Beginning in 1999, Fleming began the development of a quantitative method (FMTVDM), which accurately used nuclear imaging to truly quantify both [1], heart disease and [2], breast cancer.

The work began by asking if it was possible to develop a test, which could specifically identify breast cancer. This quantitative method began by identifying the two critical properties of blood flow and metabolism, which critically distinguish cancer from inflammation from normal breast tissue from calcium. The second task was to determine if there was a method, which could “enhance” those differences for a limited period of time. Using methods commonly employed in Cardiology, Fleming was able to develop such a diagnostic test, enhancing these differences.

The final question remained whether it was possible to accurately measure such differences, which proved to be possible after identifying problems with modulation transfer function (MTF) and Fourier Transform (FT). In addressing these problems Fleming was able to correct for the loss of up to 50% of the data, using known samples of technetium 99-m and measuring decay curves.

This method for quantitatively measuring Heart Disease and Breast Cancer was named “The Fleming Method for Tissue and Vascular Differentiation and Metabolism (FMTVDM) using same state single or sequential quantification comparisons” with the subcomponent “Breast Enhanced Scintigraphy Test” (FMTVDM–BEST) requiring imaging, beginning 5-minutes after injection of Sestamibi or Myoview, which were injected immediately following the completion of pharmacologic stress. While Tc–99m isotopes were used in one version of the patent, the patent is applicable to all isotopes and all nuclear cameras with timing based upon the specific isotope being used.

In the process of beginning acquisition of 5-minute images for breast cancer Fleming additionally obtained cardiac images, which lead to the discovery that these technetium-99m isotopes including both Sestamibi and Myoview actually redistribute beginning as early as 5-minutes following injection. Such 5-minute images had not been done previously as the isotopes being used reportedly did not redistribute and cardiac images were not typically acquired until 60-minutes after isotope injection, long after the redistribution had already happened. Despite this misinformation numerous others [1–5], had also reported this redistribution.

The combined cardiac “The Fleming Method for Tissue and Vascular Differentiation and Metabolism (FMTVDM) using same state single or sequential quantification comparisons” and Breast cancer (FMTVDM–BEST©) studies included a second set of redistribution cardiac images obtained at 60-minutes without requiring a second injection of isotope. The accurate measurement of changes in isotope redistribution in regions of interest using the corrections to MTF/FT allowed FMTVDM to be used to accurately “quantify” changes in isotope in cardiac tissue, measured changes which were then incorporated into the proprietary equations of Fleming to determine changes in coronary blood flow and accurately measure isotope “wash-in” and washout (Figure 1).

“Wash-in” being the phenomena, where the 5-minute cardiac images had diminished isotope, which later “washed-in” to the regions of critically narrowed arteries and arteries with vulnerable inflammatory plaques over the course
of 60-minutes (Figures 1,2); Thus yielding the “Health-Spectrum” of coronary artery disease (ASCAD). Without the inclusion of the initial breast cancer and cardiac images, the wash-in phenomena and the true quantitative measurement made possible by FMTVDM and the calculation of true ASCAD disease using the proprietary equations, would not have been discovered.

References


Figure 1: Measurement of isotope redistribution using FMTVDM.

Figure 2: ASCAD placing the severity and extent of disease on a “Health-Spectrum” Continuum. Proceeding from left to right, the progression of ASCAD proceeds from normal functioning coronary arteries through the build up of inflammation within the walls of the coronary arteries, impairing the flow reserve through intrusion of the inflammatory plaque into the lumen of the artery.

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