vicinity continuously irradiated by the bremsstrahlung photons during the electron beam on-time, and may result in the isocentre photon dose comparable to the prescribed electron dose. Thus the photon contamination of the electron beam and the photon dose at the isocentre are of clinical importance and both should be minimized in electron arc therapy.[15,16] We have shown recently[17] that the isocentre dose in arc therapy is inversely proportional to the characteristic angle β.

Other treatment parameters

The choice of the appropriate beam parameters in electron arc therapy also depends on the target volume, patient contour, and the desired dose distribution within the target volume. As discussed above, the isocentre position is usually determined by approximating with a section of a circle the patient contour abutting the target volume. The isocentre depths d_i are thus pre-determined for all surface points of interest. The virtual source-axis distance f is of course fixed for a given treatment machine. For a particular patient geometry the electron beam energy and the characteristic angle β are determined from a pre-measured set of depth dose data with the electron energy and β as parameters. Both the electron energy and β are chosen such that the radial depth doses for a given reference point on the patient's surface provide adequate dose distribution within the target volume. Once β and d_i are known, the required treatment field size w is calculated from Eq. 2.

Results and discussion

Validity of the angle-β concept.

The validity of the angle-β concept is confirmed in Figure 2 where we plot for a 9 MeV electron

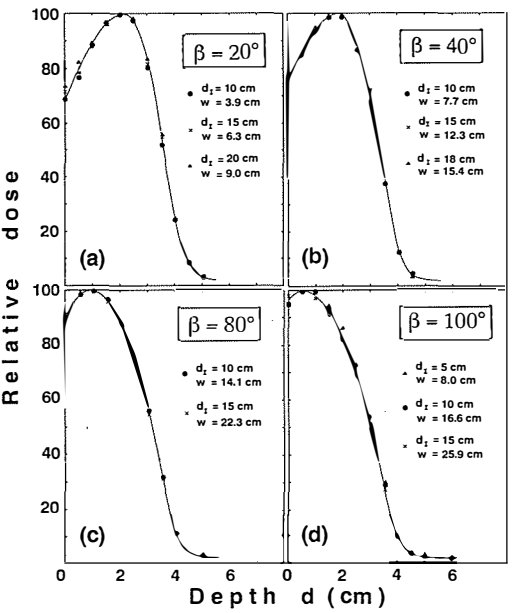


Figure 2. Radial percentage depth doses in electron arc therapy measured in a homogeneous phantom for various combinations of field size w and isocenter depth d_i giving characteristic angles β calculated from Eq. (2). Electron beam energy: 9 MeV.

beam the measured radial percentage depth doses for various combination of w and d_i giving characteristic angles β of (a) 20°, (b) 40°, (c) 80°, and (d) 100°. The agreement among the depth dose curves measured for various w and d_i giving the same β is excellent in the practical range for angle β from 20° to 100°

even though the individual beams may vary considerably in w and d_i. The solid curves of Figure 2 represent the average values for the depth dose curves measured for a given angle β with various combinations of w and d_i.

To study the effect of the angle β on the rotational electron depth doses further we display in Figure 3 the data of Figure 2, in addition to curves representing measured data for a stationary 9 MeV electron beam as well as for a rotational beam with a β of 60°. For small angles β the radial percentage depth doses are similar to those obtained for a stationary electron beam of the same energy. As the angle β increases, however, the beams become less penetrating, the depth of dose maximum moves towards the surface, and the surface doses increase. The disadvantage of a larger β in arc therapy is a lower beam penetration associated with a shallower dose fall-off beyond the depth of dose maximum. Its advantage, on the other hand, is a larger skin dose and, as shown before,[17] a lower photon dose at the isocentre.

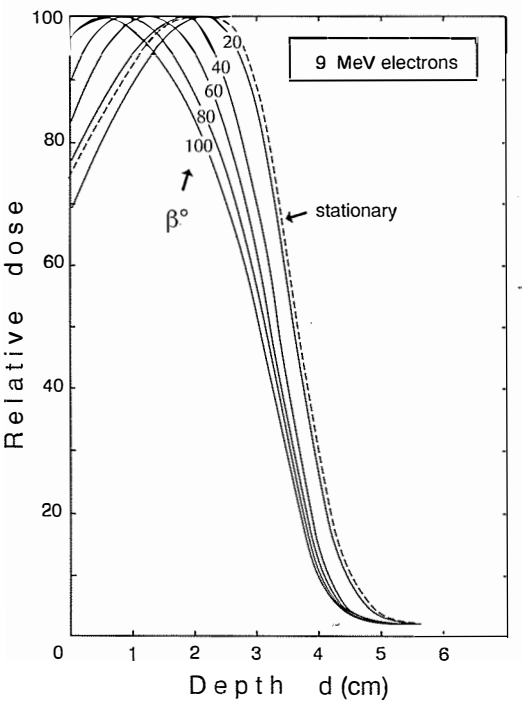


Figure 3. Radial percentage depth doses for electron arc therapy in a homogeneous phantom (solid curves) for characteristic angles β from 20° to 100°. Electron energy: 9 MeV. The dashed curve is the depth dose distribution for a stationary 9 MeV beam.

The characteristic angle-β concept thus gives a simple and practical method for the calculation of isodose distribution in electron arc therapy through a determination of radial percentage depth doses for a series of surface points in the treatment volume. The radial percentage depth doses for arbitrary surface points in the target volume can be accurately predicted from a pre-measured set of depth dose data for various angles β at a given electron beam energy. These depth doses, however, are all normalized to 100 for the dose at the depth of dose maximum d_{max} for each particular surface point of interest.

Treatment planning

The relationship between the dose at one surface point to the dose at another surface point, for a given set of pseudo arc parameters, has to be known if the radial percentage depth doses for various surface points are to be used in the determination of actual isodose distributions in the target volume. In clinical practice patient contours cannot always be approximated by segments of a circle and this then results in variations in d_i from one surface point to another in the transverse slice. Since w is constant during the arc therapy, the variation in d_i results in variations in angle β, which in turn implies variations in surface doses and relative radial depth doses from one surface point to another.

Our method to determine the isodose distributions for an arbitrary set of surface points was published recently.[11] The actual isodose distributions may be reconstructed from the knowledge of the radial percentage depth doses for various surface points in the target volume, provided that the surface dose values are renormalized to the dose at a given surface reference point. We have found experimentally that for a given f and w the measured surface doses for arbitrary surface points in electron arc therapy are related through an inverse square law incor-

porating the change in distance between the virtual source and the surface point as well as through a linear relationship accounting for the change in the characteristic angle β. The relationship between D_A, the surface dose for a reference surface point A, and D_Q, the surface dose for an arbitrary surface point Q, is empirically given by:

$$D_Q = D_A \left\{ \frac{[f - d_i(A)]}{[f - d_i(Q)]} \right\}^2 \frac{ß_Q}{ß_A} \quad (3)$$

where f is the virtual source-axis distance, and $d_i(A)$ and $d_i(Q)$ are the isocentre depths for surface points A and Q, respectively. The radial depth doses for each surface point on the central contour are thus renormalized to the value at the surface reference point, resulting in an isodose distribution, which in homogeneous phantoms quite reliably describes the actual distribution obtained during the arc therapy.

The method above assumes the same number of monitor units per each pseudo arc beam or a constant beam output in continuous arcs. If the resulting dose distribution is too inhomogeneous because of large variations in d_i, then an improvement can be achieved by adjusting the monitor units per pseudo arc beam or by modulating the beam output in continuous rotations to even out the underdosed and overdosed regions. We have studied the first method and found that with an appropriate adjustment of monitor units per pseudo arc beam we can achieve an adequate dose homogeneity even in very irregular geometries treated with pseudo arc electron beams.

The characteristic angle-β concept presented so far was valid only for homogeneous phantoms and electron beam energies equal to those for which the pre-measured set of depth doses was obtained. We have recently extended the angle-β concept to incorporate multiple electron beam energies and various tissue inhomogeneities.

Dependence of radial depth doses on electron beam energy

In electron arc therapy it is relatively simple to determine the depth dose distributions for an arbitrary electron energy E from a set of depth dose distributions measured for a nominal electron beam energy E_o. The procedure involves accounting for relative characteristic angles β of the rotating beams and practical ranges of the stationary beams with energies E and E_o. The dose at depth d for energy E, D(d, E) is calculated from the dose at depth d_o of energy E_o, $D(d_o, E_o)$ with the following empirical relationship:

$$D(d,E) = D(d_o, E_o) \frac{ß\{d_i - d_{max}(E)\}}{ß\{d_i - d_{max}(E_o)\}}, \quad (4)$$

were depth of dose maximum is represented by d_{max}, and depths d and d_o are related by the following relationship:

$$d = d_o \frac{R_p(E)}{R_p(E_o)}; \quad (5)$$

similarly $d_{max}(E)$ and $d_{max}(E_o)$:

$$d_{max}(E) = d_{max}(E_o) \frac{R_p(E)}{R_p(E_o)}, \quad (6)$$

where $R_p(E)$ and $R_p(E_o)$ are the physical ranges of the stationary electron beams with energies E and E_o, respectively.

An example is shown in Figure 4 with 12 MeV and 15 MeV rotational electron beam depth doses calculated from data measured at 9 MeV for a polystyrene cylindrical phantom with a radius of 15 cm. For all energies, the same dose was given per each beam in the pseudo arc, the characteristic angle β on the phantom surface was 40°, and doses are normalized to 100 % at depth of dose maximum for the 9 MeV rotational beam. The increase in d_{max} for the 12 MeV and 15 MeV rotational beams is predicted by Eq. (6). The relative doses at d_{max} for the 12 MeV and 15 MeV rotational beams are larger than the d_{max} dose for the 9MeV beam because the d_{max} points for the 12 MeV and 15 MeV beams are deeper in the phantom and therefore exposed to the

electron beams for a longer time during the beam rotation. The relative dose values at d_{max} for the various beam energies can be determined using Eq. (4). The agreement between the depth doses measured at 12 MeV and 15 MeV and those calculated from Equations (4), (5) and (6) for the two electron energies is excellent indicating that Eq. (4) may be used to calculate radial depth doses at an arbitrary energy E from the known set of arc therapy depth doses at a nominal energy $E_{\bullet}$.

