

TRANSIENT LOSS AND COMPLETE RECOVERY OF THE POPULATION OF PLATELETS DURING THERAPEUTIC PLASMA EXCHANGE

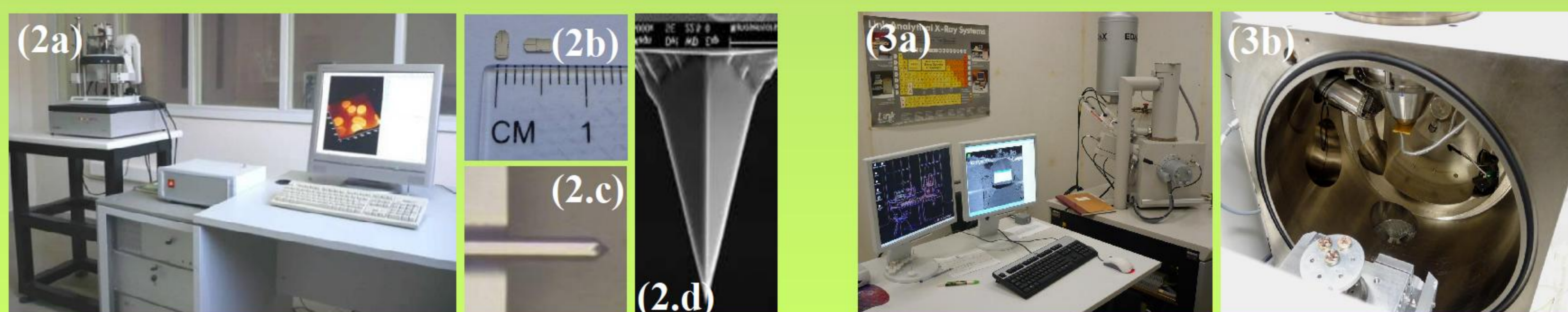
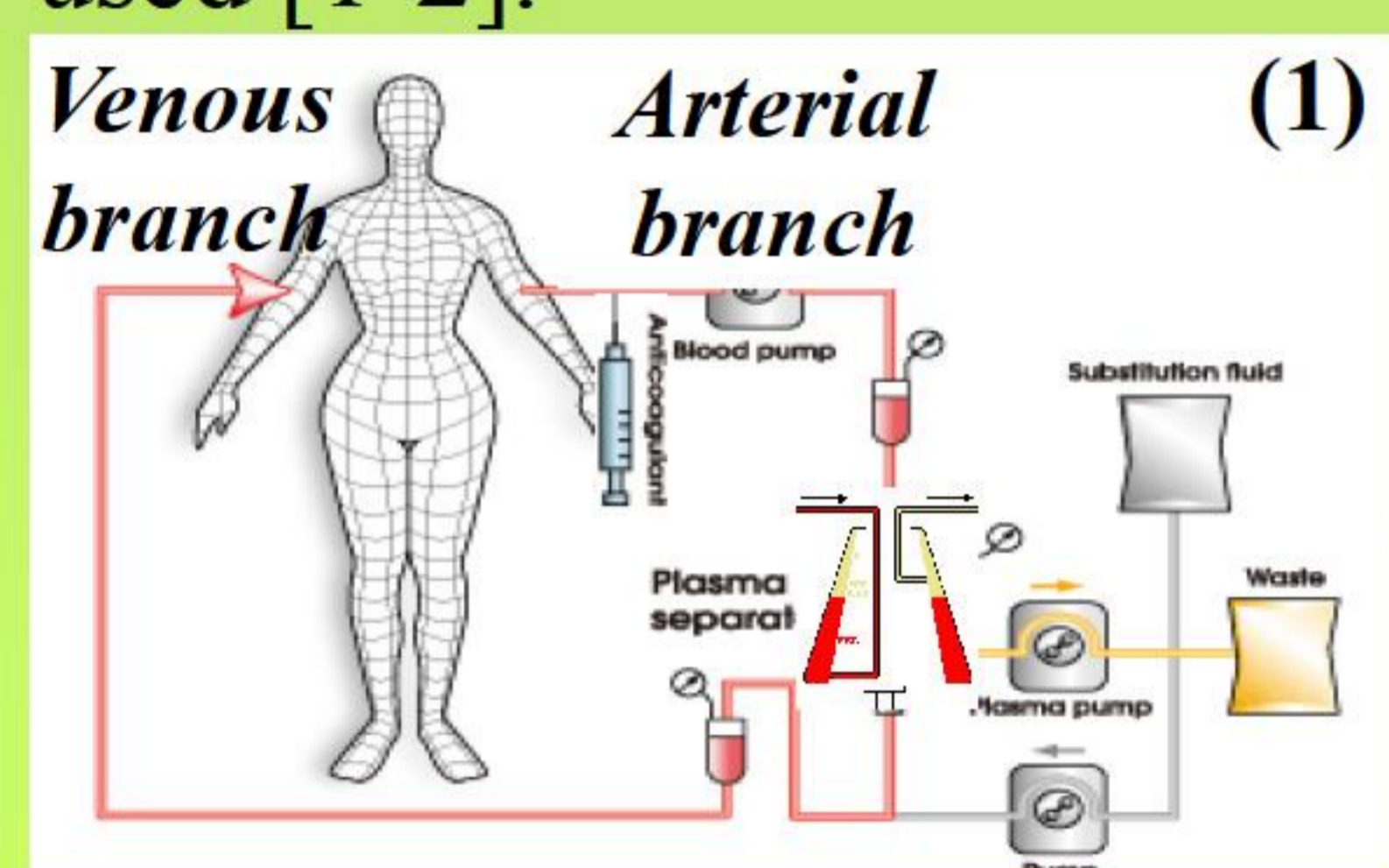
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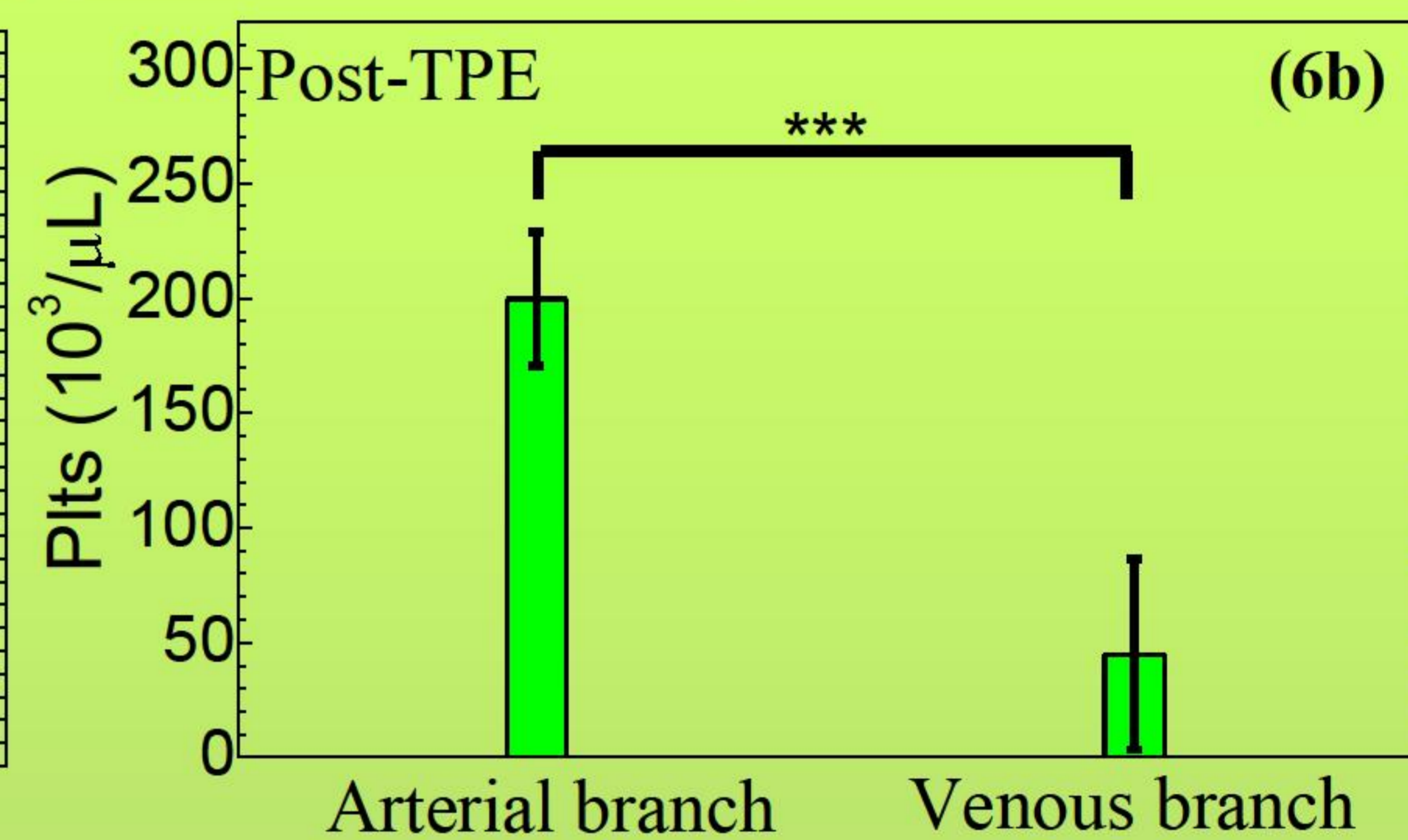
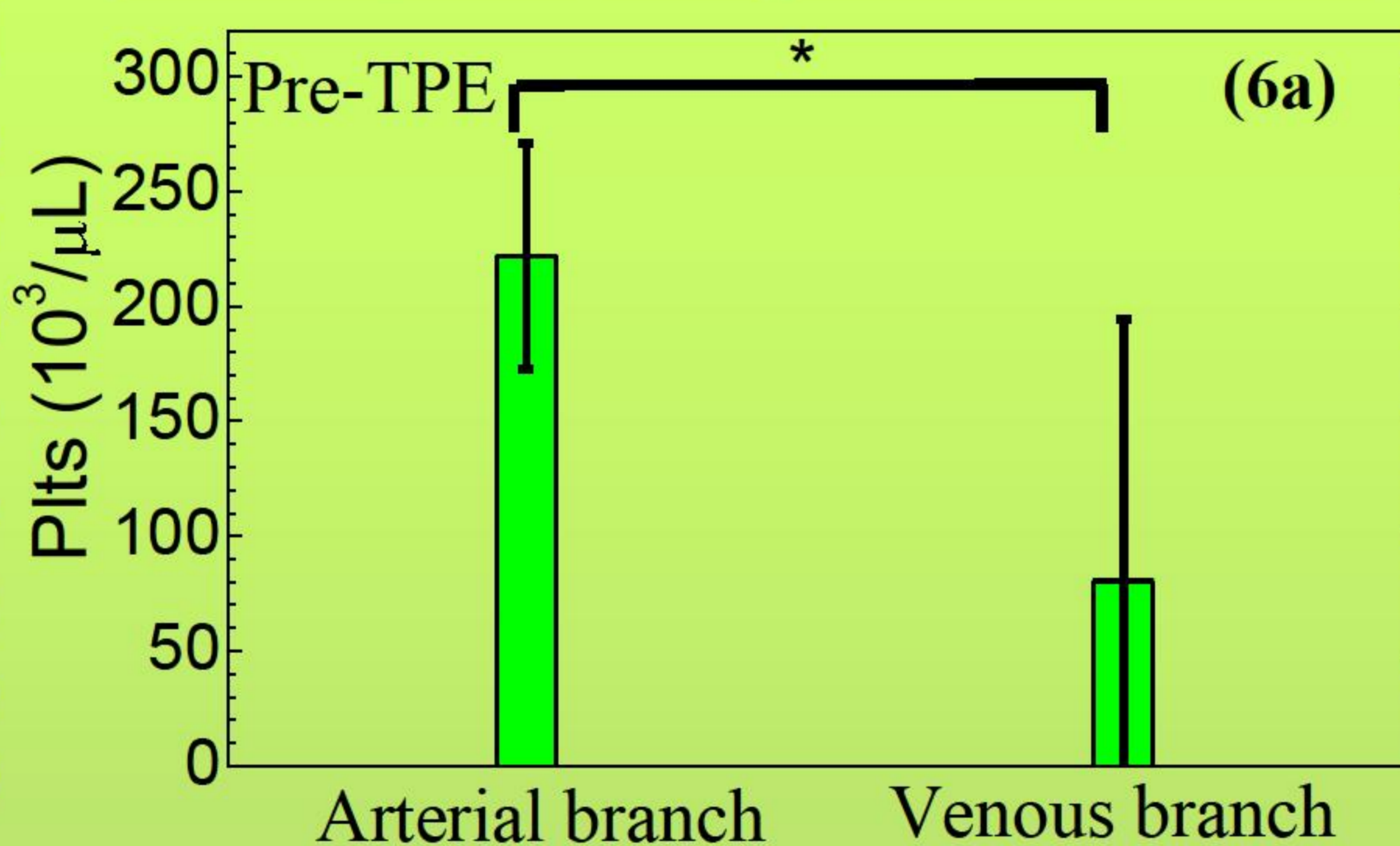
