
Composition of venous and arterial thrombi differs from that of blood and may change in different thrombogenic conditions and in antithrombotic therapy. Thrombus formation was investigated by labeling of platelets (59Fe), fibrinogen (125I) and red cells (59Fe or 51Cr). Thrombosis was induced in 3 different models: I. Formation of slow growing arterial and venous thrombi, II. rapid venous thrombus formation within 20 min by application of silver nitrate and III. production of stasis thrombi. The radiometric thrombus size was related to the thrombus weight by means of Spearman rank correlation. The specific thrombus/blood ratio (cpm g thrombus/cpm g blood) was measured in the series I in venous and arterial thrombi to be 19.1 and 50.9 (platelets), 12.6 and 6.1 (fibrinogen) and 5.1 and 1.7 (red cells) and in the series II 5.7 (fibrinogen) and 1.5 (red cells). In stasis thrombi these values resembled nearly to that of blood. Dependent on the pathogenetical mechanism of thrombus formation a different incorporation of blood elements could be stated with the greatest discrepancies in the uptake rates of red cells in arterial thrombi. Statistical analysis of methods used to measure thrombus size demonstrated only in the venous system a reliability for measuring thrombus size by isotopic methods. Correlation coefficient was 0.8 (59Cr), 0.76 (125I) and 0.83 (59Fe) in the model I and 0.84 (125I) and 0.92 (51Cr red cells) in the model II.


In 32 patients suspected of DVT 3 methods were carried out: 1. 99m Tc-UKurinase(Tc-UK) labelled according to Millar was injected s.c. Both legs were scanned with a gammacamera in search for hot spots. 2. 99m Tc-macroaggregates of albumin (To-MAA) was injected in veins on both feet simultaneously. With a gammacamera the isotope flow pattern was followed. Hot spots were registered and a lungscan carried out. 3. In 14 of these patients afterwards a Röntgen phlebography was carried out. Results: Neither MAA the presence of hot spots could be used as proof for DVT. Isotope chro- physiology gave only contributory evidence. In 9 cases out of 32 there was a discrepancy between the To-UK and To-MAA scan. There was a better positive correlation between Röntgen phlebography with the To-UK (11 out of 14) than with the To-MAA scan (7 out of 14). In 6 out of 7 patients Röntgen phlebography correlated in localisation with isotope phlebography. In 6 out of 7 in 6 patients there was an abnormal lungscan. In 6 of these patients there was also a positive To-UK lesion but only twice a positive To-MAA lesion. Conclusion: To-UK is an easy test in the diagnosis of DVT. To-MAA can give additional evidence and provides at the same time a useful lungscan.