

Exercise modulates circulating adipokine levels in hypertrophic cardiomyopathy

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KEY WORDS

adiponectin,
hypertrophic
cardiomyopathy

ABSTRACT

INTRODUCTION Studies conducted so far have shown that patients with left ventricular hypertrophy have increased adiponectin levels; however, these studies were performed only in resting condition.

OBJECTIVES The aim of the study was to compare adiponectin, resistin, and leptin levels (at rest and after exercise) between patients with hypertrophic cardiomyopathy (HCM) and healthy controls. Additionally, we examined potential relationships between the levels of the 3 adipokines and the left ventricular outflow tract (LVOT) gradient both at rest and at peak exercise.

PATIENTS AND METHODS We studied 29 patients with HCM (mean age 42.7 ± 11.9 years, 16 men and 13 women). The control group included 19 healthy subjects matched for age-, sex-, and the body mass index. After echocardiographic examination in a supine position, all patients were placed in an upright position and treadmill exercise test was performed with simultaneous continuous echocardiographic monitoring of the LVOT gradient. Adiponectin, resistin, and leptin levels were measured immediately prior to exercise in a supine position and at peak exercise in an upright position.

RESULTS At baseline (at rest), adiponectin levels tended to be increased in patients with HCM ($P = 0.09$), while resistin and leptin levels were significantly higher in patients with HCM than in healthy controls. In HCM patients, exercise induced an increase in adiponectin (20.74 ± 7.95 vs. 22.52 ± 8.10 $\mu\text{g/ml}$, $P < 0.05$), a decrease in leptin (22.78 ± 6.08 vs. 20.63 ± 5.57 ng/ml , $P < 0.05$), and no significant effect on resistin. In the control group, all biomarkers mildly decreased at peak exercise (adiponectin 19.23 ± 5.43 vs. 18.66 ± 5.31 $\mu\text{g/ml}$, $P = 0.044$), resistin 14.80 ± 1.87 vs. 13.87 ± 1.21 ng/ml , $P = 0.030$), (leptin 14.34 ± 4.74 vs. 13.50 ± 3.98 ng/ml , $P = 0.045$). In HCM patients, neither resting nor peak exercise values of the LVOT gradient correlated with any levels of adipokines.

CONCLUSIONS In patients with HCM, but not in healthy individuals, moderate exercise induced an increase in adiponectin levels and a decrease in leptin levels independent of a rise in the LVOT gradient. The potential cardioprotective role of adiponectin during exercise stress in HCM requires further studies.

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INTRODUCTION Adiponectin is an adipocyte-derived cytokine that is abundantly present in human plasma. Increased levels of adiponectin have been reported in patients with left ventricular (LV) hypertrophy and heart failure.¹⁻³ Adiponectin has been shown to be a potent modulator of hypertrophic signals in the heart.¹ Moreover, hyperadiponectinemia is associated with increased severity of ventricular dysfunction in congestive heart failure.⁴

It has been reported that increased adiponectin is associated with diastolic⁵ or systolic⁶ LV

dysfunction in hypertrophic cardiomyopathy (HCM). In nonobstructive HCM with preserved systolic function, higher adiponectin levels were correlated with diastolic LV dysfunction measured invasively.⁴ In a larger, heterogeneous group (both nonobstructive and obstructive, normal and decreased systolic function), higher adiponectin levels were associated with decreased LV contractility.⁵ However, in these 2 studies, adiponectin was measured only in the Japanese patients with HCM, and it is unclear whether the results in that population could be extrapolated to the European

patients with HCM. As regards the potential role of the LV outflow tract (LVOT) gradient in the regulation of adipokine metabolism, in the study by Unno et al.⁵ only nonobstructive patients were examined, while Kitaoka et al.⁶ analyzed a very low percentage of patients with obstructive form of HCM (below 10%) and a significant proportion (nearly 25%) of patients had apical form of HCM, which was uncommon in the European patients. To our knowledge, there have been no reports on plasma leptin levels in HCM and only 1 report on resistin.⁷ Therefore, the aim of the current study (in patients without apical hypertrophy and without LV systolic dysfunction that may depress the LVOT gradient) was:

- 1 to compare the resting and exercise levels of adiponectin, resistin, and leptin between patients with HCM and healthy controls
- 2 to correlate the levels of the 3 adipokines with the LVOT gradient both at rest and at peak exercise.

