# Effect of supraphysiological estrogen levels on arterial stiffness and hemodynamic parameters

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## Abstract

**Objective:** The present study evaluates the arterial stiffness and hemodynamic parameters in patients with a supraphysiological estrogen level due to *in vitro* fertilization (IVF) with controlled ovarian hyperstimulation (COH).

**Methods:** A total of 82 female patients aged 24–45 years were included. Their arterial stiffness parameters were analyzed before and after the appropriate COH protocol involving arteriography using Mobil-O-Graph NG (IEM GmbH, Stolberg, Germany) 24-hour ambulatory blood pressure monitor.

**Results:** Systolic, diastolic, mean, central systolic, and diastolic blood pressures, as well as peripheral vascular resistance, were significantly lower after COH therapy (p=0.001, 0.002, <0.001, <0.001, 0.040, and <0.001, respectively). In contrast, there was no statistically significant difference observed in heart rate, pulse pressure, or cardiac output. The pulse wave velocity measurement was significantly lower after COH than the baseline levels [5.3 m/s (4.5–6.9 m/s) versus 5.4 m/s (4.7–7.3 m/s,); p<0.001], but the augmentation index was not significantly different [28% (4%–41%) versus 29% (5%–43%); p=0.090]. When the patients were grouped according to the occurrence of a pregnancy after IVF therapy, all parameters were not different between the pregnancy (+) and pregnancy (-) patients (p>0.05).

**Conclusion:** Arterial stiffness and hemodynamic parameters significantly decreased in IVF patients who underwent COH therapy. The long-term clinical significance of this short-term effect should be investigated with prospective studies. There was no significant difference in all parameters before and after COH when the pregnancy (+) and pregnancy (-) patients were compared.

**Keywords:** arterial stiffness, controlled ovarian hyperstimulation, supraphysiological estrogen, *in vitro* fertilization, pulse wave velocity

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## Introduction

*In vitro* fertilization-embryo transfer (IVF-ET) is the most commonly used assisted reproduction technique in the treatment of infertility (1). Controlled ovarian hyperstimulation (COH) for *in vitro* fertilization (IVF) uses recombinant gonadotropins to stimulate follicle growth. The estradiol (E2) levels in women are normally 27–123 pg/mL in the follicular phase and 96–436 pg/mL in the luteal phase during menstrual period and then rises to approximately 4000 pg/mL during IVF therapy with COH (1, 2). Despite its safety, the treatment affects the cardiovascular system both positively and negatively due to rapid hormone changes (1, 3).

Arterial stiffness is a sign of endothelial dysfunction caused by complex and dynamic interactions of cellular and structural elements of the vessel wall. Pulse wave velocity (PWV) and the augmentation index (AIx) are used to assess the changes in arterial stiffness. PWV is accepted as the gold standard technique to measure arterial stiffness (4, 5).

The objective of this study was to investigate the influence of COH on arterial stiffness using the Mobil-O-Graph NG 24-hour ambulatory blood pressure monitor (IEM GmbH, Stolberg, Germany), a brachial cuff-based device that records oscillations and pulse waves.

## Methods

### Study population

This study enrolled 90 patients who were referred to the IVF center of the Ondokuz Mayıs University Faculty of Medicine for infertility treatment between January 2019 and June 2019. The

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**HIGHLIGHTS**

- In women undergoing *in vitro* fertilization (IVF) with controlled ovarian hyperstimulation, arterial stiffness and central hemodynamic parameters were significantly reduced compared with the baseline levels. However, when the patients were divided into 2 groups based on achieving a pregnancy with IVF therapy, there was no statistically difference in all parameters between pregnancy (+) and pregnancy (-) women. The long-term clinical significance of these short-term changes should be investigated with prospective studies.

exclusion criteria were the presence of known systemic or cardiovascular disease, hypertension, heart failure, cardiomyopathy, diabetes mellitus, ongoing medical treatment with substances that have cardiovascular effects, history of pregnancy, previous COH therapy, and improper measurement of blood pressure. After excluding 8 patients, 82 female patients aged 24–45 years who underwent COH therapy were included. Clinical data of the patients such as age, body weight, height, cardiovascular risk factors, current medications, and other systemic diseases were collected. Informed consent was obtained from all patients. This study was approved by the Ethics Committee of the Ondokuz Mayıs University Faculty of Medicine (No: 2019/193).