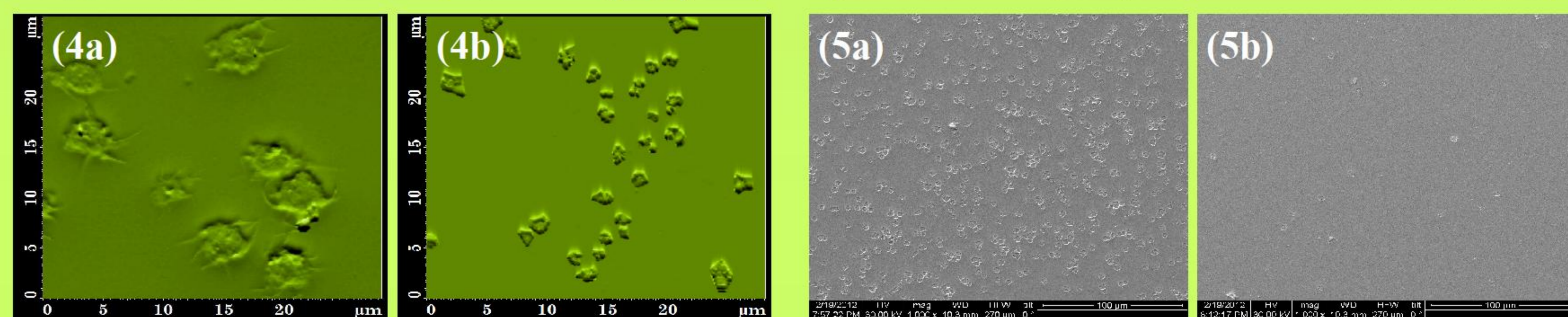
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OBJECTIVES: In therapeutic plasma exchange (TPE) thrombocytopenia is observed in some patients. Usually this is ascribed to the unintentional removal of Platelets (Plts) with the plasma that is rejected at the centrifugation stage. Except for this possibility other mechanisms could be at play: mechanical stress (MS) experienced by cells during the relatively intense centrifugation and biochemical shock (BS) exerted from the replacement medium can deconstruct Plts. Here, we studied intact Plts (iPlts) of TPE patients with standard clinical techniques and advanced microscopes.