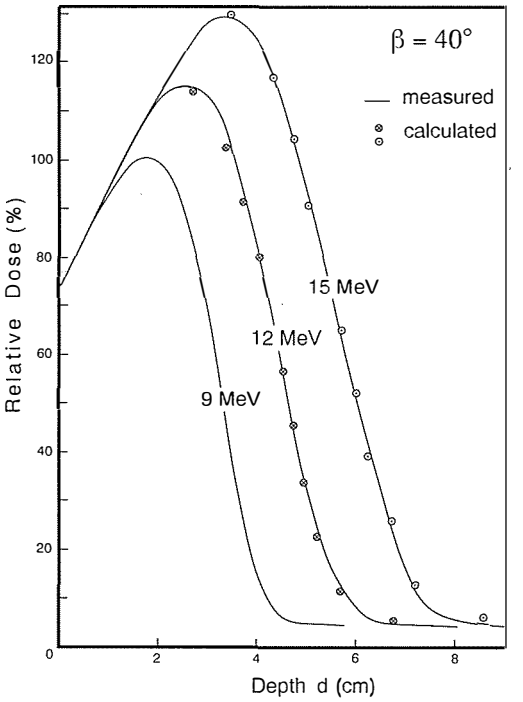


Figure 4. Radial depth doses in electron arc therapy on a homogeneous cylindrical phantom (radius: 15 cm) for electron energies of 9 MeV, 12 MeV and 15 MeV. Solid curves represent measured data, the data points for 12 MeV and 15 MeV were calculated from the 9 MeV data with Equations (4), (5) and (6).

Dependence of radial depth doses on phantom density

Using a set of radial depth dose data measured for a given electron energy in a homogeneous phantom (for example: 9 MeV electrons in a cylindrical polystyrene phantom), we can also predict dose distributions in heterogeneous phantoms with the same electron energy, as shown with the example given in Figure 5. The solid curve represents electron arc therapy radial depth doses measured in a homogeneous polystyrene cylindrical phantom (radius: 15 cm), while the dashed curve represents depth doses measured in a composite cylindrical phantom consisting of a wood cylinder (density $\varrho = 0.3\,g/cm^3$, radius: 13 cm) surrounded by a polystyrene tube with a wall thickness of 2 cm. The data points represent radial depth doses calculated with the following empirical relationship:

$$D(d, \varrho) = D(d_o, \varrho_o)\,\frac{ß\{d_i - d_{max}(\varrho)\}}{ß\{d_i - d_{max}(\varrho_o)\}} \tag{7}$$

with

$$d_{max}(\varrho) = [d_{max}(\varrho_o) - t]\,\frac{\varrho}{\varrho_o} + t \tag{8}$$

and

$$d = [d_{\bullet} - t]\,\frac{\varrho}{\varrho_o} + t \tag{9}$$

where t stands for the thickness of the polystyrene tube of density ϱ_0; d and d_0 are depths in the phantom, and all other parameters were defined above. For t = 0 the heterogeneous phantom becomes a homogeneous phantom and Equations (7), (8) and (9) become essentially identical to Equations (4), (5) and (6), respectively. For both phantoms of Figure 5, the same dose was given per each beam in the pseudo arc, the characteristic angle β on the phantom surface was 60°, and doses are normalized to 100 % at d_{max} for the homogeneous phantom. The increase in d_{max} for the heterogeneous phantom is predicted by Eq. (8) and the relative doses at d_{max} are given by Eq. (7). The composite phantom is of lower density than the unit density homogeneous phantom. This results in larger d_{max} and this in turn results in a larger relative dose because of the longer time the d_{max} point spends in the rotational beam.

As shown in Figure 5, the agreement between the radial depth doses measured in the heterogeneous phantom and those calculated from

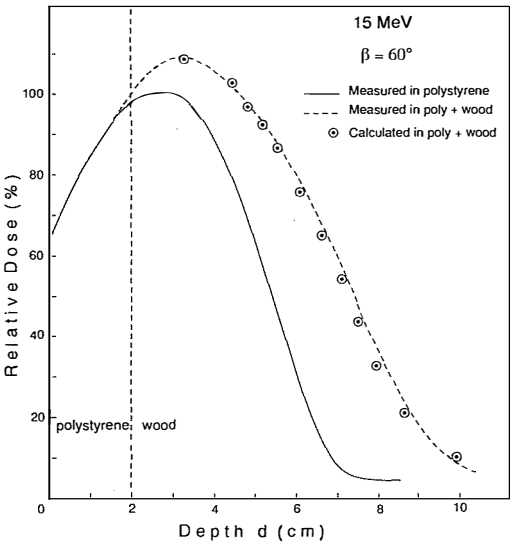


Figure 5. Radial depth doses in electron arc therapy on a heterogeneous cylindrical phantom. Solid curve: measured in a homogeneous phantom. Dashed curve: measured in a heterogeneous phantom. Data points: calculated from solid curve with Equations (7), (8) and (9).

homogeneous phantom data is excellent, indicating that the modified angle-β concept could be developed for use in dose distribution calculation in electron arc therapy involving heterogeneous media.

Conclusions

Electron arc therapy is a special radiotherapeutic technique in which a rotational electron beam is used to treat superficial tumour volumes which follow curved surfaces. While the technique is well known and accepted as clinically useful in the treatment of certain tumours, it is not widely used because it is relatively complicated and its physical characteristics are poorly understood. Moreover, the dependence of dose distributions on a large number of physical and treatment parameters makes treatment planning in electron arc therapy very difficult even for homogeneous media. Current commercial treatment planning software has considerable difficulties accounting for the various physical parameters affecting the dose distribution in arc therapy, even more so when tissue heterogeneities are involved. These difficultes seriously impede a widespread and routine use of electron arc therapy.

The excellent clinical results achieved by the few pioneers in this field during the past two decades have certainly stimulated an increased interest in electron arc therapy, both for curative treatments as well as for palliation. In fact, manufacturers of linacs now offer the continuous electron arc therapy mode as one of the standard treatment options. While this option is usually purchased with a new linac since it is relatively inexpensive, it is rarely used clinically because of the difficulties discussed above.

In this paper we have discussed the characteristic angle-β concept, an empirical approach which we have developed for calculation of dose distributions in electron arc therapy. The concept has been used clinically for homogeneous phantoms and we have shown here that there is a potential for its extension to multiple electron beam energies and tissue heterogeneities. Preliminary measurements in cylindrical phantoms have shown that, based on a set of radial depth dose data measured in a homogeneous phantom at a given electron beam energy, we can predict (i) radial depth doses in homogeneous phantoms at other electron beam energies, and (ii) radial depth doses in heterogeneous phantoms for the same electron beam energy. In both cases the radial depth doses are calculated on an absolute scale, an approach which will lend itself well for the development of arc therapy dose calculation software both in homogeneous and heterogeneous media.

References

1. McKenzie MR, Freeman CR, Pla M, Guerra J, Souhami L, Podgorsak EB. Clinical experience with electron pseudo arc therapy. *Brit J Radiol* 1993; **66:** 234–40.
2. Khan FM, Fullerton GD, Lee JMG, Moore VC, Levitt SH. Physical aspects of electron beam arc therapy. *Radiology* 1977; **124:** 497–500.
3. Ruegsegger DR, Lerude SD, Lyle D. Electron beam arc therapy using a high energy betatron. *Radiology* 1979; **133:** 483–9.

4. Blackburn BE, Moreland R. An improved method of electron arc therapy for chest wall irradiation. *Int J Radiat Oncol Biol Phys* 1980; **6:** 1373.

5. Boyer AL, Fullerton GD, Mira JG. An electron beam pseudo arc technique for irradiation of large areas of chest wall and other curved surfaces. *Int J Radiat Oncol Biol Phys* 1982; **8:** 1969–74.

6. Peacock LM, Leavitt DD, Gibbs FA, Stewart JR. Electron arc therapy: Clinical experience with chest wall irradiation. *Int J Radiat Oncol Biol Phys* 1984; **10:** 2149–53.

7. Leavitt DD, Peacock LM, Gibbs FA, Stewart JR. Electron arc therapy: Physical measurements and treatment planning techniques. *Int J Radiat Oncol Biol Phys* 1985; **11:** 987–99.

8. McNeely LK, Jacobson GM, Leavitt DD, Stewart JR. Electron arc therapy: Chest wall irradiation of breast cancer patients. *Int J Radiat Oncol Biol Phys* 1988; **14:** 1287–94.

9. Leavitt DD, Stewart JR, Moeller JH, Earley L. Optimization of electron arc therapy doses by multi-vane collimator control. *Int J Radiat Oncol Biol Phys* 1989; **16:** 489–96.

10. Leavitt DD, Stewart JR, Moeller JG, Lee WL, Takach GA. Electron arc therapy: Design, implementation and evaluation of a dynamic multi-vane collimator system. *Int J Radiat Oncol Biol Phys* 1989; **17:** 1089–94.

11. Pla M, Podgorsak EB, Pla C, Freeman CR, Souhami L, Guerra J. Physical aspects of the angle-β concept in electron arc therapy. *Int J Radiat Oncol Biol Phys* 1990; **20:** 1331–9.

12. Pla M, Pla C, Podgorsak EB. The influence of beam parameters on percentage depth dose in electron arc therapy. *Med Phys* 1988; **15:** 49–55.

13. Hogstrom KR, Leavitt DD. Dosimetry of electron arc therapy. In: Kereiakis JG, Elson HR, Born CG eds. *Radiation Oncology Physics-1986,* New York: American Institute of Physics. (Medical Physics Monograph No. 15): 1987; 265–95.

14. Pla M, Podgorsak EB, Pla C, Freeman CR. Determination of secondary collimator shape in electron arc therapy. *Phys Med Biol* 1993; **38:** 999–1006.

15. Kase KR, Bjärngard BE. Bremsstrahlung dose to patients in rotational electron therapy. *Radiology* 1979; **133:** 531–2.

16. Leavitt DD, Gibbs FA, Moeller JH. Electron arc therapy: influence of heterogeneities on dose to blood-forming organs. *Radiology* 1986; **161:** 248.

17. Pla M, Podgorsak EB, Pla C. Electron dose rate and photon contamination in electron arc therapy. *Med Phys* 1989; **16:** 692–7.

Radiol Oncol 1994; 28: 58-62.

Is a single film-dosimeter enough for monitoring the radiation dose to the interventional radiologist?