### Controlled ovarian hyperstimulation

Patients who were to undergo COH were referred to the cardiology department for baseline measurements prior to starting treatment. COH was performed according to an antagonist protocol involving a recombinant follicle-stimulating hormone (Gonal-f; EMD Serono Inc., Rockland, MA, USA) and a gonadotropin-releasing hormone antagonist (Cetrotide, 0.25 mg; EMD Serono Inc., Rockland, MA, USA). Recombinant human chorionic gonadotropin (hCG) (Ovitrelle, 250 mg; EMD Serono Inc., Rockland, MA, USA) was administered for oocyte maturation when two or more follicles (≥17 mm in length) were determined via serial ultrasonographic evaluation. The patients were followed up every 3 days with serial transvaginal ultrasonography and the level of estradiol was measured by an obstetrician. Oocyte pick-up (OPU) was performed 36 hours after the administration of hCG therapy. Control arterial stiffness parameters were measured on the day of OPU, when the estradiol level was at its highest and the oocyte maturation was evaluated as appropriate by the obstetrician.

Blood sampling to determine basal laboratory parameters and estradiol level was performed before beginning COH therapy. The obstetrician monitored the estradiol level and recorded its peak after administering COH therapy. All of the blood samples were taken from antecubital vein between 9:00 and 10:00 am in the same room and analyzed under the same laboratory.

### Measurement of arterial stiffness

Mobil-O-Graph NG 24-hour ambulatory blood pressure monitor was used to evaluate arterial stiffness and cardiovascular hemodynamic parameters. A cuff of suitable dimensions was used to record measurements via the brachial artery. Prior to the assessment, the patients avoided coffee, tea, or other stimulants and were seated in a quiet, temperature-controlled room (22°C–24°C) at a morning appointment. The cuff was automatically pumped by the device to at least 35 mmHg above the measured pressure level until no blood could flow through the brachial artery. The recorded brachial blood pressure and brachial waveforms were uploaded to the Hypertension Management Software: Client Server 5.1 software (IEM GmbH, Stolberg, Germany). Hemodynamic parameters such as systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate, pulse pressure (PP), central systolic blood pressure (cSBP), central diastolic blood pressure (cDBP), AIx, peripheral vascular resistance (PVR), PWV, and cardiac output (CO) were estimated from central aortic pressures and carotid-femoral PWV (Fig. 1). The validity of the measurements of this device to estimate arterial stiffness and determine hemodynamic parameters has been demonstrated in previous studies using both invasive and noninvasive methods (6-12).

### Statistical analysis

The collected data were transferred to a computer and analyzed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to determine the normality of distribution of continuous variables. Categorical variables were represented as numbers and percentages, and continuous variables were represented as mean (standard deviation) values on normal distribution and as median (minimum-maximum) if the variables revealed non-normal distribution. A paired t-test or Wilcoxon signed-rank test was used to assess the variables at baseline and follow-up in the same group according to the type of distribution. The patients were divided into two groups based on the occurrence of pregnancy in patients who underwent COH therapy. The Mann-Whitney U test was used to determine statistically significant differences in the variables without normal distribution between the groups, and Student's t-test was used for normally distributed parameters. Pearson's chi-square test and Fisher's exact test were used to assess categorical variables. A $p$ value of <0.05 denoted statistical significance.

## Results

A total of 90 patients were initially enrolled in the study, 8 of whom were excluded: 4 were newly diagnosed with hypertension (>140/90 mm Hg) and 4 others did not complete the arterial stiffness measurement protocol. The data of 82 patients included for analysis were baseline descriptive characteristics, laboratory parameters, and basal and peak estradiol levels (Table 1).

SBP, DBP, MAP, cSBP, cDBP, and PVR measurements were statistically significantly lower after COH therapy than those before treatment ($p$=0.001, 0.002, <0.001, <0.001, 0.040, and <0.001, respectively). There was no statistically significant difference in the heart rate, PP, or CO before and after ovarian

