

INTERACTION BETWEEN CIRCADIAN BLOOD PRESSURE RHYTHM AND AUTONOMIC NERVOUS FUNCTION IN RENAL TRANSPLANT RECIPIENTS



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Introduction

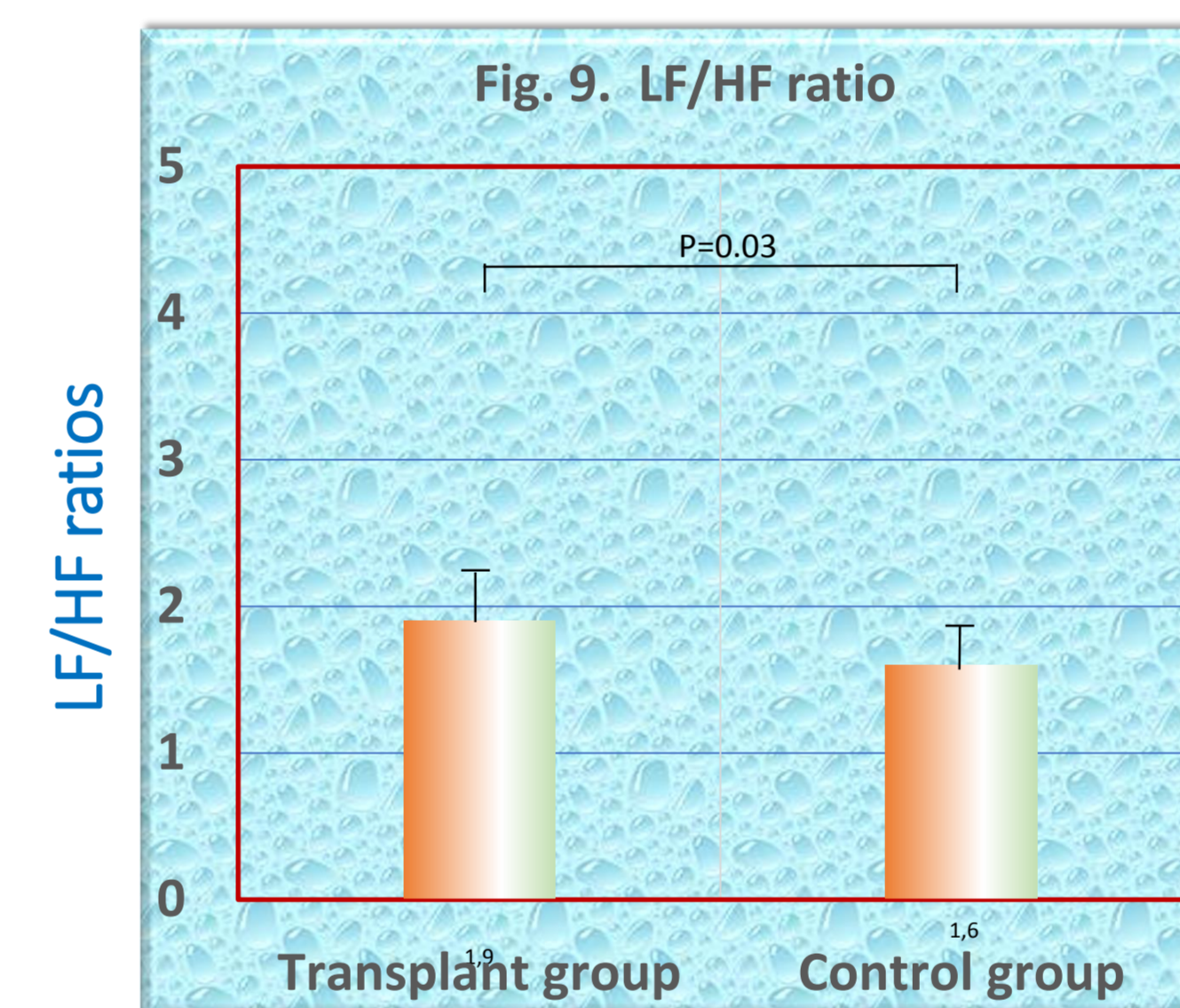
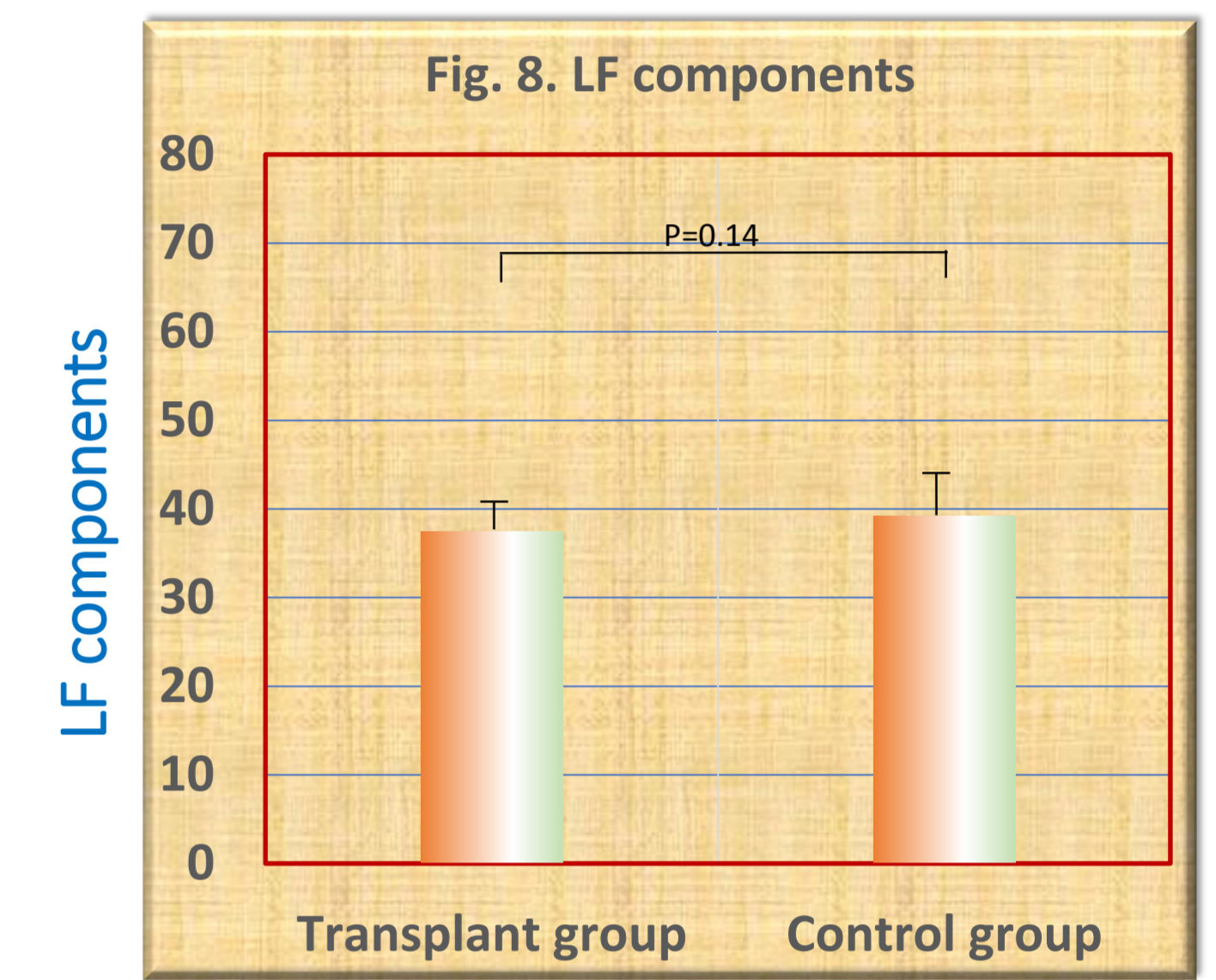
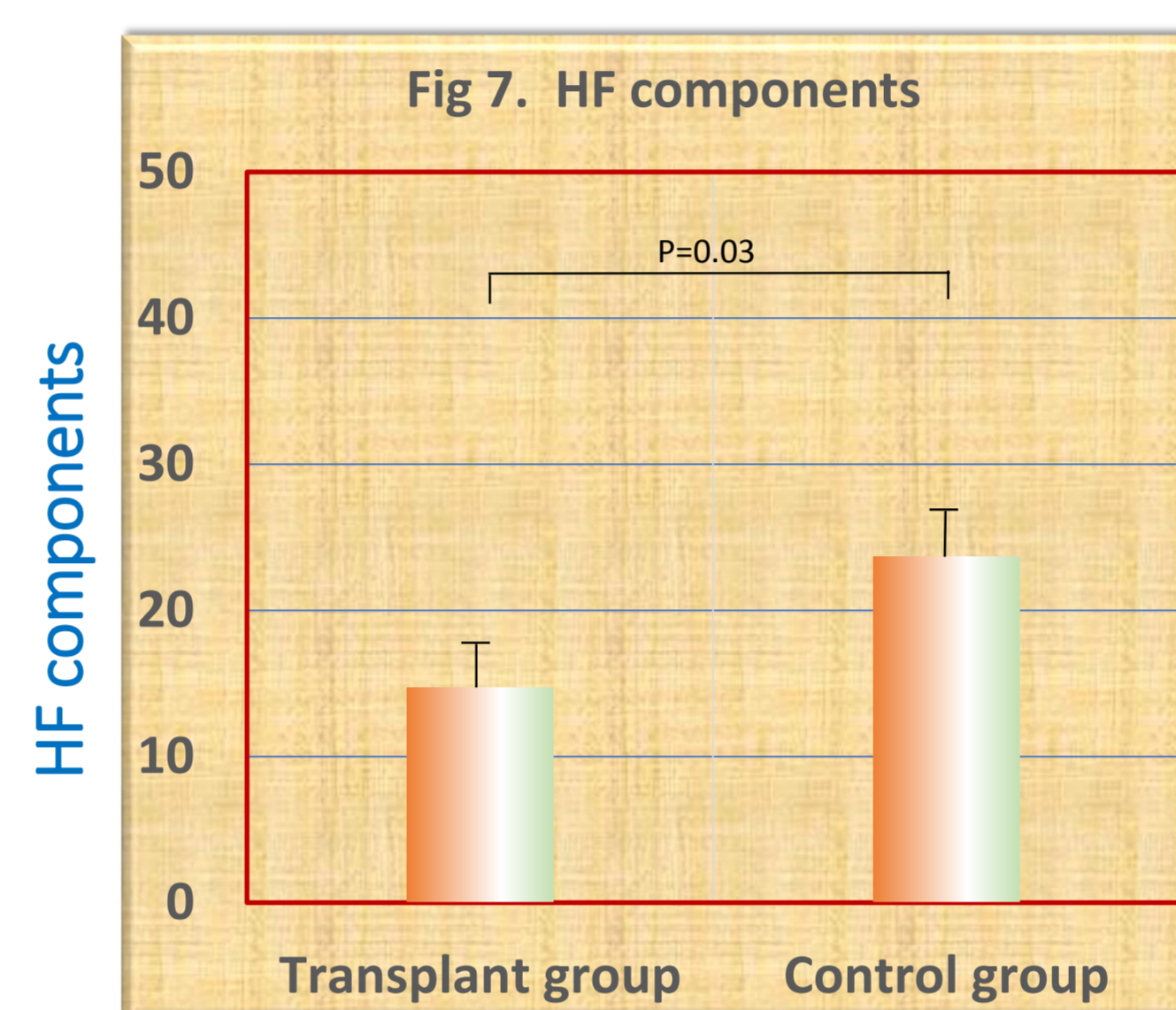
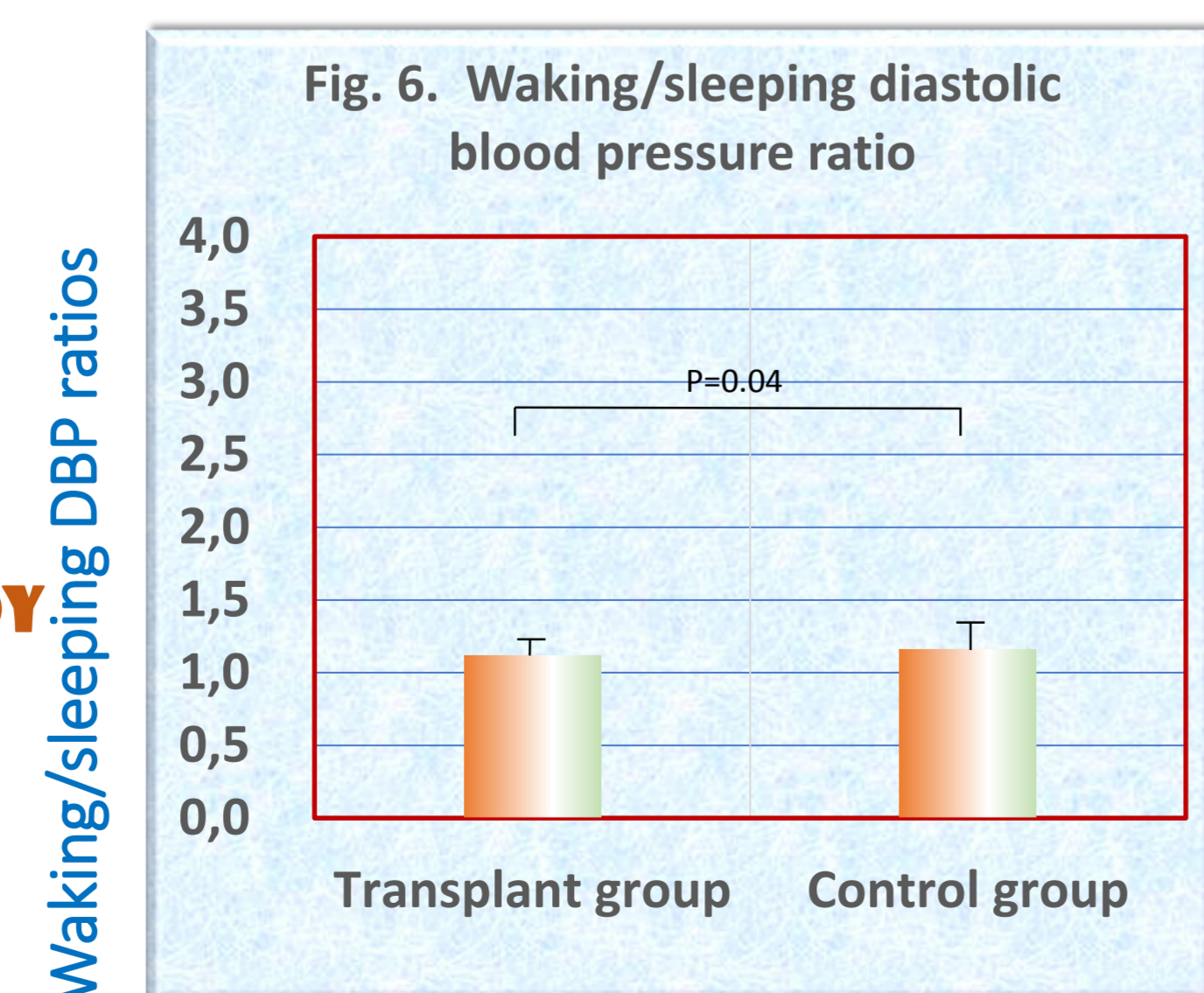
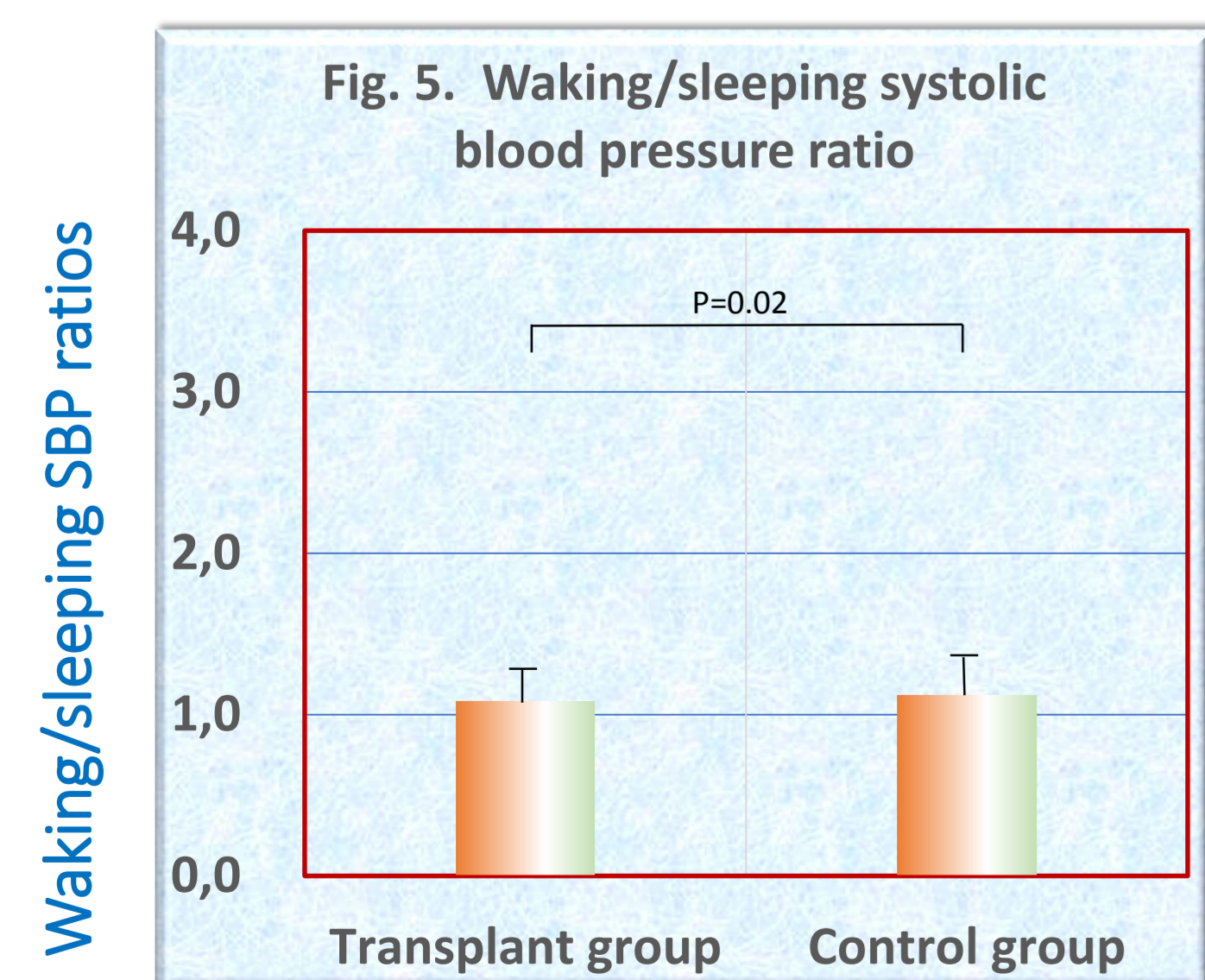
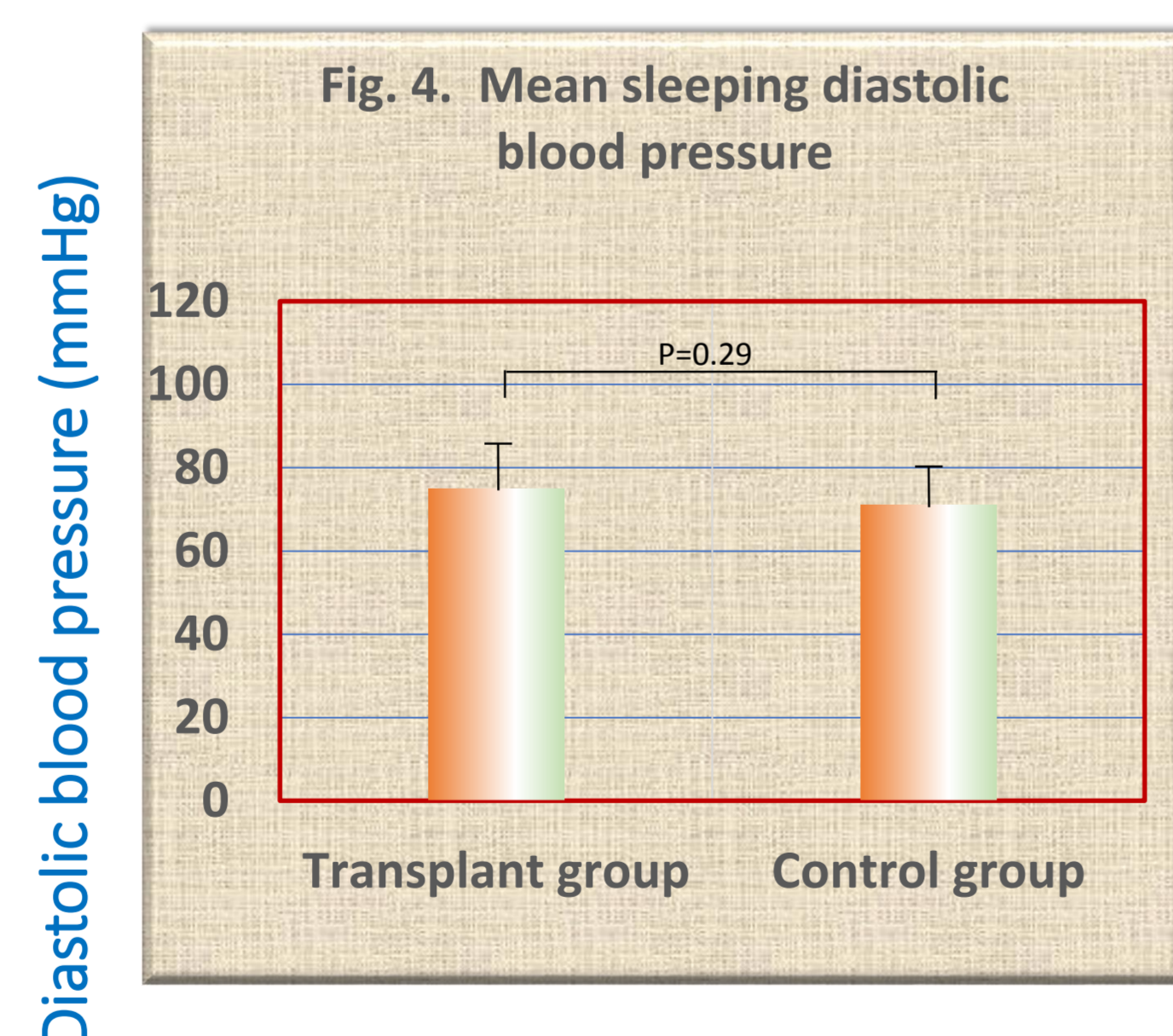
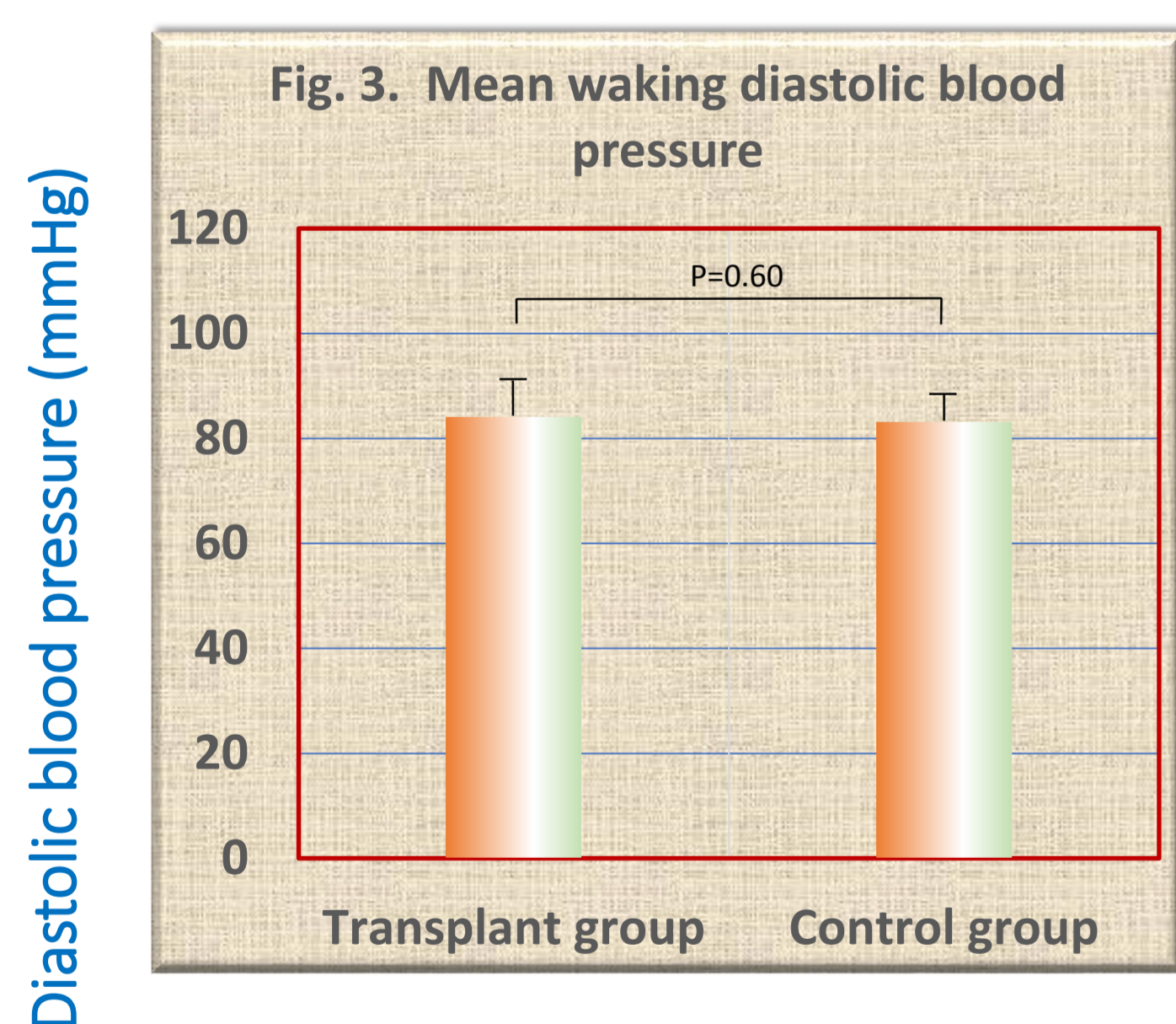
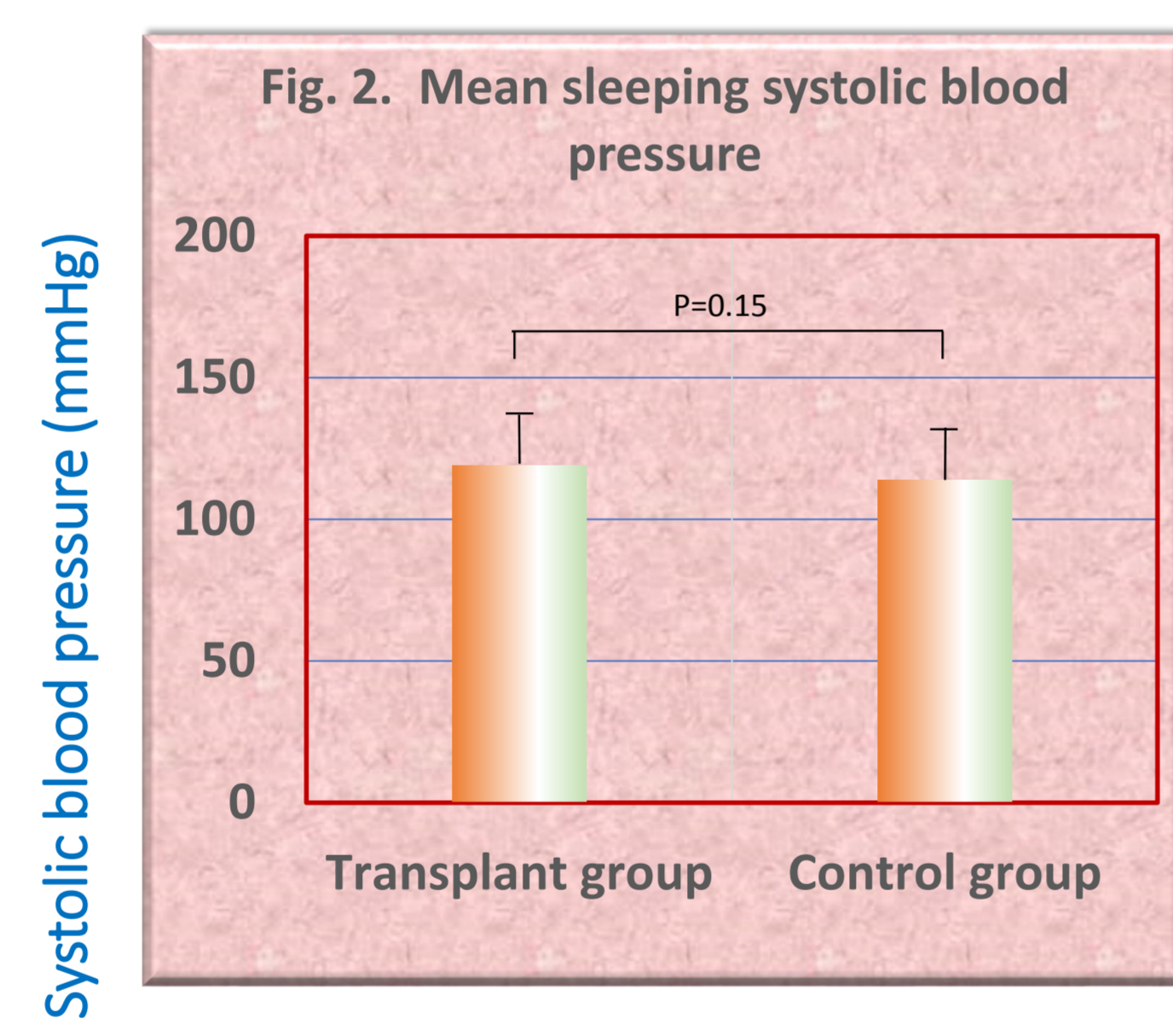