PATIENTS AND METHODS We studied 29 patients with HCM (mean age 42.7 ± 11.9 years; 16 men and 13 women). Patients were diagnosed with HCM on the basis of typical clinical, echocardiographic, and hemodynamic features.⁸ The cardiac exclusion criteria were as follows: concomitant atrial fibrillation, valvular heart disease, previous myocardial infarction (natural or induced by alcohol septal ablation), and history of systemic hypertension. Noncardiac “metabolic” exclusion criteria were acute illness, cancer, renal or hepatic dysfunction, diabetes or metabolic syndrome.

In resting supine echocardiographic examination, the severity and distribution of LV hypertrophy were assessed and maximal LV wall thickness was detected at septum in all patients. Based on echocardiographic data, no patients with LV cavity dilatation or depressed LV contractility or apical form of HCM were included in the study. Only patients without resting LVOT obstruction were included into exercise study. At peak exercise, a significant LVOT obstruction was detected in 9 patients.

The University Ethical Committee approved the study, and all study participants gave their informed consent.

The control group included 19 subjects matched for age, sex, and the body mass index (BMI).

Echocardiographic study Transthoracic echocardiographic examination was performed, and in each patient M-mode and 2-dimensional echocardiograms were obtained, followed by pulsed and continuous-wave Doppler ultrasound examinations. Conventional techniques were used for the measurement of the LV size. LV contractility was assessed by fractional shortening. The use of any calculation formula based on geometrical assumption (elipic LV shape) are inadequate in HCM, in which the LV cavity is very irregular. The measure of fractional shortening is pre-

ferred in HCM patients as shown in an important, previously conducted study.⁹

The LVOT gradient was measured using continuous wave Doppler ultrasound. Care was taken to report only those gradients derived from Doppler velocity profiles typical of subaortic obstruction that avoided contamination by the mitral regurgitation jet.

After supine examinations, all patients were placed in an upright position and treadmill exercise test was performed using the modified Bruce protocol, and LVOT gradients were continuously monitored echocardiographically. Patients were exercised only to a moderate level of workload, since we wanted to obtain an optimal quality of the Doppler signal.¹⁰ According to a previously tested protocol,¹⁰ the exercise was stopped at 8 minutes or earlier if patients were unable to continue exercise (dyspnea in 10 patients). At peak exercise, the LVOT gradient was measured as the only monitored echocardiographic parameter.

For adequate comparison, healthy controls were exercised to a similar level of workload (exercise was stopped at 8 min). The peak exercise heart rate was similar in HCM patients and controls. No symptoms during exercise were reported by control subjects.

Blood samples were taken between 8 a.m. and 9 a.m. (after an overnight fast) at rest and immediately after peak exercise echocardiography. Adiponectin, leptin, and resistin levels were measured using Bio-Rad Luminex flow cytometry (Millepore, Billerica, Massachusetts, United States). Coefficients of variation for the 3 analytes ranged from 6.5% to 10%.

Statistical analysis Data were given as the mean $\pm$ standard deviation. The Kolmogorov-Smirnov test was used to assess conformity with a normal distribution. The Student's *t*-test was used to test differences. Relationships between variables of interest were examined with Pearson's correlation analyses. $P < 0.05$ was considered significant.

RESULTS Demographic and clinical characteristics of the study patients and controls were similar (TABLE 1). The peak exercise heart rate was comparable between HCM patients and controls.