András Kónya and Zoltán Vígváry

Department of Radiology, Semmelweis University of Medicine H-1082 Üllöi út 78/a, Budapest, Hungary

Personnel exposure to radiation was investigated during vascular interventions (VI) and percutaneous biliary interventions (PTBD). In this study TLDs were applied for each of the following sites: radiologist's eyes, thyroid, hands as well as on the trunk at chest and gonad level under the apron. Dose during vascular sessions (75 min.) and PTBD sessions (58 min.) was measured by TLDs, from these data the dose rates relevant to the given regions were calculated. The radiologist' s dose rates were 41.3, 45.3, 52.0 and 24.0 μGy/min to the forehead, thyroid, left hand and right hand, respectively, during VIs. The corresponding values were 67.2, 112.0, 767.2 and 305.1 μGy/min, respectively, during PTBDs. The dose rates at chest level under the apron were 0.293 μGy/min during VIs and 1.91 μGy/min during PTBDs. The relative values expressing the relationship between the absorbed doses of the unprotected regions and the chest dose measured simultaneously under the apron were as follows: 140, 154, 177 and 81 as well as 35, 58, 400 and 159, to the radiologist's forehead, thyroid, left and right hand, during VIs and PTBDs, respectively. An individual dosimeter worn on the trunk at chest level under the protective apron appears insufficient for ensuring that the dose equivalent to any unprotected region does not exceed the relevant limits during interventional radiology procedures.

Key words: radiology, interventional-manpower; film dosimetry; individual dosemeters, personnel exposure to radiation.

Introduction

As an overwhelming majority of interventional radiology procedures are performed under fluoroscopy, this results in a considerable increase in radiation burden to the personnel, especially to the examiner. Parallelly to the development of interventional radiology, a discussion has been stirred up whether the radiation dose suffered by the staff could be adequately monitored by a single film-dosimeter, especially if this is worn under a protective apron. In the case of interventional radiologist, the doses received under the apron are numerically reported above the value of 0.4 mGy, but in such cases in unprotected regions very significant doses go unregistered.[1]

Correspondence to: Prof. András Kónya, Department of Radiology, Semmelweis University of Medicine, H-1082 Üllöi út 78/a, Budapest, Hungary.

UDC: 614.876

Our studies were conducted to estimate the exposure incurred by certain, significantly exposed body regions of the radiologist during both vascular and biliary interventions (VIs and PTBDs). Our investigations were also aimed at comparing the radiologist's relevant doses during two basically different types of interventions as well as at determining the numerical relationship between the doses absorbed by the unprotected regions and the chest dose measured simultaneously under a lead apron.

Material and methods

Interventions were carried out with an overhead x-ray tube and undercouch image intensifier (Siemens Biangulix 125/12/50 tube, focus diameter 1.3 mm, with 4 mm Al filtration, Tridoros 5S basic equipment with dose automatics). The radiation field was 12 × 10 cm, the average diameter of the patient's body was 20 cm. The x-ray beam was characterized by 95–110 kV, 4.5 – 6.5 mA.

The LiF thermoluminescent dosimeters (TLD) were prepared in the form of a disc or a capsule on a ring, attached to the proximal phalanx of the third finger of the radiologist's right and left hands, his collar outside the apron (thyroid), to the centre of his forehead and on the trunk at chest and gonad level under the protective apron.

We monitored the doses absorbed by radiologist during VIs and PTBDs where the fluoroscopy time was 75 min. and 58 min., respectively. From these data we calculated the dose rate relevant to the given regions (Table 1). The received doses were determined by a comparative method which has been described in detail in these galleys.[2]

Results

Absorbed doses as well as the calculated dose rates in different sites of the radiologist are presented in Table 1.

In the vascular studies the dose rate was the highest on the left hand (52.0 μGy/min) but these values were just slightly lower on the radiologist's forehead and thyroid (41.3 and 45.3 μGy/min). The dose measured on the righ hand (24.0 μGy/min) was about a half of the value registered on the left hand.

Owing to the radioprotective effect of the protective apron the doses measured under it were significantly lower. The relevant dose rate registered on the trunk at chest and gonad level were 0.293 and 0.1 μGy/min, respectively.

From the above mentioned data we can calculate relative values expressing the relationship between the radiation exposures of different body parts and the corresponding chest dose. These relative values were 140.9, 154.5, 177.2 and 81.8 to the radiologist forehead, thyroid, left and right hands, respectively, during VIs.

The highest dose during PTBDs (in 58 min.) was measured on the left hand 44.5 mGy which corresponds to 767.2 μGy/min dose rate. Less than a half of this value was registered on the right hand (305.1 μGy/min) while the third

Table 1. Radiation doses incurred by different body parts of radiologist with dose rate and relative values expressing the relationship between the doses to different uncovered parts and the corresponding chest doses under the apron.

Vascular (fluoroscopy time: 75 min)				Biliary (fluoroscopy time: 58 min)		
Measured dose (mGy)	Dose rate (μGy/min)	Relative value	Regions of body	Measured dose (mGy)	Dose rate (μGy/min)	Relative value
3.1	41.3	140.9	forehead	3.9	67.2	35.1
3.4	45.3	154.5	thyroid	6.5	112.0	58.5
0.022	0.293	1	chest	0.111	1.91	1
0.0076	0.1	0.345	gonad	0.09	1.55	0.81
3.9	52.0	177.2	left hand	44.5	767.2	400.9
1.8	24.0	81.8	right hand	17.7	305.1	159.4

highest dose rate was 112.0 μGy/min measured in the jugulum (thyroid).

It is noteworthy that the chest and gonad doses were significantly higher than the corresponding doses measured in the vascular studies (1.91 and 1.55 μGy/min vs. 0.293 and 0.1 μGy/min). This means that the interventionalist's chest and gonad regions are exposed to 6.5 times and 15.5 times higher doses, respectively, during PTBDs than VIs (Table 2).

Table 2. Dose rates for different body parts of the radiologist during PTBDs and VIs and their comparison to one another.

Body regions	Biliary μGy/min	Vascular μGy/min	Relative value
Forehead	67.2	41.3	1.62
Thyroid	112.0	45.3	2.47
Chest (under the apron)	1.91	0.293	6.51
Gonad (under the apron)	1.55	0.1	15.5
Left hand	767.2	52.0	14.75
Right hand	305.1	24.0	12.71

In view of the significant difference between the doses measured under the lead apron during PTBDs and VIs, the determination of the relative values expressing the relationship between doses of uncovered parts and the chest dose becomes of greater importance. The relative values were 35.1, 58.5, 400.9 (!) and 159.4 to the interventional radiologist's forehead, thyroid, left and right hands, respectively.

The great difference between the radiohygienic conditions during the vascular and biliary interventions can be demonstrated even more unequivocally if we compare the corresponding dose rates. Table 2 shows that while the radiologist's forehead and thyroid is exposed to "only" 1.5 – 2.5 times higher doses during PTBDs (1.62 and 2.47) compared to VIs's conditions, these relative values are much higher considering the under-the-apron values (6.5 and 15.5 at the heart and gonad level, respectively) and extremely high for the hands (14.7 and 12.7), taking into account the fact that these values expressed in absolute numbers are much higher because they are measured in uncovered regions.

Discussion

Interventional radiologists receive different but very significant doses directly associated with the type of work performed. One has to take into consideration that body parts not protected by the apron, e.g. hands and arms and the head and neck region, are exposed unshielded to the scattered radiation.

As the radiation field is quite inhomogenious, and the yearly limit of different regions is not the same, it is clear, however, that a single film-dosemeter worn under the apron at the chest level appears a rather inadequate tool for the determination of actually received doses.[1,3]

Buchan found the attenuation factor for the protective apron to be 0.04 – 0.005, i.e., areas left unprotected are exposed to 25-200 times higher doses compared to the areas under the apron.[1] These data are clearly confirmed by our observations. Since the dose measured under the apron at the chest level is only about one hunredth or less of that of the unprotected regions, it is easy to realize that the use of a film-dosemeter is totally insufficient.

E.g., if on the radiologist's bimonthly controlled dosemeter the value is under 0.4 mGy and the numerical value though not given is actually 0.3 mGy, the lens received in the same period at least 42 mGy, even if the radiologist performs only vascular interventions which are much more favourable from radiohygienic point of view. If we extrapolate this for a whole year, we get 252 mGy, a value above the annual radiation limit for the lens (150 mGy).

Taking into account the extreme situation when the radiologist's whole body dose is 0.39 mGy for two months during the whole year, he/she is notified that his/her dose is below the 0.4 mGy bimonthly limit. He/she is lulled into a false sense of security, but the received dose (6 × 0.39 mGy) 2.34 mGy, although being only a minute portion of the wole body maximal permissible dose (50 mGy), means 330 mGy for the lens, 360 mGy for the jugulum and 414 mGy for the left hand, provided that the radiologist performs only vascular interventions.

If the interventional radiologist has to perform also PTBDs which are much more unfavourable from radiohygienic point of view, his/her dose measured under the apron will be significantly increased mainly because he/she has to be in the closest proximity of the radiation beam during all the interventions. Moreover, the PTBDs usually require more lenghty manipulation compared to vascular interventions.

During PTBDs the under-the-apron value at the chest level increases considerably (the dose rate is 6.5 times higher than in vascular interventions) and so the relative values expressing the relationship between the doses at the chest level and at the forehead and thyroid of the radiologist are numerically smaller, the relevant dose rates are still higher, compared to those measured during VIs.

One has to pay special attention to the fact that some kinds of interventions such as PTBDs and percutaneous nephrostomies require much longer fluoroscopy time, and the radiologist's both hands specially the left one are exposed to an extremely high dose of scattered radiation. From our data it is clear that the radiologist reaches his/her yearly dose equivalent limit to his/her left hand in 656 min. (10.9 hours) which may cover lengthy manipulations needed for 10-12 patients' care. Self-evidently, in the given year the radiologist should not perform any other interventions.

Though during PTBDs significant doses to the uncovered parts of the radiologist remain unregistered, yet – since the under-the-apron values are considerably higher –, the whole body dose monitored by a film-dosemeter will nevertheless reach the 0.4 mGy limit, and therefore, the not negligible amount of under-the-apron dose will not go numerically undetermined at least.

Unfortunately, we have to use an old equipment with an overhead tube for interventional radiology. In certain regions (e.g. forehead and the lens) an overhead x-ray tube increases the scattered radiation level by 30 times compared to the undercouch position of the same tube.[4] Even our extremely high dose rates make it possible to draw a conclusion regarding to whether a single film-dosimeter worn under the apron is an efficient and reliable tool for estimating the whole body dose equivalent. Though, using an undercouch x-ray tube, the radiologist's hands are exposed to an amount of scattered radiation dose which is by one order of magnitude lesser than that registered with the use of an overhead tube,[5,6] this radiation burden still remains dangerous, entailing the risk of exceeding the yearly dose limit for uncovered parts of the radiologist.

