Cancerous tissue differs from normal tissue in a number of subtle ways which enables it to invade surrounding tissues. This ability appears to be determined mainly by the surface properties of the cancer cell and differences have been found in the receptor characteristics and the antigenic determinants of the cancer cell surface in comparison with the equivalent of normal cells. Cancer diagnosis no longer relies on its physical attributes, size, shape, position, space occupation, density, water content or reflectivity, etc., as for conventional and advanced radiology, but through the essential and specific cancerousness of a cancer.

Nuclear medicine techniques have been designed to take advantage of the different antigenic determinants of cancerous from normal tissue in order to demonstrate the tumour, its recurrence and its metastases using radiolabelled monoclonal antibodies, radioimmunoscintigraphy, RIS.

Four factors are required for RIS: a selective antibody for detecting the cancer; a radiolabel to give the best signal; a radiolabelling method with appropriate quality control to give a reagent suitable for human use while maintaining the full immunoreactivity of the antibody; and an imaging system appropriate to the radionuclide and the region under study.

The choice of tissue antigen, fixed or released, normal, tumour-associated or oncofetal; the choice of antibody, whole, fragment, chimeric, CDR (complementarity determinant region) grafted or bifunctional; the choice of radiolabel, $^{123}$I, $^{111}$In or $^{99m}$Tc; and the choice of imaging, early and late, planar and SPET (Single Photon Emission Tomography) are discussed. The routine application of RIS in the clinical practice of colorectal surgery and gynaecological oncology is demonstrated.

Radioimmunotherapy, RIT, requires two important additions over RIS. Whereas RIS can cope with a high tissue background, this is a major problem for RIT for only a few cent of the injected antibody is taken up by tumour. Secondly, for RIS, superficial tumour uptake allows imaging but for therapy, deep tumour penetration and a long residence time is required. The first solution is locoregional: intraperitoneal, intratumour, intrathecal, intraarterial injection of radionuclide labelled antibodies. The second approach is systemic, which has some benefit in blood malignancies but not in solid tumours. The key new advance is to use a two or three phase system.

The biotin-avidin system has been successful. Biotin-Antibody finds tumour. Avidin clears unbound antibody and links to biotin antibody on tumour. Biotin-radionuclide ligand is then targeted to the tumour, the unbound small ligand being excreted rapidly. Alternatively a bifunctional antibody, reacting both with tumour and the radionuclide ligand, can be used. This approach uses differential biological residence time to improve the therapeutic ratio. It also allows long lived beta emitters such as $^{32}$P Phosphate to be used as the radionuclide of choice. Genetically engineered, bifunctional, designer molecules with a cassette to bind tumour, a cassette to bind $^{99m}$Tc to prove tumour uptake, and a cassette to link to a small, rapidly cleared, long lived radionuclide will bring success to this new, directed form of cancer therapy.