In echocardiograms recorded in resting condition, septum at end-diastole was significantly thickened (22.65 ± 3.38 mm). LV cavity size was normal (LV end-diastolic diameter 42.93 ± 6.58 mm, LV end-systolic diameter 24.13 ± 5.18 mm). The LV contractility was preserved (fractional shortening $43.2\% \pm 6.8\%$). The resting LVOT gradient increased significantly at peak exercise in patients with HCM (11.96 ± 8.09 vs. 26.93 ± 19.67 mmHg, $P < 0.0001$).

At baseline (at rest), the adiponectin level tended to be increased in patients with HCM ($P = 0.09$), while resistin and leptin levels were significantly higher in patients with HCM than in controls. The baseline levels and effect of exercise in both

TABLE 1 Baseline characteristics of the study group

	HCM	Healthy controls	<i>P</i>
age	42.7 ± 11.9	40.2 ± 8.9	0.43
sex, n (% men)	15 (55)	11 (58)	0.84
BMI, kg/m ²	24.7 ± 3.4	25.1 ± 3.2	0.68
diabetes mellitus, n (%)	0	0	
hypertension, n (%)	0	0	
current smoking, n (%)	2 (6.8)	1 (5.2)	0.82
cardiovascular treatment			
calcium antagonists	10	0	
β-blockers	9	0	
without treatment	10	19	

Abbreviations: BMI – body mass index, HCM – hypertrophic cardiomyopathy

TABLE 2 Effect of exercise on adipokines

	Baseline rest	Post exercise	<i>P</i>
HCM patients			
adiponectin, µg/ml	20.74 ± 7.95	22.52 ± 8.10	0.00002
resistin, ng/ml	16.65 ± 2.58	16.61 ± 2.28	0.94
leptin, ng/ml	22.78 ± 6.08	20.63 ± 5.57	0.036
healthy subjects			
adiponectin, µg/ml	19.23 ± 5.43	18.66 ± 5.31	0.044
resistin, ng/ml	14.80 ± 1.87	13.87 ± 1.21	0.030
leptin, ng/ml	14.34 ± 4.74	13.50 ± 3.98	0.045

Abbreviations: see [TABLE 1](#)

groups are presented in [TABLE 2](#) and [FIGURES 1, 2](#), and [3](#).

Neither the resting or peak exercise values of the LVOT gradient correlated with any levels of adipocytokines. The percentage increase of the LVOT gradient during exercise did not correlate with the percentage change of any level of adipokines. There were no associations between adipokines and other echocardiographic variables or LV dimensions, or between fractional shortening

and adipokines. Septum thickness was not associated with the 3 adipokines. In HCM, only adiponectin was correlated with age (strong negative correlations: $r = -0.86$, $P < 0.0001$).

DISCUSSION The current study has been the first to show that moderate exercise induces an increase in adiponectin levels and a decrease in leptin levels independent of a rise in the LVOT gradient in HCM patients.

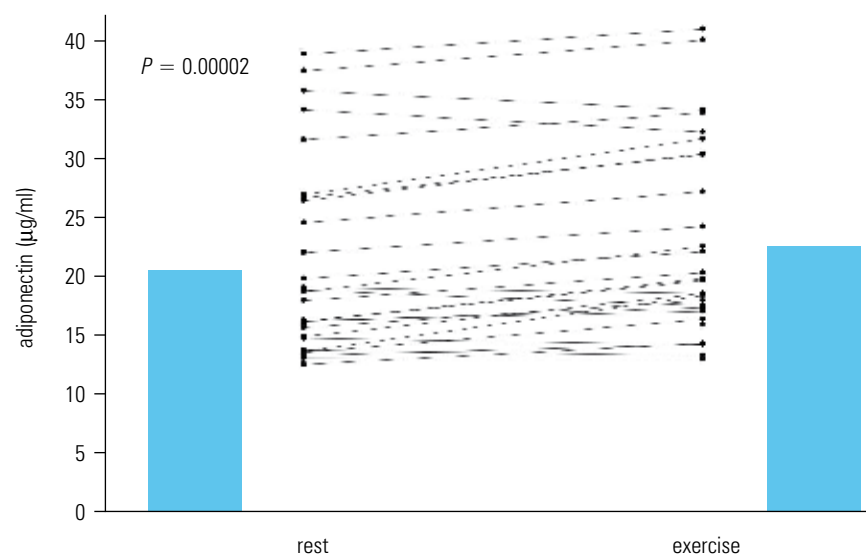
FIGURE 1 Comparison of adiponectin levels at rest vs. exercise in patients with hypertrophic cardiomyopathy