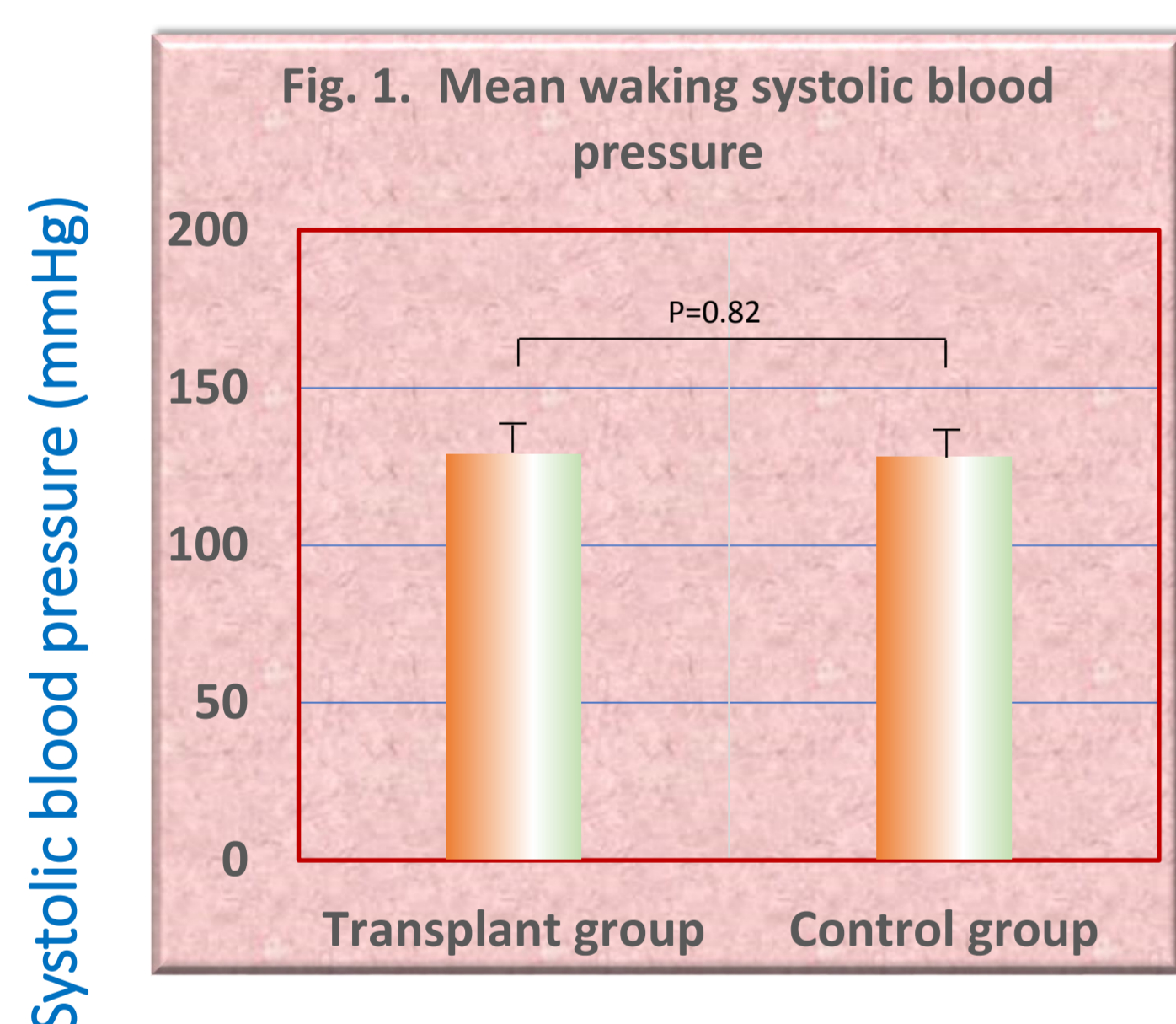
Autonomic nervous dysfunction is often seen in patients with renal transplant. They frequently show loss of the nocturnal decline of blood pressure (BP) and imbalance in autonomic nervous function. In these patients, however, the relation between autonomic nervous function and the circadian BP rhythm is not yet fully understood. We evaluated 24-hour BP patterns in renal transplant patients using ambulatory BP monitoring (ABPM) devices. We also analyzed the power spectrum of heart rate variability as an index of autonomic cardiovascular modulation.

Materials and Methods

Twenty-one renal transplant recipients (mean age, 35 ± 9 years old; men/women, 12/9; estimated glomerular filtration rate >60 mL/min) as the transplant group and age- and sex-matched twenty healthy subjects (mean age, 34 ± 8 years old; men/women, 11/9; estimated glomerular filtration rate >60 mL/min,) as the control group were enrolled in the study (Table). Patients in the transplant group were receiving prednisolone between 5 and 10 mg/day and cyclosporine between 50 and 200 mg/day. Each subject underwent ABPM and was assessed the heart rate variability. Waking and sleeping time BP ratios were calculated as an index of the loss of nocturnal BP decline. Power spectral analysis of the heart rate variability was analyzed for the high frequency (HF) components, as an index of parasympathetic nervous activity, and low frequency (LF) components, as an index of sympathetic nervous activity. The ratio of LF components to HF components was determined as an index of sympathovagal balance.

Table 1 Subjects participated in the study

	Renal transplant recipients	Control subjects
• Number	21	20
• Age(years old)	35 ± 9	34 ± 8
• Men/women	12/9	11/9
• Serum creatinine concentrations (mg/dL)	1.3 ± 0.6	1.1 ± 0.5
• Estimated glomerular filtration rate (ml/min/1.73m ³)	48 ± 12	69 ± 9



Results