As we have stated earlier, equipment with an overhead tube is not suitable and should not be used for performing interventional radiology procedures.[2] Where the expected doses are low, it is recommended that a dosimeter under the apron be worn; this should be combined with one or more additional dosimeters on the unprotected parts of the body when higher doses are expected.[1]

A value for effective dose equivalent can not be properly derived by wearing a single dosimeter either under the apron or outside it (e.g. at collar level, suggested by Jones[7]). It is advisable to wear one or more additional monitors (and/or dose-rate meters) when an occupationally exposed person works under unusual exposure conditions due to uneven doses such as may occur in interventional radiology. Even in the case of ideal radioprotective conditions (x-ray pulse generator, undercouch tube) the use of individual radioprotective devices (goggles, gloves) have not yet become unnecessary.[8]

References

1. BIR. Recommended position for wearing of personal dosemeters. Statement of the British Institute of Radiology *Brit J Radiol* 1988; **61:** 349-50.
2. Kónya A, Vigváry Z. Personnel exposure to radiation at biliary interventional radiological procedures with an overhead tube. *Radiol Oncol* 1992; **26:** 150-6.
3. Law J. Doses to head and arms of radiologist during fluoroscopy (Short communication). *Brit J Radiol* 1985; **58:** 187-8.
4. Hoffman JR, Staiger JW, Wollan RO et al. The Minnesota Special procedure room. *Radiology* 1971; **98:** 551-9.

5. Geterud K, Larsson A. Radiation dose to patients and personnel during fluoroscopy at percutaneous renal stone extraction. *Acta Radiologica* 1989; **30:** 201-6.

6. Gustaffson M, Lunderquist A. Personnel exposure to radiation at some angiographic procedures. *Radiology* 1981; **140:** 807-11.

7. Jones JR. Monitoring of staff wearing lead-rubber aprons. *Brit J Radiol* 1986; **59:** 1051-3.

8. Kónya A, Vigváry Z. The role of flexible protecting gloves in some interventional radiology procedures. Journal of Interventional Radiology (submitted for publication).

Radiol Oncol 1994; 28: 63.

Book review

Imaging of bone tumors

By Morrie E. Kricum, M. D., Profesor of Radiology, Department of Radiology, University of Pennsylvania Shool of Medicine, Philadelphia, Pennsylvania: Saunders Company Philadelphia, 1993

The objective of this text, as stated in the original preface, is to provide a comprehensive presentation of bone tumors and the methods of imaging.

The seven chapters of Part I, Conventional Radiography, written by the highly competent radiologist Dr. Kricum do full justice to the important role played by medical imaging in the diagnosis, staging and follow-up of patients afflicted with bone and soft tissue tumors. In Chapter 1 the various parameters of plain film diagnosis are reviewed together with a discussion of the pathophysiology and differential diagnosis of the radiographic signs of bone lesions.

The second chapter, Tumors of Long Bones renders a full account of the radiographic, pathologic and pathophysiologic aspects of the tumors. Furthermore, clinical features including the age of onset, incidence and local patterns of tumors are dealt with. In chapters 3 through 7 a thorough analysis is made of tumors located on different anatomic structures inlcuding the hands, feet, spine, ribs and pelvis. These chapters have been written mainly with a view to presenting a radiographic approach that in some cases differs from the radiographic approach to long bone tumors. A further reason for creation of these chapters is the great degree of subspecialization in the field of orthopedic hand and spine surgery. Nor should the extensive reference list for further research remain unnoted.

The chapters in Part II are technique oriented and analyse the value of magnetic resonance imaging, computed tomography, radionuclide imaging, angiography, and sonography, in the staging of tumors and their importance in treatment planning. CT and MRI are the most efficacious methods for assessing the intracompartmental and extracompartmental extent of the lesion. Although MRI is superior to CT in demonstrating the intramedullary extent of a lesion while the latter is better in the evaluation of cortical destruction, calcification, ossification and endosteal or periosteal reaction. Angiography can still be useful in patient management. It remains an important tool when CT and MRI do not show clearly the relationship of a musculoskeletal tumor to the major neurovascular bundle. Angiography has become increasingly important for therapeutic embolisation and intraarterial chemotherapy.

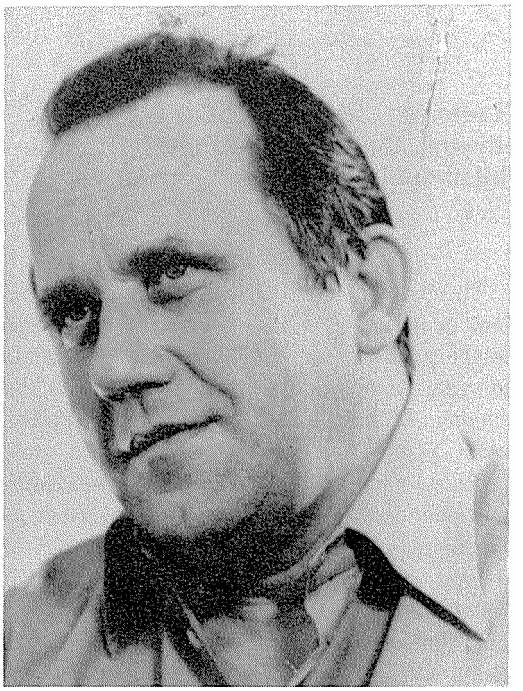
The two Chapters in Part III. deal with the pathologist's and the surgeon's perspectives on their role and on the role of imaging for patient with bone tumor.

I found this book an excellent and easily readable sours of information on diagnosis and staging of bone tumors. I would recommend it to every radiologist, oncologist and orthopedist that has to do with bone tumors.

Breda Jančar, M.D.
Institute of Oncology,
Ljubljana, Slovenia

Radiol Oncol 1994; 28: 64-6.

On the 70th birthday of Professor Dr. Ludvik Tabor



Professor Dr Ludvik Tabor a distinguished radilogist who recently celebrated his 70th birthday, has made an important contribution to the development of Slovenian radiology over a period of almost four decades. Born in Ljubljana, where in 1951 he obtained his M. D. at the Medical Faculty, he early decided to specialise in radiology, this somewhat less attractive area, and passed his Board specialist Exam in 1957. His academic interests became apparent soon after moving to the Radiology Institute at the Clinical Hospitals of Ljubljana when his first professional articles were published at home and abroad. In 1964 he was appointed Assistant Professor of radiology at the Medical Faculty of Ljubljana. His thesis on "Genital Tuberculosis in Women and the Problem of Modern Radiological Diagnostics" was used for years by radiologists as an important textbook in their preparation for B Exams. In 1970, he successfully defended his doctoral thesis entitled "Radiological Clinical Study of Congenital Anomalies of the Spine and Potential Disability", and he was the first to obtain a D. Sc. degree in radiology of Slovenia. He remained faithful to osteoarticular radiology to the end of his professional carrer. In 1974 he was elected an Associate Professor, and in 1978 became a Professor of radiology at the Medical Faculty of Ljubljana. In the academic years 1977/78 and 1978/79 he was Deputy Dean and for 13 years – from 1981 until his retirement – head of the Chair of Radiology. As a teacher and lecturer, he shared his knowledge with many generations of medical and stomatology students as well as students at the Department for Radiographers at the Medical Workers College in Ljubljana. Also important was his educational and tutorial work with specialists in radiology, particulary in the osteoarticular field.

He continued his education at a number of foreign institutions: the Cochin Hospital in Paris, the Orthopaedic Clinic of St. Gallen Cantonal Hospital, Institute of Radiology at the Zürich Cantonal Hospital and the Institutes for Radiology of Bonn and Tübingen.

Dr Tabor focused his professional interest on radiologycal diagnosticst of osteoarticular diseases, particularly in the area of orthopaedics, haematology, osteoarticular injuries, and radiology in gynaecology and stomatology. Some of his most important research works are in the field of radiological diagnostics of genital tuberculosis in women (his assistant professor thesis), research of congenital anomalies of the spine and potential disability (doctoral thesis), research on the injuries to the axial skeleton and the possibilities of modern radiological diagnostics, and research on the importance of limphography in gynaecology. His extensive biblio-

graphy includes 131 professional articles published in national and foreign journals, and 14 scientific research works. He has published three books independently, and further two as a co-author an organiser and lecturer he participated at many congresses and meetings of radiologists, orthopaedists and traumatologists in Slovenia as well as in numerous European and international centres.

He has been a permanent associate in the field of archeology at the National Museum of Ljubljana and the Regional Museum of Koper since 1970. He conducted three studies: "Anthropological, Anatomical and Pathoanatomical Representation of Skeletons From Burial Chambers in the Original Parish of St Lawrence Church on Monte di Buja – an Attempt to Present the Problem" (1986), "Anthropological, Anatomical and Pathoanatomical Representation of Skeletons from Burial Chambers in the Vicinity of Udine Castle" (1988) and "Anatomical and Pathoanatomical Study of Skeletons from Graves and Burial Chambers in the Former Parish of St Clare's Church in Koper" (1991).

Dr Tabor actively participated in the founding and all phases of development of the Institute for Radiology of the Clinical Centre in Ljubljana, the leading institution of its kind in the country. In 1976, together with Italian colleagues, he helped to establish the Alps-Adriatic community which resulted in regular annual meetings of radiologists from Italy, Austria and Slovenia. He was a co-founder, and from 1976 to 1981, Editor-in-chief of the journal RADIOLOGIA IUGOSLAVICA. Between 1958–1969 he presided over the Section for Radiology and Nuclear Medicine at the Slovenian Medical Society. For many years he was secretary-General of the Yugoslav Association of Radiology and Nuclear Medicine.

He was a member of the International Society of Limphography, the European and International Association of Radiologists, Slovenian Medical Society, Radiology Department at the Slovenian Medical Society, Yugoslav Association of Radiologists and the Yugoslav Association of Orthopaedists and Traumatologists. He is an honorary member of the Yugoslav Society of Senior Radiographers.

He has received many awards for his work; a Golden Plaque from the Yugoslav Association of Medical Societies (1971), an award from the Medical Faculty of Ljubljana (1978), a Silver Plaque form the Yugoslav Association of Orthopaedists and Traumatologists (1981), the Order of Labour with Gold Wreath (1980), a Gold Plaque from the Yugoslav Association of Orthopaedists and Traumatologists (1981), a Plaque from the Yugoslav Association of Radiologists (1988) and an Award from the Orthopaedic Clinic (1993).