METHODS: During the TPE sessions the 15 patients studied here were given a combination of colloid and crystalloid media (Human Albumin 5%, Hydroxyethyl Starch 6% and saline NaCl 0.9%). The Cobe® Spectra and Spectra Optia® units were employed. For the determination of the TPE dose and replacement-medium volume the standard Nadler and Allen formula was used. The iPlts investigation was conducted comparatively in samples drawn *simultaneously* from the *venous* and *arterial* branches, Fig. (1), at both the *beginning* and *end* of TPE session. Standard clinical tests and two powerful Microscopes, the Atomic Force (AFM), Figs. (2a)-(2d), and Scanning Electron (SEM), Figs. (3a)-(3b), were used [1-2].



RESULTS: Discrepancies were observed in the count of Plts across the extracorporeal circuit. Specifically, the combined microscopy, Figs. (4a)-(4b) and (5a)-(5b), and clinical, Figs. (6a)-(6b), data revealed that in 5 out of 15 TPE patients the Plts exhibited a statistically significant ($p < 0.05$) *intense reduction in the venous branch and complete recovery in the arterial branch* in the samples obtained at both the *beginning* (venous: $80.6 \pm 114.0 \times 10^3/\mu\text{L}$ and arterial: $221.8 \pm 49.1 \times 10^3/\mu\text{L}$) and *end* (venous: $44.8 \pm 41.5 \times 10^3/\mu\text{L}$ and arterial: $199.6 \pm 29.3 \times 10^3/\mu\text{L}$) of TPE session, Fig. (6a)-(6b). We speculate that there is a mechanism of Plts reconstruction occurring inside the patient.



No statistically significant ($p > 0.05$) difference between the *beginning* and *end* of the TPE session in both the *venous* (beginning: $80.6 \pm 114.0 \times 10^3/\mu\text{L}$ and end: $44.8 \pm 41.5 \times 10^3/\mu\text{L}$) and *arterial* (beginning: $221.8 \pm 49.1 \times 10^3/\mu\text{L}$ and end: $199.6 \pm 29.3 \times 10^3/\mu\text{L}$) branches was observed. Based on the data we estimate that the Plts 'loss' observed at the venous branch measures almost 70% of the extracorporeal population, while there is a complete Plts recovery at the arterial branch at the end of TPE session. A question naturally arises on the mechanism of Plts recovery since, according to existing knowledge, the spleen can reserve only 30-50% of total Plts mass. We speculate that there is a mechanism of Plts reconstruction occurring inside the patient.

CONCLUSIONS: In some cases across the extracorporeal circuit Plts may transiently be deconstructed up to 70% due to MS and BS that they experience. A possible consequence is the observation of discrepancies in Plts count since automated analyzers do not register particles of size below a threshold value ($1 \mu\text{m}$) such as granules of deconstructed Plts. Since the spleen can only reserve 30-50% of Plts total mass we speculate that the Plts that are transiently deconstructed at the extracorporeal circulation are efficiently reconstructed when returned to the biochemically fertile cardiovascular system of the patient.

REFERENCES

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