FIGURE 2 Comparison of resistin levels at rest vs. exercise in patients with hypertrophic cardiomyopathy

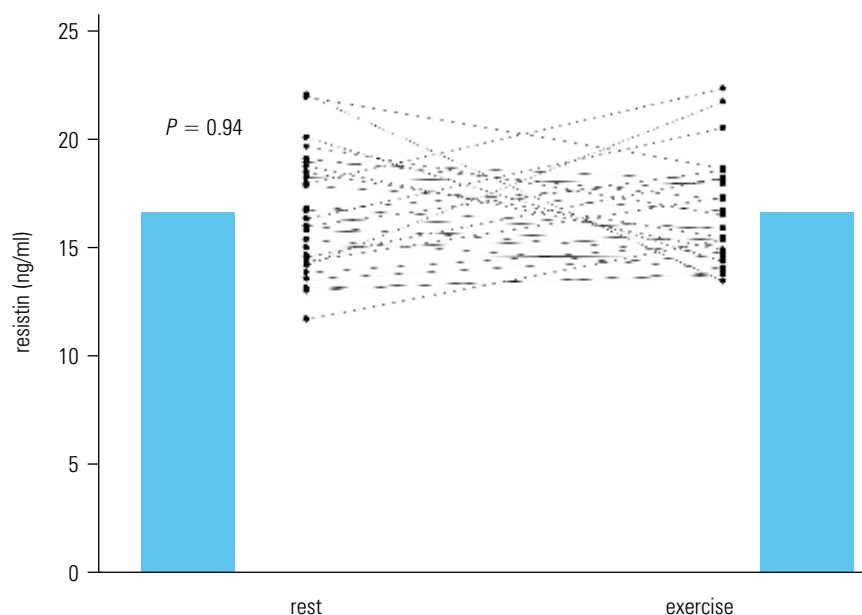
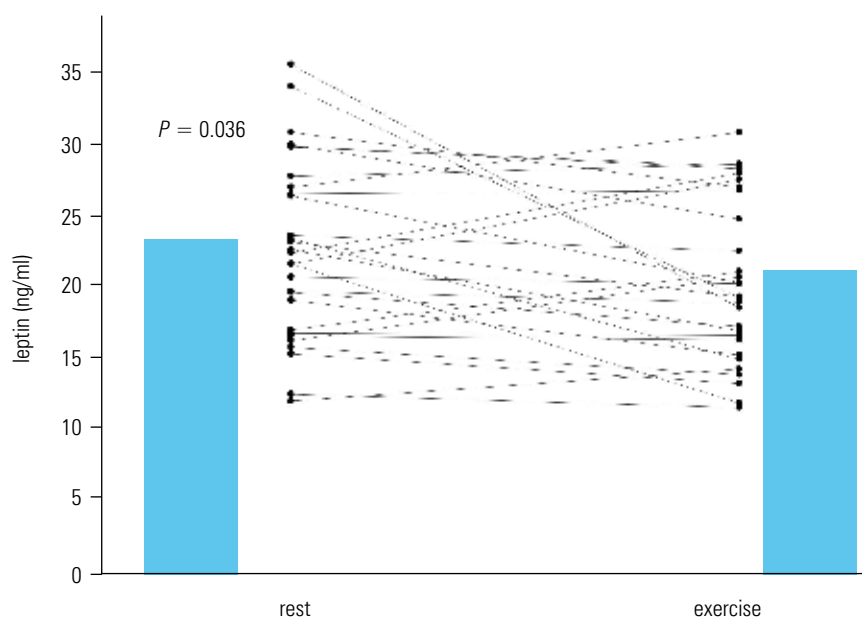


