

**VHe 16**

CHANGES IN PERIPHERAL LYMPH NODES AS SEEN VIA ULTRASOUND  
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Sonographic examinations of 2346 patients performed between 1980 and 1985 with particular reference to the inguinal, axillary and neck regions have shown that it is quite possible to effect sonographic differentiation between enlarged lymph nodes with cicatricial indurations after non-specific inflammation on the one hand, and primary lymphomas or metastases of lymph nodes on the other. In many cases, it is also possible to differentiate between the enlargement of a total lymph node (acute inflammatory, primary malignant) and metastases in a lymph node. Positive findings in the soft parts were seen in 379 patients; of these, 236 were confirmed histologically and cytologically (via puncture with sonographic control). One finding was revealed as false positive. In 132 patients the sonographic finding was confirmed by means of lymphographies (n = 15), computed tomography (n = 46) or via the subsequent clinical course, proving the fact that it is also possible to detect sonographically the ability of lymphomas to respond to treatment with antibiotics, cytostatics, and radiation; the effects can be recognised and interpreted via changes of the echo patterns.

Using high resolution real-time scanners with near focus capability it is possible to differentiate lymphnode metastases of 0,3 cm diameter from surrounding soft tissue. They appear as round, nearly echofree structures, sometimes with far-wall echoes. The not yet affected peripheral areas of the nodes are in general not visualized. Post-inflammatory lymphnode enlargement can also not be demonstrated. Primaries or inflammatory lymphomas are as echolucent as metastases, not round but bean-shaped, as in these cases the whole lymphnode becomes demonstrable. Under chemo or radiation therapy the lesions become echogenic within a few days as in a hint of sufficient therapy.

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**VHe 17**

SONOMORPHOLOGY OF FOCAL LESIONS OF THE LIVER AND SPLEEN IN HODGKIN AND NONHODGKIN LYMPHOMA  
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The sonographic patterns of focal lesions of the liver and spleen in 26 patients with Hodgkin and nonHodgkin disease are described. 13 out of 19 hepatic lesions and 9 out of 11 splenic lesions corresponded to a nodular lymphomatous involvement. The remaining cases involved benign focal lesions of the liver and spleen. The vast majority of the hepatic and splenic lymphomatous nodules had an hypoechoic pattern with indistinct edge contours. Target lesions of the liver were only seen in nonHodgkin lymphoma. Echogenic lymphomatous nodules could not be detected. All echogenic lesions (n = 3) were biopsied and turned out to be benign. Whereas a focal liver involvement almost always occurred in combination with a hepatomegaly, only 2 out of 9 patients with splenic foci had a splenomegaly. The response of hepatic and splenic lymphoma to chemotherapy is described by follow-up examinations in 11 cases. Because of the inherent risk of overtreatment due to false-positive interpretation of hepatic nodules, the large-scale use of needle biopsy in sonographically equivocal lesions is highly advocated.

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**VHe 18**

THYMIC HYPERPLASIA FOLLOWING ANTINEOPLASTIC CHEMOTHERAPY IN ADULT CANCER PATIENTS  
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Up till now only five cases of thymic hyperplasia following chemotherapy in adult cancer patients (four with Hodgkin's disease, one with ovarian endodermal sinus tumor) have been reported. In our oncological out-patient-department we observed in the last two years three male patients, age 19 resp. 23 resp. 35 years, with proven or strongly suspected benign thymic enlargement respective hyperplasia. These patients (two with disseminated testicular cancer, one with osteogenic sarcoma) developed the thymic enlargement after an intensive polychemotherapy without signs of relapse. The time interval between the completion of chemotherapy and the detection of the enlarged thymus was 4 resp. 8 resp. 9 months. In one case the suspected diagnosis "thymic hyperplasia" was proven by sternotomy and histological examination. In the other cases the diagnosis was made through repeated chest computed tomography. Conventional x-ray has always shown to be normal. The origin of thymic enlargement following chemotherapy is suspected as being an immunologic rebound-phenomenon after immunosuppressive therapy.

Conclusion: In patients with testicular cancer, osteogenic sarcoma and - as reported in literature - ovarian endodermal sinus tumor and Hodgkin's disease (and possibly with other tumors) benign etiologies of a newly growing mediastinal mass after completion of chemotherapy such as thymic hyperplasia must be strongly considered and possibly excluded.

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**Bra 01**

COMPARATIVE ANTITUMOR ACTIVITY OF 1-NITROSO-1-(2-CHLOROETHYL)-3-(2-HYDROXYETHYL)-UREA (HECNU) AND 1,3-BIS-(2-CHLOROETHYL)-1-NITROSOUREA (BCNU) AGAINST INTRACEREBRALLY INOCULATED GLIOMA G616

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A comparison of the antitumor activity of BCNU and the water soluble analog HECNU against intracerebrally implanted L 1210 and L 5222 leukemias revealed a distinct superiority of HECNU versus BCNU. Only HECNU was able to cure 100% of the animals at the optimal dosage in both tumor models. Now we compared HECNU and BCNU against an intracerebrally inoculated glioma of the rat (G616). Methods: Inoculated cell number: 15,000. Dosage, µmol/kg (treatment schedule, days): 50 (6,7); 60 (6,7); 70 (7,8). A comparison of the survival times of treated and control groups showed that both nitrosoureas were significantly active in all three dosage groups (p ≤ 0.05). Comparing the survival times of BCNU- and HECNU-treated groups revealed a significant superiority of HECNU after 2 x 60 µmol/kg (p = 0.007). 2 x 70 µmol/kg were already toxic for both compounds as indicated by the decreased survival compared to untreated controls. In addition, the data indicate a favorable therapeutic ratio of HECNU after repeated administration in this rat model. The results confirm that HECNU, although being 30 times more water soluble than BCNU (11% as compared to 0.4%), has an excellent activity against intracerebrally inoculated tumors. Institut für Toxikologie und Chemotherapie, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-6900 Heidelberg