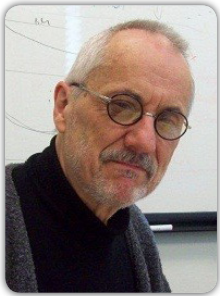


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When do we stop? Modern sequential therapies for hematologic malignancies and end of life questions


With modern medical developments various malignant disorders such as leukemia or solid organ cancers, have been transformed from acute life-threatening into chronic diseases. This transition continues with the growing number of novel agents that become available to treat diseases from chronic leukemias to breast cancer, or carcinoma of the lung. Patients are certainly aware of that, and a frequent question is: "Well, doctor, I know I have already received two treatments and have not responded. What are we going to do if this next treatment is not going to work?" In fact, it happens that you treat patients and basically have conveyed the message that we are at the end of the road, the patient considering transition to palliative or hospice care, when a new study is published with yet another agent, opening the door again, basically returning the patient from hospice to active therapy. These situations come with considerable psychological stress. In addition,

however, the cost of this type of management to the health care system is phenomenal. Some groups have admonished physicians to exert some financial stewardship. Others have argued that we cannot withhold treatment if such treatment is available. Do we need a new set of ethical rules? No one likes to set priorities or ration care. Discussions within the medical community alone will not lead to substantial change. We must have a conversation within the society at large.

Speaker Biography

H Joachim Deeg completed his MD in Wilhelms Universitaet, Germany. Presently he is working as a Professor of medicine in the University of Washington. He is also a member of the Fred Hutchinson Cancer Research Center. He is also a visiting professor at Carl Carus University, Dresden, Germany. His research interests are Pathophysiology, genetics and epigenetics of MDS (role of transcription factors in regulation) Inflammatory responses and GVHD (effects of alpha1 anti-trypsin [AAT]), Separation of GVHD and GVL effects by AAT, Iron and allogeneic responses.

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