No description of the profilic life and work of Ludvik Tabor could be complete without mentioning his painting, which has ben been gaining in importance by the time passing. He has exhibited a part of his extensive colleciton of water colours in 23 independent and 30 group exhibitions. With all probability, it is the painting that has enabled him to sucessfully escape the constraints inevitably imposed by a successful career in medicine and enter a world of absolute freedom, to which he often refers by saying that the only profession that ensures absolute independence is that of a simple farmer; as if seeking in the colours of nature and his watercolors a substitute for the years of life spent among the grey and black of the Radiological profession. Dr Tabor's attitude towards nature and art is perhaps most faithfully reflected in the following statements of his: "Art is an unlimited record of the expansiveness of spiritual comprehension and a feeling for everything that nature unselfishly offers. What could be more inviting than a record of the Credo of life to Nature, records of the colouring of the light and shade of the personally experienced and witnessed, a record of an ever-present fortissimo, crescendo and pianissimo in the rhythm of nature. In the face of today's consumer-oriented, trying to find happiness amassing material goods and ephemeral pleasures and incapable of experiencing inner peace, the only escape left to those still capable of thinking and acting differently is to run for the cover of what nature can still provide. Some wonder at this,

unable to understand why more and more people from all walks of life dedicate their free time to the fine and other arts. The answer is simple: Given what modern civilisation of tihs angst-ridden world offers or simply forces upon us, the arts offer a sanctuary where one can recover and rebuilds one's shattered spirituality. The arts and nature are a source of opportunities, a gift to prevent man from simply surrendering and drowning in the time we live in."

When he decided to retire in 1993, Ludvik Tabor had many plans for the future. First of all, to compile his vast professional experience and condense it in a book entitled "Where, When and How in Radiological Skeletal Diagnostics". He wanted to compile in one place the answers to the key questions constantly facing radiologists. The illness he has learnt to live with and control has unfortunately prevented him, as yet, from achieving this goal. Slovenian radiologists would like to convey their most warmest congratulations to Ludvik Tabor on the occarion of his 70th birthday, and wish him to retain his inspiration and life energy.

Prof. Dr Vladimir Jevtić

Notices

Notices submitted for publication should contain a mailing address, phone and/or fax number of a contact person or department.

Science editors

The European Association of Science Editors (EASE) conference will be held in Budapest, Hungary, *April 24–28, 1994.*

Contact Secretary-Treasurer Ms. Maeve O'Connor, 49 Rossendale Way, London, NWL, OXB, United Kingdom; or call + 44 71 388 9668. Fax: + 44 71 383 3092.

Ecosystem health & medicine

The 1st International Symposium on Ecosystem Health & Medicine vill be offered in Ottava, Ontario, Canada, *June 19–23, 1994.*

Contact Office of Continuing Ecucation, 159 Johnston Hall, University of Guelph, Guelph, Ontario, Canada, N1G 2Wl; or call + 519 767 5000. Fax: + 1 519 767 0785.

Diagnostic radiology

Seminar "The Initiation to Neuro–imaging and Endovasuclar Radiology" will be held in Marseille, France, *July 9–12, 1994.*

Contact ECR-Office, European Congress of Radiology, Neutorgasse 9/2a, A-1010 Vienna, Austria; or call + 43 1 533 40 64. Fax: + 43 1 533 40 649.

IAEA Scientific meeting

The interregional seminar on isotope techniques in arid and semi-arid land hydrology will be offered in Vienna, Austria, *August 15–26, 1994.*

Contct International Atomic Energy Agency, P. O. Box 100, Vienna International Centre, A-1400 Vienna, Austria.

Nuclear medicine

The European Congress of The European Association of Nuclear Medicine will be held in Dusseldorf, Germany, *August 20–24, 1994.*

Contact Die Kongress–Partner, Eberhardt–Gastell & Neumann GmbH, Bottenhorner Weg 16, D-60489 Frankfurt/Main, Germany; or call + 49 69 785 050. Fax: + 49 69 785 049.

Oncology

The international meeting "Growth Control and Therapy of Cancer" will take place in Convention Center, Budapest, Hungary, *August 21–24, 1994.*

Contact GCTC '94, P. O. Box 6, CH-4005 Basel, Switzerland; or call + 41 61 691 51 11. Fax: + 41 61 691 81 89.

Medical physics and biomedical engineering

The "10th International Conference on Medical Physics" and the "17th International Conference on Medical and Biomedical Engineering will be held in Rio de Janeiro, RJ CEP 20040, Brazil, *August 21–26, 1994.*

Contact Secretariat, Congress Brazil do Ouridor 0/414, Rio de Janeiro, RJ CEP 20040, Brazil; or call + 55 21 224 6080.

IAEA Scientific meeting

The interregional seminar on radiotherapy dosimetry: radiation dose in radiotherapy from prescription to delivery will be offered in Rio de Janeiro, Brazil, *August 27–30, 1994.*

Contact International Atomic Energy Agency, P.O. Box 100, Vienna Intenational Centre, A–1400 Vienna, Austria.

Radiotherapy

ESTRO teaching course "Radiation Physics for Clinical Radiotherapy" wil be offered in Leuven, Belgium, *September 4–8, 1994.*

Contact the Estro Secretariat, Radiotherapy Department, University Hospital St Rafael, B-3000 Leuven, Belgium; or call + 32 16 33 64 13. Fax: + 32 16 33 64 28.

IAEA Scientific meeting

The 2nd IAEA/FAO seminar for Africa on animal Trypanososmiasis: Vector and disease control using nuclear techniques will be offered in Uganda, *September, 1994.*

Contact International Atomic Energy Agency, P. O. Box 100, Vienna International Centre, A-1400 Vienna, Austria.

Sarcomas

The ESO teaching course will be held in *September 5–7, 1994.*

Contact Miss Gollubics, ESO-Vienna-Office, Arztekammer fur Wien, Fortbildungsreferat Weihburggasse 10-12, A-1010 Vienna, Austria; or call + 43 1 51501 293. Fax: + 43 1 51501 240.

IAEA Scienfific meeting

The conference on nuclear power option will be held in Vienna, Austria, *September 5–9, 1994.*

Contact International Atomic Energy Agency, P. O. Box 100, Vienna Interntional Centre, A-1400 Vienna, Austria.

Breast cancer

The "6th EORTC Breast Cancer Working Conference" will take place in Amsterdam, The Netherlands, *September 6–9, 1994.*

Contact Bureau PAOG Amsterdam, Tafelbergweg 25, 1105 BC Amsterdam, The Netherlands; or call + 31 20 556 4801. Fax: + 31 20 696 3228.

Medical oncology

The course on molecular bilogoy for clinical oncologists will be held in Ascona, Switzerland, *September 11–15, 1994.*

Contact ESMO Central Secretariat, Via Soldino 22, 6903 Lugano, Switzerland; or call + 41 91 575 411. Fax: + 41 91 575 744.

Uroradiology

The 4th European symposium on uroradiology will be held in Florence, Italy. *September 12–15, 1994.*

Contact Studio Congressi, Sistiana 59/M, I–34019 Trieste, Italy; or call + 39 402 91384.

Oncology

The 22nd international meeting "Basic Research and Clinical Application in Human Tumor Immunology and Molecular Biology" will be held in Groningen, The Netherlands, *September 18–22, 1994.*

Contact Dr. Henk W. A. de Bruijn, General Secretary ISOBM '94, Laboratory for Obstetrics and Gynaecology, University Hospital, Oostersingel 59, 9713 EZ Groningen, The Netherlands; or call + 31 50 614 337. Fax: + 31 50 613 474.

Radiotherapy

The ESTRO pre-meeting teaching course "The use of modern diagnostic imaging techniques in radiotherapy planing" will be offered in Granada, Spain, at *September 22–24, 1994.*

Contact the ESTRO Secretariat – University Hospital St. Rafael, Radiotherapy Department, Capucijnenvoer 35, 3000 Leuven, Belgium; or call + 32 16 33 64 13. Fax: + 32 16 33 64 28.

Radiology

Seminar "The Radiology of Gastrointestinal Tract Neoplasms" will be held in Iraklion, Crete, Greece, *September 22–24, 1994.*

Contact ECR-Office, European Congress of Radiology, Neutorgasse 9/a, A-1010 Vienna, Austria; or call + 43 1 533 40 64. Fax: + 43 1 533 40 649.

Radiotherapy

The second ESTRO postgraduate teaching course for radiotherapy technologists will be offered in Granada, Spain, at *September 22–24, 1994.*

Contact the ESTRO Secretariat – University Hospital St. Fafael, Radiotherapy Department, Capucijnenvoer 35, 3000 Leuven, Belgium; or call + 32 16 33 64 13. Fax: + 32 16 33 64 28.

FIGO 1994

The 14th world congress of gynecology and obstetrics will be held in Montreal, Canada, *September 24–30, 1994.*

Contact Secretariat du congres FIGO 1994, 4260 Girouard, Suite 100, Montreal (Quebec), Canada H4A 3C9; or call + 1 514 485 0855. Fax: + 1 514 487 6725.

LUNG CANCER BIOLOGY AND CLINICAL ASPECTS

Under the Auspices of
International Association for the Study of Lung Cancer

13–16 April 1994

Ljubljana – Slovenia

Second announcement

Call for abstracts
& registration

Important dates

Deadline for Early Registration at reduced rate	28 February 1994
Deadline for Cancellation Requests	15 March 1994
Deadline for Guaranteed Hotel Accommodation	21 March 1994
Date of the Conference	13–16 April 1994
Opening Ceremony	13 April 1994

Organizing committee

Chairman: J. Orel
Secretary: M. Bitenc
Treasurer: M. Sok
Members: B. Hrabar, V. Kovač, T. Rott, F. Šifrer, S. Vidmar

Venue

CANKARJEV DOM
Cultural and Congress Centre,
Prešernova 10,
61000 Ljubljana, Slovenia.
Tel.: + 386 61 210 956
Fax: + 386 61 217 431

Conference organizers

CANKARJEV DOM
Congress Department,
Prešernova 10,
61000 Ljubljana, Slovenia.
Tel.: + 386 61 210 956
Fax: + 386 61 217 431

Conference secretariat

Department of Thoracic Surgery
University Medical Centre,
Zaloška 7,
61105 Ljubljana, Slovenia.
Tel.: + 386 61 317 582
Fax: + 386 61 1316 006

Croatian Medical Association
CROATIAN SOCIETY OF RADIOLOGY
Zagreb – Croatia
and
Rijeka University School of Medicine
CLINICAL INSTITUTE OF RADIOLOGY
Rijeka – Croatia

THE FIRST CONGRESS OF THE CROATIAN SOCIETY OF RADIOLOGY

Grand Hotel "ADRIATIC" Opatija – Croatia
October 11–15, 1994

SCIENTIFIC PROGRAMME

Topics:
- Diagnostic Radiology
- Interventional Radiology
- Workshops
- Posters

TECHNICAL EXHIBITION

Organizing Committee President	Croatian Society of Radiology President
(Prof. Ivo Lovasić MD, PhD)	(Prof. Slavko Šimunić MD, PhD)

Unabridged articles which should be submitted in triplicate to the Organizing Committee by the beginning of the Congress will be published in No. 4/94 of RADIOLOGY AND ONCOLOGY.