Mean waking and sleeping systolic BP were 129 ± 14 (SD) and 119 ± 17 mmHg in the transplant group, respectively, which were as high as ($P > 0.05$) 128 ± 13 and 114 ± 15 mmHg in the control group (Figures 1, 2). Mean waking and sleeping diastolic BP were 84 ± 12 and 75 ± 10 mmHg in the transplant group, respectively, which were also as high as ($P > 0.05$) 83 ± 10 and 71 ± 11 mmHg in the control group (Figures 3, 4). There were, however, differences ($P < 0.05$) in the waking and sleeping systolic/diastolic BP ratios between $1.08 \pm 0.10/1.12 \pm 0.09$ in the transplant group and $1.12 \pm 0.11/1.16 \pm 0.12$ in the control group (Figures 5, 6), showing the loss of nocturnal BP decline in the transplant group. The 24-hour HF components in the transplant group showed 14.7 ± 13.3 , which was lower ($P = 0.03$) than 23.7 ± 14.0 in the control group (Figure 7), but 24-hour LF components were not different between the two groups (37.5 ± 18.1 vs. 39.2 ± 21.1 , $P = 0.14$, Figure 8). Consequently, there were significant differences ($P = 0.03$) in 24-hour LF/HF ratios between 1.9 ± 0.7 in the transplant group and 1.6 ± 0.6 in the control group (Figure 9).

Conclusions

Our findings suggest that sympathovagal imbalance occurs in the renal transplant patients and this causes loss of the nocturnal decline of BP. It is suspected that affected autonomic nervous innervation to transplanted kidneys as well as native kidneys, and immunosuppressive medicines contribute to the impaired circadian BP rhythm.