FIGURE 3 Comparison of leptin levels at rest vs. exercise in patients with hypertrophic cardiomyopathy



Monitoring the dynamic concentration changes of adipokine may be useful for clinical practice in several cardiovascular diseases.^{2-4,11-13} Pathological characteristics of HCM involve a number of various mechanisms. Consequently, measurements of biomarkers may be useful to assess pathophysiological pathways and disease severity.^{14,15} In a previous study,⁷ it has been shown that increased resistin levels at rest were significantly associated with HCM. Kitaoka et al.⁶ have reported that LVOT obstruction is not correlated with adiponectin; however, obstructive subgroup in their study was relatively small in number. In the present study, we decided to explore this problem more comprehensively by extended analysis of adipokine changes during exercise as a provocative test for an increase in the LVOT gradient. In our measurements (with dynamic stress test), the lack of relation between adipokines and the LVOT gradient was confirmed for the European patients.

Protective role of adiponectin Leptin, adiponectin, apelin, and visfatin have all been shown to protect against ischemia/reperfusion injury and pressure overload. Experimental findings have shown that adiponectin has beneficial effects in the cardiovascular system by directly acting on the component cells of the heart and blood vessels.¹⁶

In a recent study in HCM patients,⁵ high adiponectin levels correlated with LV diastolic dysfunction, which may be associated with impaired utilization of adiponectin in the heart. Accordingly, Shibata et al.¹⁷ have demonstrated in experiments on mice that adiponectin protects the heart from injury by accumulating in tissues subject to ischemic damage through leakage from the vascular compartment.

Methodological consideration The intergroup similarities of the baseline findings are important from a methodological point of view.

In a study by Norrelund et al.,¹⁸ the effect of exercise on adiponectin was assessed in upright moderate exercise (7–10 min), and patients with heart failure were compared with healthy controls. In the current study, the exercise was also performed in an upright position and the exercise time was similar, and, importantly, both patients with HCM and controls were exercised into a comparable level. Additionally, both groups were comparable in terms of age, sex, and the BMI (important factors affecting adipokines).

The effect of short-term (acute exposure) was measured in healthy subjects only in a few studies, and usually exercise tests were longer and more stressed than in our protocol, and the effect on adiponectin was neutral.^{19,20} In one study with continuous and progressively intense intermittent exercise (27 minutes and 2 last minutes with maximal exercise), adiponectin was slightly increased at peak exercise.²¹

In a very short acute exercise, adiponectin and leptin response to exercise may be different between men and women.²²

Most recently, it has been reported²³ that adipokine levels may be modulated even by a very short passive stimulus (warm-water bath).

In subjects without cardiovascular diseases, acute exercise induces a striking short-term increase in adipose tissue interstitial adiponectin concentration in both overweight and lean subjects, a decrease in adipose tissue adiponectin mRNA, and no major changes in plasma adiponectin concentration.²⁴ It is possible that adiponectin has an autocrine/paracrine function in the adipose tissue during exercise.

In an ideal stress test for LVOT gradient provocation, maximum stressor would combine high inotropic stimulation with preload reduction.²⁵ Measuring the LVOT gradient in an upright position at peak exercise may be a more potent provocation and be more physiologic than in another stress test.^{25,26} Upright exercise stress echocardiography induces a significant LVOT gradient and is an optimal stress test for modification of pharmacotherapy.^{27,28}

Methodological aspects of adiponectin measurement Some differences in adiponectin levels between the current and previous studies might have resulted from the use of different methods to determine this parameter. An enzyme-linked immunosorbent assay (ELISA), the most common method used in the previous studies, involves lower concentrations of adipokines. We applied a recently introduced assay based on the BioRad Luminox system. Our results obtained in the control group were similar to those reported in another study that also used this method.^{13,29}

Limitation of the study Our study has several limitations. First, the number of patients enrolled in this study was small, mostly owing to numerous exclusion criteria. This resulted in a quite homogeneous population sample, which together with

a well-matched control group mitigates against significant recruitment bias.