INFORMATIONS

Prof. Ivo Lovasić MD, PhD, Clinical Institute of Radiology – Clinical Hospital Center, 51000 Rijeka, Tome Strižića 3, Croatia, Phone + 38/51/44 18 99, Fax + 38/51/37 536

ECR'95 - VIENNA
9th European Congress of Radiology
NEW DATE: March 5 - 10, 1995 - Austria Center Vienna

A CENTURY OF X-RAYS 1895 - 1995

President: Prof. Dr. med. Albert L. Baert (Leuven/B)

For information please contact:

ADMINISTRATIVE AND SCIENTIFIC SECRETARIAT
European Congress of Radiology - ECR'95
Neutorgasse 9/2a
A - 1010 Vienna/Austria
Phone: (+ 43/1) 533 40 64, Facsimile: (+ 43/1) 533 40 649

TECHNICAL EXHIBITION
MAW
International Exhibitions & Advertising
Freyung 6
A - 1010 Vienna/Austria
Phone: (+ 43/1) 533 23 20, 533 21 99, Facsimile: (+ 43/1) 535 60 16

TRAVEL - ACCOMMODATION - SOCIAL PROGRAMME
MONDIAL Congress
Faulmanngasse 4
A - 1040 Vienna/Austria
Phone: (+ 43/1) 58 804-0, Facsimile: (+ 43/1) 56 91 85

Deadline for submission of scientific papers:
September 20, 1994

HEPATOBILIARY SCHOOL

LJUBLJANA, SLOVENIA
JUNE 27 - JULY 1, 1994

2nd POSTGRADUATE COURSE ON HEPATOLOGY

POSTGRADUATE COURSE ON HEPATOBILIARY SURGERY

Two courses, surgical and medical, will be held simultaneously with a number of sessions assembled. The aim of the courses is to expand practical methological and rational diagnostic knowledge as well as therapeutic approach to the most common diseases of the liver and biliary tract.

Teaching will include lectures, seminars, case presentations and workshops.

INVITED LECTURERS

Bengmark S., Boeckl O., Coggi G., Czygan P., Ferenzi P., Krejs G., Mazziotti M., Paquet K.-J., Schaffner F., Scheele J., Schmid R., Tiribelli C., Wiechel K. L., and others

SURGICAL COURSE

Liver resections
Liver trauma
Hydatid disease of the liver

SURGICAL WORKSHOP

ASSEMBLED SESSIONS

Extra hepatic biliary obstructions
Primary liver tumors
Liver transplantation

HEPATOLOGY COURSE

Viral hepatitis
Liver cirrhosis
Portal hypertension
Cholestasis

MAILING ADDRESS

E. GADŽIJEV
Medical Center
Dept. of Gastroenterologic Surgery
Zaloška 7, 61000 Ljubljana, Slovenia
tel 386 61 322 282,
fax 386 61 316 096

VENUE

University of Ljubljana,
Faculty of Medicine,
Ljubljana, Slovenia

FEES

650 USD up to March 31
850 USD up to May 31
950 USD at registration desk

MAILING ADDRESS

S. MARKOVIČ
Institute of Oncology, Building B,
Zaloška 2, 61000 Ljubljana, Slovenia
tel, fax 386 61 302 828

DEADLINE FOR REGISTRATION

May 31, 1994

LANGUAGE

English

NUMBER OF PARTICIPANTS

Limited -
30 for surgery, 50 for medicine!

2nd POSTGRADUATE COURSE ON HEPATOLOGY

POSTGRADUATE COURSE ON HEPATOBILIARY SURGERY

NAME

ADDRES

TEL FAX

❑ MEDICINE ❑ SURGERY

POSTER ❑ YES ❑ NO

HOTEL ACCOMMODATION ❑ YES ❑ NO

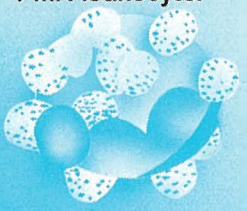
Klimicin®

(Clindamycin)

Klimicin is an effective bactericidal or bacteriostatic as evidenced by the MIC/MBC ratio.

It stimulates the action of polymorphonuclear leukocytes (PMN) principal factors in host immune system.

PMN leukocyte.



Succesful and reliable treatment for life-threatening infections

Aerobes

Streptococcus spp. (including Streptococcus pyogenes), except Enterococcus

Pneumococcus spp.
Staphylococcus spp. (including B-lactamase producing strains)

Anaerobes

Bacteroides spp. (including Bacteroides fragilis)
Fusobacterium spp.
Propionibacterium
Eubacterium
Actinomyces spp.
Peptococcus spp.
Peptostreptococcus spp.
Clostridium perfringens

Contraindications: In patients hypersensitive to lincomycin and clindamycin.

Precautions: Klimicin should be prescribed with caution to elderly patients and to individuals with a history of gastrointestinal disease, particularly colitis.

Side Effects: Gastrointestinal disturbances (abdominal pain, nausea, vomiting, diarrhea). When significant diarrhea occurs, the drug should be discontinued or continued only with close observation of the patient. The posibility of pseudo-membranous colitis must be ruled out.

lek Pharmaceutical and Chemical Company d.d. Ljubljana

Abaktal®

(pefloxacin)

tablets 400 mg
ampoules 400 mg

A new potent drug against infections

- May be given orally and parenterally
- Effecive in life-threatening infections caused by nosocomial strains resistant to many drugs
- May be given to patients hypersensitive to penicillins and cephalosporins
- Its favourable pharmacokinetic properties allow twice-a-day dosage
- Is very well tolerated

Contraindications

Pefloxacin is contraindicated in patients with known hypersensitivity to quinolones, in pregnant women, nursing mothers, children under 15 years of age, and patients with inborn glucose-6-phosphate dehydrogenase deficiency.

Precautions

During pefloxacin therapy exposure to strong sunlight should be avoided because of the risk of photosensitivity reactions. In patients with a severe liver disorder dosage of pefloxacin should be adjusted.

Side effects

Gastro-intestinal disturbances, muscle and/or connective tissue pains, photosensitivity reactions, neurologic disturbances (headache, insomnia), and thrombocytopenia (at doses of 1600 mg daily) may occur.

Dosage and administration

The average daily dosage for adults and children over 15 years of age is 800 mg.
Oral: 1 tablet twice daily after meals.
Parenteral: the content of 1 ampoule 400 mg diluted in 250 ml of 5 % glucose as a slow 1-hour infusion twice daily. The maximum daily dosage is 8 mg of pefloxacin per kg bodyweight. In severe hepatic insufficiency pefloxacin is administered only once daily (jaundice), once every 36 hours (ascites), and once every 48 hours (jaundice and ascites).

lek Pharmaceutical and Chemical Company d.d.
Ljubljana

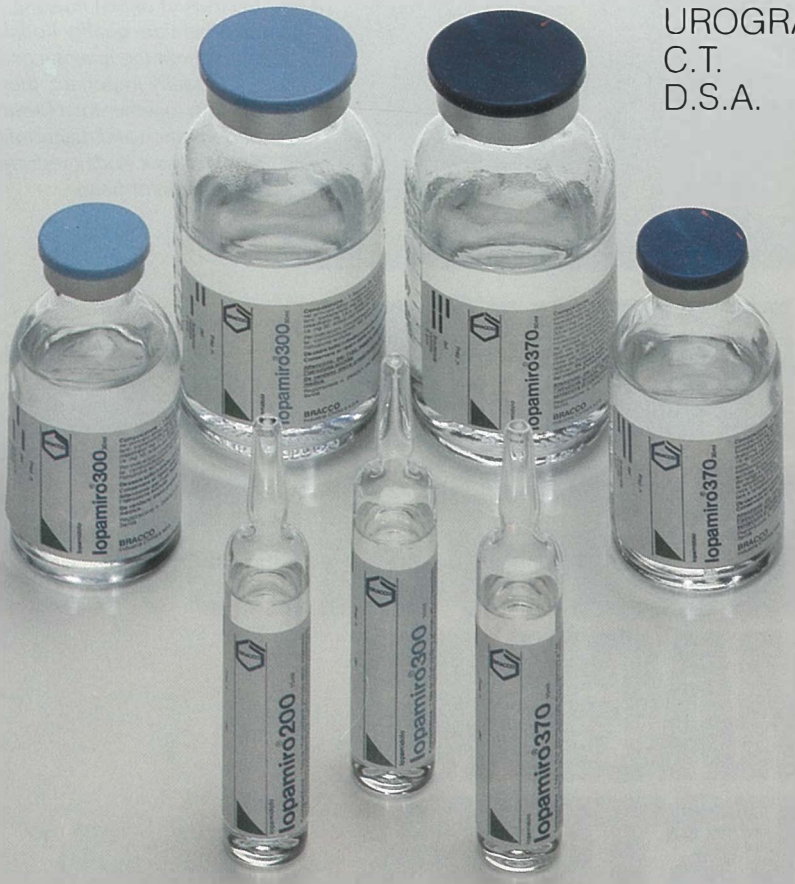
Iopamiro®

Iopamidol

150 - 200 - 300 - 370 mgI/ml

FOR ALL RADIOLOGICAL EXAMINATIONS

MYELOGRAPHY
ANGIOGRAPHY
UROGRAPHY
C.T.
D.S.A.



