Despite technical advances, such as the introduction of three dimensional treatment planning systems and modern dose delivery techniques (intensity-modulated radiation therapy and image guided radiation therapy), radiation exposure to non-target organs like the bladder during radiation therapy for patients with prostate cancer remains unavoidable (1-4).

Today radiotherapy (RT) has become an increasingly popular treatment for prostate cancer with an excellent long term outcome. Early-stage prostate cancer is often treated with RT alone, with 5-year disease-specific control rates more than 95% (5,6). One of the key organs at risk (ORs) in RT of prostate cancer is the bladder (7,8).

In patients undergoing prostate RT, two phases of radiation-induced changes in the urinary bladder are observed, with both a reduction in bladder storage capacity and a consequential increase in micturition frequency (5,9). An early phase occurs 2–6 weeks after the start of fractionated irradiation, which is characterized morphologically by hyperemia and mucosal. A chronic phase develops with latent times that are inversely dose-dependent and can range up to 10 years or longer. The morphological correlate in the initial late phase is a progressive mucosal breakdown, ranging from superficial denudation to ulceration and even the formation of fistulae. The urothelial changes are accompanied by urothelial areas of compensatory hyperproliferation. Vascular changes and signs of local ischaemia have been described. These processes progress into secondary fibrosis of the bladder wall. Telangiectasia can result in severe bleeding episodes. The early changes clearly correlate with the chronic radiation sequelae, illustrating a strong consequential component (6,9).

After the first parameterization of dose–volume effects based on the experience from the 2D RT era reported by Kuthcher et al. (10), the availability of 3D dose–volume information dramatically increased the amount of quantitative data. These efforts translated into both the proposal of reliable dose–volume constraints able to reduce toxicity, and the development of normal tissue complication probability (NTCP) models properly fitting these data in a usable way.

Considering a mixed serial–parallel behavior of the bladder (11,12), for NTCP modeling, various phenomenologic or empirical models (Lyman with the dose-volume histogram [DVH] reduced to the equivalent uniform dose [LEUD], mean dose, and Logit-EUD [LOGEUD], as well as tissue architecture models (the relative seriality [RS] or Kallman S and 2 functional subunit [FSU]-based models known as the individual and population critical volume [CV] models) can be used (13-15).

However, the process of establishing dose/volume parameters being predictive for normal tissue morbidity following RT is likely to be influenced by the geometric uncertainties of the concerned organ (16,17), with potential implications for the estimated dose/volume parameters (2,3). The bladder, displays considerable motion (inter- and intra-fraction) during the treatment period. Bladder motion causes both random and systematic errors which will blur and shift, respectively, the dose distribution relative to the target (2,3).

In order to predict bladder complications after prostate RT, knowledge of how the internal bladder motion influences the dose/volume parameters is central to obtain accurate information of the dose/volume constraints (4).

A simple strategy to account for internal organ motion...
and set-up uncertainties and capture the dose distribution in the volume space in which the OR is likely to move within is to expand (i.e. add margins to) the OR (18). This approach is commonly denoted the planning organ at risk volume (PRV) concept and was first introduced in the International Commission on Radiation Units and Measurements (ICRU) Report 62 (19).

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