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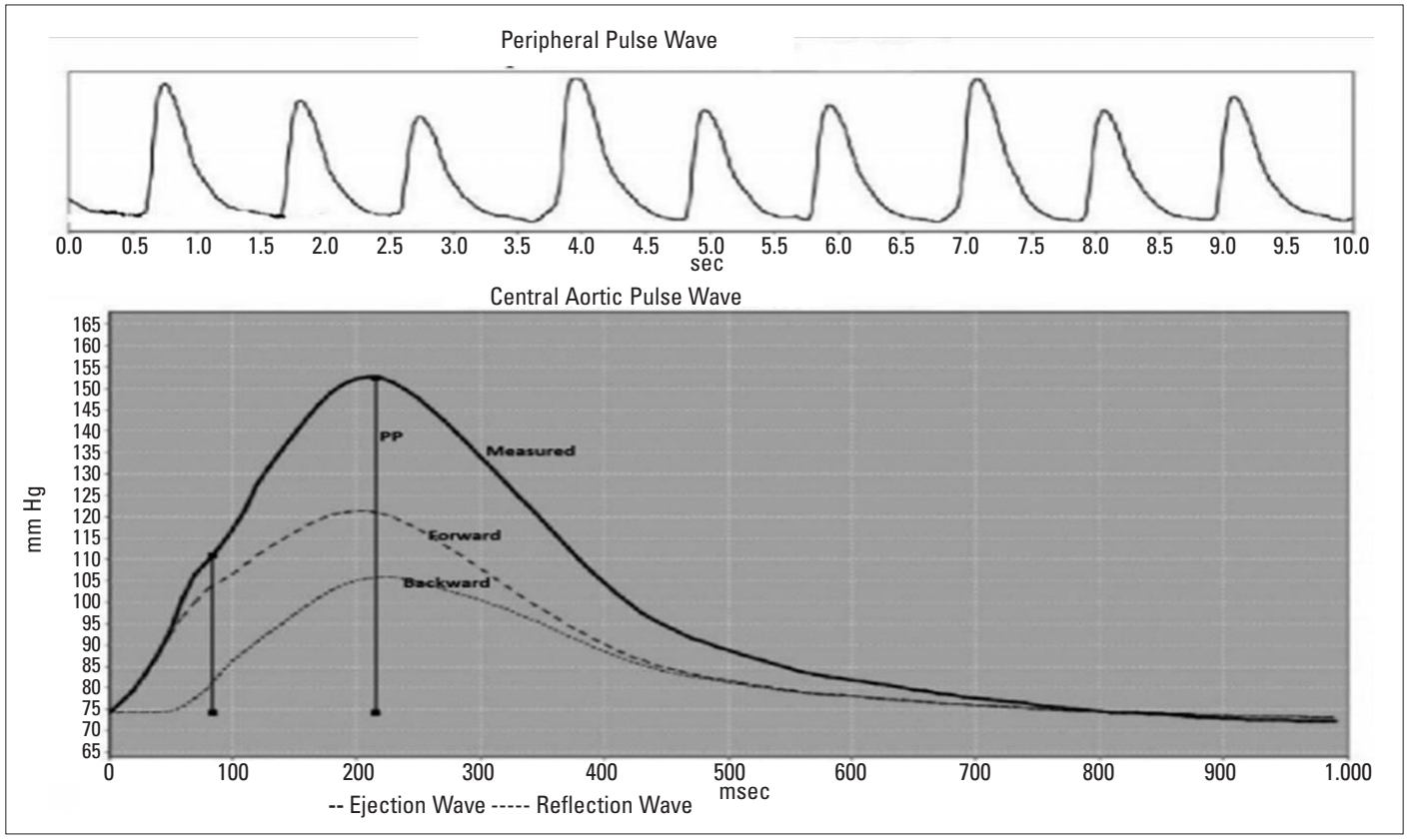


**Figure 1.** Assessment of aortic pulse wave velocity using a transfer function from brachial pressure wave analysis

**Table 1. Clinical characteristics and laboratory parameters of the study population**

| Variable | | IVF therapy (n=82) Mean ± SD Median (min-max.)* |
|---|---|---|
| Age (year) | | 33.4±5.5 |
| BMI (kg/m$^2$) | | 25.8±3.9 |
| Cigarette, n (%) | | 16 (19.5%) |
| Basal estradiol (pg/mL) | | 35.9 (5-125)* |
| Peak estradiol (pg/mL) | | 1200 (196-4160)* |
| Glucose (mg/dL) | | 88 (65-122)* |
| Creatinine (mg/dL) | | 0.57 (0.44-0.82)* |
| Hemoglobin (g/dL) | | 13.3±1.0 |
| White blood cell (10$^3$/mL) | | 7.7±1.8 |
| Platelet (10$^3$/mL) | | 285±66.7 |
| AST (U/L) | | 17 (10-97)* |
| ALT (U/L) | | 14 (6-74)* |
| Drugs | Levotiroxin, n (%) | 5 (6.1%) |
| | Anti-depressants, n (%) | 2 (2.4%) |
| Pregnancy | (+) n (%) | 18 (22%) |
| | (-) n (%) | 64 (78%) |