THE FIRST WATER SOLUBLE READY TO USE NON-IONIC CONTRAST MEDIUM

Manufacturer:

Bracco s.p.a.
Via E. Folli, 50
20134 - Milan - (I)
Fax: (02) 26410678
Telex: 311185 Bracco I
Phone: (02) 21771

BRACCO

Distributor:

Agorest s.r.l.
Via S. Michele, 334
34170 - Gorizia - (I)
Fax: (0481) 20719
Telex: 460690 AF-GO I
Phone: (0481) 21711

Kodak systems provide dependable performance for advanced diagnostic imaging. Our quality components are made to work together from exposure to viewbox.

Kodak X-Omat processors are the most respected in the field. Kodak X-Omatic cassettes are known the world over for unexcelled screen-film contact and durability. Kodak multiloaders have earned an enviable reputation for reliability. The Kodak Ektascan laser printer is changing the look of digital imaging. The list goes on. There are quality Kodak products throughout the imaging chain.

Equally important, they are made to work together to achieve remarkable performance and diagnostic quality. Contact your Kodak representative for more information.

ADVANCED DIAGNOSTIC IMAGING SYSTEMS. KODAK

Quality imaging worldwide

Kodak

POOPERATIVNA SLABOST IN BRUHANJE

NELAGODJE IN STISKA
OGROŽATA USPEH OPERATIVNEGA POSEGA

PREPREČEVANJE
Ena sama intravenska injekcija po 4 mg pri uvajanju v anestezijo

ZDRAVLJENJE
Ena sama intravenska injekcija po 4 mg

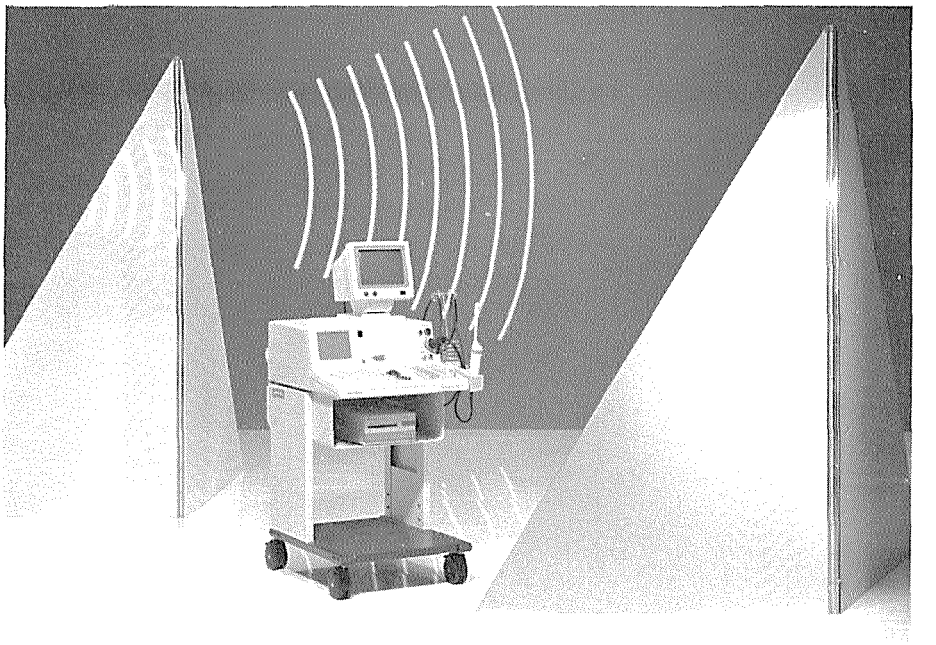
Zofran
ondansetron

Glaxo

Podrobnejše informacije dobite pri: Glaxo Export Limited
Predstavništvo Ljubljana, Tržaška cesta 132, 61111 Ljubljana
tel. (061) 272-570; fax (061) 272-569

SIEMENS

Vaš partner v ultrazvočni diagnostiki:

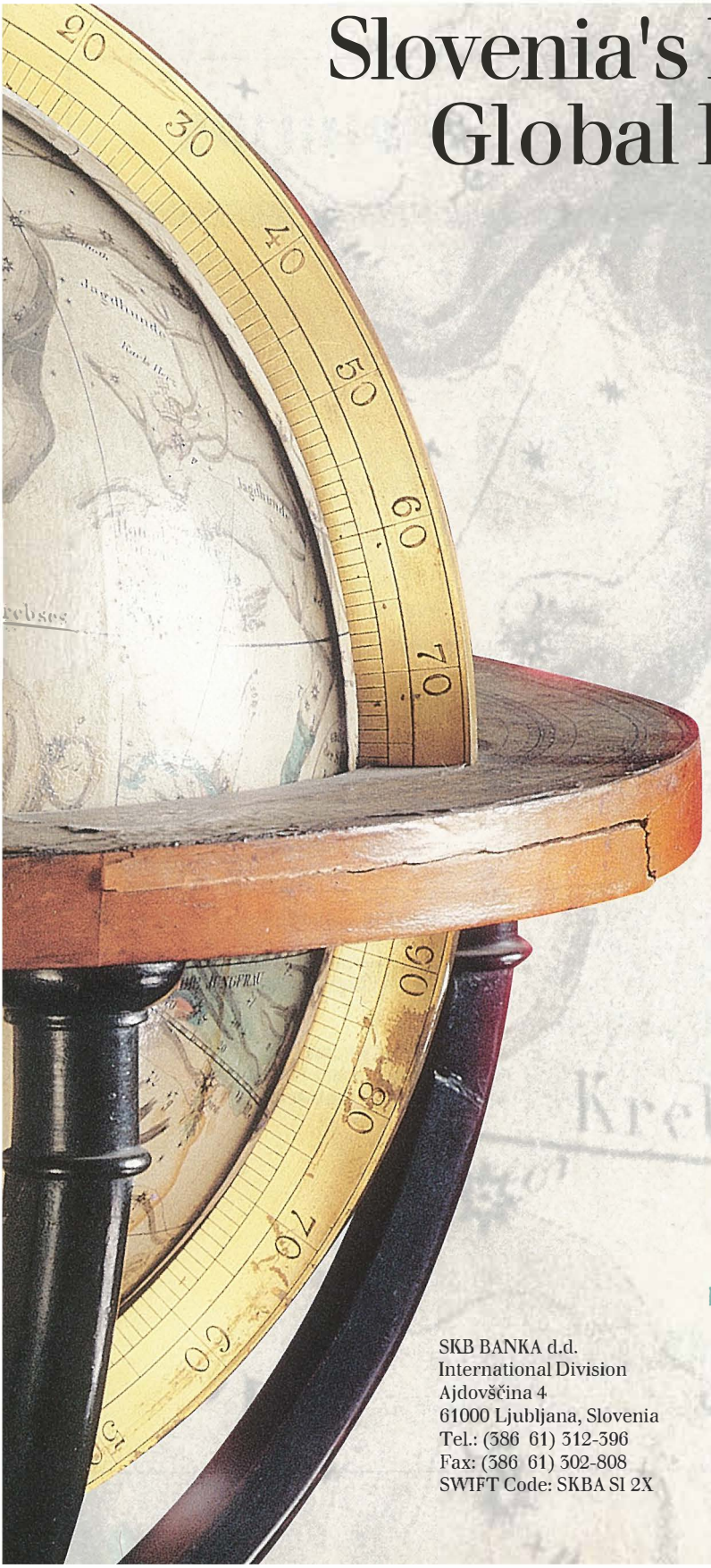


SIEMENS * SONOLINE SL-1

* **Možnost priključka sektorskega, linearnega, endo-p in endo-v aplikatorja**
* **Izredno ugodna cena (možnost kredita ali leasing-a)**
* **Servis v Sloveniji z zagotovljenimi rezervnimi deli in garancijo**
* **Izobraževanje za uporabnike**

SIEMENS D.O.O.
Dunajska 47, Ljubljana
Tel. 324-670
Fax. 132-4281

Slovenia's Bank for Global Business



SKB BANKA d.d. is an independent joint-stock company and the second largest Slovenian bank in terms of branch network and capital.

The large amount of capital on its balance sheet and its foreign currency reserves guarantee customers security and excellent prospects for further expansion.

SKB BANKA d.d. offers a wide range of banking services including:

- International payments and account services
- Non-resident account services, private and corporate
- Trade finance services
- International finance services
- Treasury and foreign exchange services
- Securities services
- Real estate financing, trading and renting

SKB BANKA d.d. has a network of first-class foreign correspondents on all five continents.

The range of services offered by the bank, is complemented by the services offered through its three bank-owned companies:
SKB Real Estate and Leasing Ltd.,
SKB Investment Company Ltd.,
SKB Aurum- Trade in precious metals, works of art, antiques and safekeeping of valuables and securities Ltd.

SKB BANKA D.D.

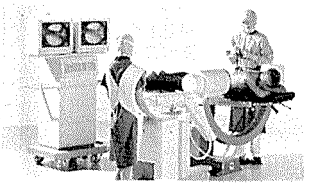
SKB BANKA d.d.
International Division
Ajdovščina 4
61000 Ljubljana, Slovenia
Tel.: (386 61) 312-396
Fax: (386 61) 302-808
SWIFT Code: SKBA SI 2X

SKB BANKA D.D.
Representative Office London,
37-39 Eastcheap,
London EC3M 1 DT,
United Kingdom
tel.: (44 71) 929-2174,
fax: (44 71) 929-2175

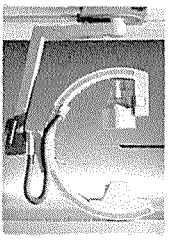
PHILIPS

PHILIPS

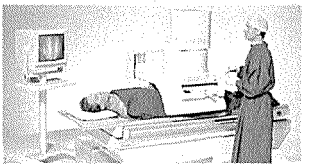
Philips Medical Systems



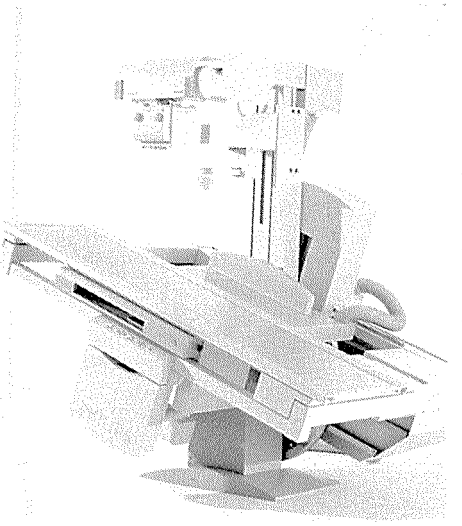
BV 29



INTEGRIS C 2000



DIAGNOST 76 +



DIAGNOST 93

NOV ČLAN DRUŽINE PHILIPSOVIH TELEDIRIGIRANIH RENTGENSKIH STATIVOV, KI JIH ODLIKUJE KVALITETNA MODULARNA IZDELAVA, VISOKA ZANESLJIVOST, ENOSTAVNA UPORABA IN IZREDNA KVALITETA SLIKE.