The (*) sign shows median, minimum and maximum values. AST - aspartate transaminase; ALT - alanine transaminase; BMI - body mass index; IVF - in vitro fertilization, SD - standard deviation

hyperstimulation therapy ($p$=0.235, 0.480, and 0.100, respectively) (Table 2). The PWV was significantly lower after COH than at baseline [5.3 m/s (4.5–6.9 m/s) versus 5.4 m/s (4.7–7.3 m/s); $p$<0.001], but the AIx between groups were not significantly different [28% (4%–41%) versus 29% (5%–43%); $p$=0.090] (Table 2).

In comparing the baseline descriptive characteristics and laboratory parameters of the two groups created based on achieving pregnancy, results showed that cigarette use was higher in the pregnancy (-) group than in the pregnancy (+) group ($p$=0.020). No other significant differences were noted between the groups (Table 3). The arterial stiffness and central hemodynamic parameters before and after COH therapy were not significantly different between the pregnancy (+) and pregnancy (-) groups ($p$>0.05) (Table 4).

## Discussion

For this study, an oscillometric method was used to assess arterial stiffness in IVF patients who underwent COH therapy, and the result demonstrated a significant decrease after treatment. To our knowledge, no research has been conducted that directly evaluated the influence of a supraphysiological estrogen level on the arterial stiffness and cardiac hemodynamics.

Arterial stiffness, a marker of endothelial dysfunction, can be used in estimating cardiovascular risk and early detection of vascular damage (4, 5, 13). It can be measured invasively and directly and estimated noninvasively by many different methods.

**Table 2. Distribution of arterial stiffness and central hemodynamic parameters before and after controlled ovarian hyperstimulation therapy**

| Variable | Before COH therapy (n=82) Mean ± SD Median (min.-max.)* | After COH therapy (n=82) Mean ± SD Median (min.-max.)* | *P* value |
|---|---|---|---|
| Systolic BP (mm Hg) | 116.5±10.6 | 113.5±9.2 | **0.001** |
| Diastolic BP (mm Hg) | 75.8±9.5 | 73.1±9.0 | **0.002** |
| Heart rate (beat/min) | 85.3±11.2 | 84.3±10.1 | 0.235 |
| Mean BP (mm Hg) | 94.3±9.8 | 91.7±8.2 | **<0.001** |
| Pulse pressure | 40.8±7.2 | 40.2±7.2 | 0.480 |
| cSystolic BP (mm Hg) | 111.9±10.06 | 108.4±9.6 | **<0.001** |
| cDiastolic BP (mm Hg) | 75.8±10.8 | 74.0±8.6 | **0.040** |
| Peripheral vascular resistance (sec*mm Hg/mL) | 1.3 (1.0-1.6)* | 1.2 (1.0-1.6)* | **<0.001** |
| Cardiac output (CO) (lt/min) | 4.5 (3.4-5.8)* | 4.5 (3.4-5.9)* | 0.100 |
| Augmentation index (%) | 29 (5-43)* | 28 (4-41)* | 0.090 |
| Pulse wave velocity (m/s) | 5.4 (4.7-7.3)* | 5.3 (4.5-6.9)* | **<0.001** |

The (*) sign shows median, minimum and maximum values. BP - blood pressure; c - central; COH - controlled ovarian hyperstimulation

**Table 3. Distribution of basic clinical and laboratory characteristics of patients with pregnancy (+) and pregnancy (-) after IVF therapy**

| Variable | Pregnancy (+) n=18 (22%) Mean ± SD Median (min-max.)* | Pregnancy (-) n=64 (78%) Mean ± SD Median (min-max.)* | *P* value |
|---|---|---|---|
| Age (year) | 33.3±3.8 | 33.5±5.9 | 0.870 |
| BMI (kg/m$^2$) | 25.4±3.6 | 25.9±3.9 | 0.680 |
| Cigarette, n (%) | 0 | 16 (25%) | **0.020** |
| Basal estradiol (pg/mL) | 44.2 (5-125)* | 33.2 (5-104)* | 0.240 |
| Peak estradiol (pg/mL) | 1229 (411-2352)* | 1166 (196-4160)* | 0.170 |
| Glucose (mg/dL) | 85 (77-121)* | 88 (65-122)* | 0.880 |
| Creatinine (mg/dL) | 0.53 (0.44-0.80)* | 0.58 (0.45-0.82)* | 0.062 |
| Hemoglobin (g/dL) | 13.4±1.1 | 13.4±1.0 | 0.820 |
| White blood cell (10$^3$/mL) | 7.7±2.1 | 7.7±1.8 | 0.890 |
| Platelet (10$^3$/ml) | 256±62.8 | 293±65.9 | 0.040 |
| AST (U/L) | 19 (13-24)* | 17 (10-97)* | 0.191 |
| ALT (U/L) | 13 (7-31)* | 14.2 (6-74)* | 0.674 |

The (*) sign shows median, minimum and maximum values. AST - aspartate transaminase; ALT - alanine transaminase; BMI - body mass index; IVF - in vitro fertilization, SD - standard deviation

PWV has been recognized as the gold standard for the evaluation of arterial stiffness. The AIx is another parameter that considers increased age and blood pressure (4, 13). An increase in PWV indicates arterial stiffening caused by long-term organic and functional changes in the vessel wall. However, PWV may change with short-term endothelial functional changes (12). The reliability and validity of Mobil-O-Graph arteriography system have been demonstrated in many previous studies, and owing to its easy, rapid, inexpensive, noninvasive technique, and it has become the best reproducible method for the assessment of large arterial stiffness. It gives the estimated PWV and hemodynamic parameters (4-10).

Experimental and epidemiological studies have shown that female sex hormones have beneficial influences on the cardiovascular system (11, 12). The vascular endothelium and smooth muscle cells are positively affected by estrogen, and possibly progesterone, so age-dependent vascular degeneration is postponed in women until menopause. The incidence of cardiovascular morbidities is greater after menopause, and hormone replacement therapy has positive and negative changes (11-16). One study found that the PWV was increased in postmenopausal women and could cause death due to coronary artery disease and stroke in the following 10–12 years (14). There is no consensus on the association of arterial stiffness with the phases of the menstrual cycle, menopause status, and pregnancy trimester (11, 13, 15-18). Some studies suggested that the central arterial stiffness does not change during the follicular and luteal phases of the menstrual cycle despite marked differ-

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**Table 4. Distribution of arterial stiffness and central hemodynamic parameters in patients with pregnancy (+) and pregnancy (-), before and after IVF therapy**

| Variables | Pregnancy (+) n=18 (22%) Mean ± SD Median (min.-max.)* Before COH After COH | Pregnancy (-) n=64 (78%) Mean ± SD Median (min.-max.)* Before COH After COH | *P* value |
|---|---|---|---|
| Systolic BP (mm Hg) | 115.7±10.8<br>115.9±10.0 | 116.7±10.6<br>112.9±9.0 | 0.732<br>0.233 |
| Diastolic BP (mm Hg) | 74.6±9.1<br>72.5±9.5 | 76.1±9.7<br>73.3±8.9 | 0.560<br>0.760 |
| Heart rate (beat/min) | 88.4±11.0<br>85.3±8.9 | 84.4±11.2<br>84.0±10.4 | 0.185<br>0.640 |
| Mean BP (mm Hg) | 93.0±9.2<br>92.6±8.9 | 94.7±9.9<br>91.5±8.0 | 0.522<br>0.620 |
| Pulse pressure | 41.9±8.5<br>41.7±8.0 | 40.5±6.9<br>39.7±6.9 | 0.480<br>0.302 |
| cSystolic BP (mm Hg) | 111.1±10.6<br>109.1±10.8 | 112.1±11.1<br>108.2±9.4 | 0.723<br>0.736 |
| cDiastolic BP (mm Hg) | 74.4±9.8<br>73.2±9.5 | 76.2±11.1<br>74.2±8.4 | 0.534<br>0.656 |
| Peripheral vascular resistance (sec*mm Hg/mL) | 1.2 (1.1-1.4)*<br>1.1 (1.0-1.4)* | 1.3 (1.0-1.6)*<br>1.2 (1.0-1.6)* | **0.054**<br>**0.024** |
| Cardiac output (lt/min) | 4.6 (3.4-5.6)*<br>4.6 (3.4-5.5)* | 4.5 (3.5-5.8)*<br>4.4 (3.4-5.9)* | 0.444<br>0.080 |
| Augmentation index (%) | 29 (17-38)*<br>27 (17-36)* | 30 (5-43)*<br>28 (4-41)* | 0.340<br>0.452 |
| Pulse wave velocity (m/s) | 5.4 (4.7-6.1)*<br>5.4 (4.7-6.2)* | 5.4 (4.7-7.3)*<br>5.3 (4.5-6.9)* | 0.920<br>0.286 |