APARAT JE MOGOČE OPREMITI S SISTEMOM ZA DIGITALNO RADIOGRAFIJO PREKO SVETLOSTNEGA OJAČEVALNIKA (DSI), KI OLAJŠA DELO UPORABNIKOM, MANJŠA DOZE, NIŽA STROŠKE IN KRAJŠA PREISKAVE.

ZA VSE DODATNE INFORMACIJE SE OBRNITE NA ZASTOPNIKA PHILIPS MEDICAL SYSTEMS V SLOVENIJI:

skupina
avtotehna d.d.

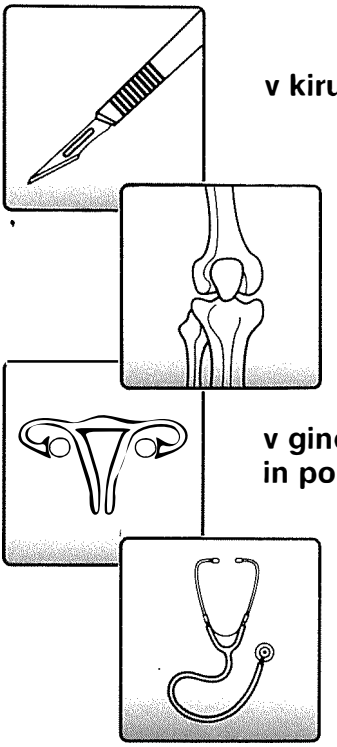
AVTOTEHNA d.d. Ljubljana, Slovenska 54, tel.: (061) 320 767, faks: (061) 322 377

NOVO

Tramal®

tramadol HCl

~~zanesljiv analgetik s~~ centralnim delovanjem



v kirurgiji

v travmatologiji in ortopediji

v ginekologiji in porodništvu

v interni medicini

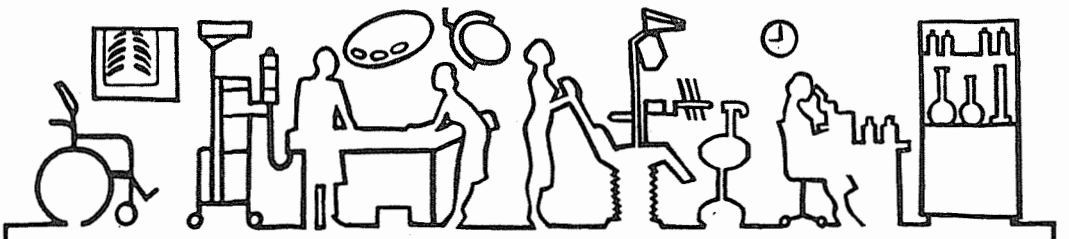
Oblike: Tramal 50: 5 ampul po 50 mg tramadola/ml
Tramal 100: 5 ampul po 100 mg tramadola/2 ml
Tramal kapljice: 10 ml raztopine (100 mg tramadola /ml)
Tramal kapsule: 20 kapsul po 50 mg tramadola
Tramal svečke: 5 svečk po 100 mg tramadola

Izdeluje: Bayer Pharma d.o.o. Ljubljana
po licenci Grünenthal GmbH

GRUNENTHAL

SANOLABOR

za ljudi v belem



Pri nas dobite vse za rentgen!

▫ KODAK ▫ AGFA GEVAERT ▫ POLAROID ▫ 3M ▫ PHILIPS ▫
▫ GENERAL ELECTRIC ▫ SIEMENS ▫ TOSHIBA ▫ HITACHI ▫
▫ NICHOLAS ▫ BYK GULDEN ▫ MAVIG ▫ CAWO ▫

- rentgenski filmi in kemikalije
- kontrastna sredstva
- rentgenska zaščitna sredstva
- rentgenski aparati, aparati za ultrazvočno diagnostiko, stroji za avtomatsko razvijanje in druga oprema za rentgen

SANOLABOR Cigaletova 9, 61000 LJUBLJANA
☎ 061/1333231 FAX: 061/325 - 395

Instructions to anthors

The journal **Radiology and Oncology** publishes original scientific papers, professional papers, review articles, case reports and varia (reviews, short communications, professional information, ect.) pertinent to diagnostic and interventional radiology, computerised tomography, magnetic resonance, nuclear medicine, radiotherapy, clinical and experimental oncology, radiobiology, radiophysics and radiation protection.

Submission of manuscript to Editorial Board implies that the paper has not been published or submitted for publication elsewhere: the authors are responsible for all statements in their papers. Accepted articles become the property of the journal and therefore cannot be published elsewhere without written permission from the Editorial Board.

Manuscripts written in English should be sent to the Editorial Office: Radiology and Oncology, Institute of Oncology, Vrazov trg 4, 61000 Ljubljana, Slovenia; Phone: +386 61 1320 068, Fax: +386 61 1314 180.

Radiology and Oncology will consider manuscripts prepared according to the Vancouver Agreement (N Engl J Med 1991; 324: 424-8.; BMJ 1991; 302: 6772.).

All articles are subjected to editorial review and review by two independent referees selected by the Editorial Board. Manuscripts which do not comply with the technical requirements stated here will be returned to the authors for correction before the review of the referees. Rejected manuscripts are generally returned to authors, however, the journal cannot be held responsible for their loss. The Editorial Board reserves the right to require from the authors to make appropriate changes in the content as well as grammatical and stylistic corrections when necessary. The expenses of additional editorial work and requests for reprints will be charged to the authors.

General instructions: Type the manuscript double spaced on one side with a 4 cm margin at the top and left hand side of the sheet. Write the paper in grammatically and stylistically correct language. Avoid abbreviations unless previously explained. The technical data should confirm to the SI system. The manuscript, including the references may not exceed 15 typewritten pages, and the number of figures and tables is limited to 4. If appropriate, organise the text so that it includes: Introduction, Material and methods, Results and Discussion. Exceptionally, the results and discussion can be combined in a single section. Start each section on a new page and number these consecutively with Arabic numerals. Authors are encouraged to submit their contributions besides three typewritten copies also on diskettes (5 1/4") in standard ASCII format.

First page:

- name and family name of all authors,
- a brief and specific title avoiding abbreviations and colloquialisms,
- complete address of institution for each author,
- in the abstract of not more than 200 words cover the main factual points of the article, and illustrate them with the most relevant data, so that the reader may quickly obtain a general view of the material.

Introduction is a brief and concise section stating the purpose of the article in relation to other already published papers on the same subjects. Do not present extensive reviews of the literature.

Material and methods should provide enough information to enable experiments to be repeated.

Write the **Results** clearly and concisely and avoid repeating the data in the tables and figures.

Discussion should explain the results, and not simply repeat them, interpret their significance and draw conclusions.

Graphic material (figures and tables). Each item should be sent in triplicate, one of them marked original for publication. Only high–contrast glossy prints will be accepted. Line drawings, graphs and charts should be done professionally in Indian ink. All lettering must be legible after reduction to column size. In photographs mask the identities of patients. Label the figures in pencil on the back indicating author's name, the first few words of the title and figure number: indicate the top with and arrow. Write legend to figures and illustrations on a separate sheet of paper. Omit vertical lines in tables and write the next to tables overhead. Label the tables on their reverse side.

References should be taped in accordance with Vancouver style, double spaced on a separate sheet of paper. Number the references in the order in which they appear in the text and quote their corresponding numbers in the text. Following are some examples of references from articles, books and book chapters:

1. Dent RG, Cole P. *In vitro* maturation of monocytes in squamous carcinoma of the lung. *Br J Cancer* 1981; **43:** 486-95.
2. Chapman S, Nakielny R. *A guide to radiological procedures.* London: Bailliere Tindall, 1986.
3. Evans R, Alexander P. Mechanisms of extracellular killing of nucleated mammalian cells by macrophages. In: Nelson DS ed. *Immunobiology of macrophage.* New York: Academic Press, 1976: 45-74.

No pain with...

TADOL®

capsules, drops, suppositories, injection

tramadol

- **centrally acting analgesic for the alleviation of moderate to severe pain**
- **efficacy and relatively few adverse reactions**

Indications: Moderate to severe acute and chronic pain. **Contraindications:** The drug should not be given to children younger than 1 year. Tramadol is contraindicated in the event of acute intoxication with alcohol, hypnotics, analgesics, and other drugs acting on CNS. Tramadol should be given to a pregnant woman only if clearly needed. In treatment during lactation, the fact that 0.1 % of the drug is excreted in human milk should be noted. Tramadol should be used with caution in patients with increased reactivity to opioids. Careful observation is necessary during the treatment of patients with central convulsions. **Interactions:** Tramadol should not be used concomitantly with MAO inhibitors. Concomitant administration of tramadol and other drugs that act on the central nervous system may produce a synergistic effect resulting in enhanced sedation and a more favourable analgesic effect. **Warnings:** Overdosage may result in respiratory depression. Caution is advised in patients with known hypersensitivity to opioids, in elderly patients, and those with myxedema and hyperthyroidism. In impaired liver or kidney function the dosage should be reduced. Patients should be warned against driving a car or operating machinery during the treatment. **Dosage and administration:** *Adults and children over 14 years of age:* 50 — 100 mg i.v., i.m., s.c.; intravenous injection should be given slowly or diluted in an infusion. Capsules: 1 capsule with little liquid; drops: 20 drops with little liquid or on a sugar cube — if the effect is not satisfactory the dose should be repeated after 30 to 60 minutes. Suppositories: 1 suppository, if the effect is not achieved, the dose should be repeated after 3 to 5 hours. *Children from 1 to 14 years of age:* 1 to 2 mg/kg body weight. The daily dose of 400 mg given in whatever formulation need not to be exceeded. **Side effects:** sweating, dizziness, nausea, vomiting, dry mouth, and fatigue. Rarely, palpitation, orthostatic hypotension, or cardiovascular collapse may occur. Only occasionally convulsions may appear. **Supply:** 5 ampoules of 1 ml (50 mg/ml), 10 ml of solution (100 mg/ml), 20 capsules of 50 mg, 5 suppositories of 100 mg.

For further information please contact the manufacturer.

KRKA
SLOVENIA