The (*) sign shows median, minimum and maximum values. BP - blood pressure; c - central; COH - controlled ovarian hyperstimulation

ences in the levels of estradiol, progesterone, and renin-angiotensin-aldosterone system hormones (19, 20). The elevation of angiotensinogen and aldosterone during the luteal phase of the menstrual cycle may counteract the vasodilatation effects of estradiol and may be responsible for the lack of change in arterial stiffness between the phases of the menstrual cycle. Progesterone, in particular, increases the aldosterone level to inhibit the hormone at the mineralocorticoid receptor (17, 20). Arterial stiffness has been reported to increase from the second trimester to term and postnatally using brachial-ankle PWV, but other studies showed no change in PWV during gestation or a similar decrease in PWV and AIx during pregnancy (21, 22). Robb et al. (23) showed that despite the increased serum estradiol and progesterone levels in advanced gestation, arterial stiffness increases in the third trimester due to factors other than sex steroids.

A supraphysiological estradiol level has varied effects on the cardiovascular system (24). Yiginer et al. (1) observed that the supraphysiological estradiol level in COH patients was associated with an increase in the QTc interval, but not to a pathological level. In a study of female rats, PWV was used to evaluate arterial distensibility after estrogen replacement and results showed that estrogen replacement increased arterial stiffness with several regional effects on vasodilator functions (2). Von Wowern et al. (11) reported that digital pulse wave analysis detected no change in arterial stiffness during the follicular phase or in early pregnancy during COH for IVF treatment, but arterial stiffness was increased in the central and peripheral arteries during the early luteal phase, suggested to result from hormonal hemodynamic activation that reduces the influence of estrogen. Digital pulse wave analysis is another technique to evaluate arterial stiffness and endothelial function, which has been correlated to the tonometry variables of PWV and AIx (11, 19, 24). In comparing the cardiovascular functions of 42 children born to mothers with ovarian hyperstimulation syndrome, 34 children born to mothers with non-ovarian hyperstimulation syndrome and underwent IVF, and 48 children who were spontaneously born, another study determined that children born to ovarian hyperstimulated women showed markedly impaired cardiac diastolic function and endothelial function because of the influences of supraphysiological estradiol and progesterone (25).

In our study, although the PWV was significantly lower after COH therapy, there was no statistically significant variation in the AIx values, possibly due to the absence of a change in cardiac output. In our study, the SBP, DBP, MAP, cSBP, cDBP, and PVR measurements were statistically significantly lower after COH therapy values than those before treatment. There was no statistically significant difference in the heart rate, PP, or CO. This supports earlier evidence that the vasodilator effects of a supraphysiological estrogen level are different from the normal menstrual cycle. Again, the estrogen values observed in hormone replacement therapy, often used in menopause, are not supraphysiological and the same effects are not seen. The differences in the parameters at baseline and after COH treatment in those who had pregnancy (+) or pregnancy (-) with IVF therapy were assessed. In the retrospective evaluation, when the patients were grouped in terms of achieving pregnancy with IVF therapy, did arterial stiffness parameters differ at baseline and after COH treatment? All parameters were not different between the pregnancy (+) and pregnancy (-) patients with before and after COH treatment.

### Study limitations

The major limitation of this study is that long-term effects of decreased arterial stiffness following COH therapy could not be assessed. In addition, the number of patients in the study group was small. Finally, the prior use of assisted reproductive techniques other than IVF is unknown.

## Conclusion

Arterial stiffness and hemodynamic parameters significantly decreased in IVF patients who undergo COH therapy. However,

when the patients were grouped based on achieving pregnancy, arterial stiffness and central hemodynamic parameters were not statistically different between pregnancy (+) and pregnancy (-) patients before and after IVF therapy. The long-term effects of this short-term supraphysiological estrogen levels on cardiovascular system should be investigated with prospective studies.

**Conflict of interest:** None declared.

**Peer-review:** Externally peer-reviewed.

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