At peak exercise, only 1 echocardiographic parameter was measurable (i.e., the LVOT gradient). Therefore, the assessment of diastolic function was not possible. We need further study with invasive assessment of LV diastolic dysfunction during exercise as previously reported^{30,31} in relation to adiponectin changes. According to Geske et al.,³² only invasive LV diastolic assessment is valid because precise characterization of LV filling pressure in HCM patients cannot be determined with the use of these noninvasive parameters.

Moderate exercise instead of maximal symptom-limited exercise was another limitation. Exercise beyond usual activity (e.g., to the higher value of heart rate in nonphysiological exercise) was not performed because measurement is difficult at peak exercise with high heart rate gradient. Additionally, Joshi et al.³³ suggested that symptom-limited exercise is rarely performed by patients with heart disease; thus, achieving 85% of predicted heart rate on the Bruce protocol is, for most, an artificial situation. Consequently, in the current study, natural (real-life) moderate exercise has been preferred to maximal/submaximal nonphysiological effort, which is probably very rarely achieved in normal daily activity. This approach may be useful in the diagnosis of syncope – a serious complication of HCM.³⁴

Conclusions and perspectives In patients with HCM, moderate exercise induces an increase in adiponectin levels and a decrease in leptin levels independent of a rise in the LVOT gradient. The potential role of adiponectin as a cardioprotective substance during exercise stress in HCM requires further studies with precise ischemia detection using myocardial exercise scintigraphy.

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Wysiłek modyfikuje stężenia adipokin u pacjentów z kardiomiopatią przerostową

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SŁOWA KLUCZOWE

adiponektyna,
kardiomiopatia
przerostowa

STRESZCZENIE

WPROWADZENIE Z przeprowadzonych dotąd badań wynika, że u chorych z przerostem mięśnia sercowego stężenie adiponektyny jest zwiększone, jednak badania te prowadzono jedynie w warunkach spoczynkowych.

CELE Celem badania było porównanie wartości stężeń adiponektyny, rezystyny i leptyny (spoczynkowych i po próbie wysiłkowej) u chorych z kardiomiopatią przerostową (*hypertrophic cardiomyopathy* – HCM) i grupą kontrolną osób zdrowych. Dodatkowo przeanalizowano ewentualne korelacje pomiędzy 3 adipokinami i gradientem w drodze odpływu lewej komory (*left ventricular outflow tract* – LVOT) zarówno w spoczynku, jak i w szczytowym momencie wysiłku.

PACJENCI I METODY Przebadano 29 chorych z HCM (średni wiek: $42,7 \pm 11,9$ roku, 16 mężczyzn i 13 kobiet). Grupę kontrolną stanowiło 19 zdrowych osób dobranych pod względem wieku, płci oraz indeksu masy ciała. Po badaniu echokardiograficznym w pozycji leżącej wszystkich chorych poddano testowi wysiłkowemu w pozycji pionowej (na bieżni) z jednoczesnym ciągłym echokardiograficznym monitorowaniem gradientu LVOT. Stężenie adipokin mierzono tuż przed wysiłkiem w pozycji leżącej i tuż po wysiłku w pozycji stojącej.

WYNIKI W badaniu spoczynkowym stężenie adiponektyny wykazywało podwyższony trend ($p = 0,09$) natomiast stężenie rezystyny i leptyny było istotnie zwiększone u chorych z HCM w porównaniu z grupą kontrolną. Próba wysiłkowa u chorych z HCM spowodowała zwiększenie stężenia adiponektyny ($20,74 \pm 7,95$ vs $22,52 \pm 8,10$ $\mu\text{g/ml}$; $p < 0,05$), zmniejszenie stężenia leptyny ($22,78 \pm 6,08$ vs $20,63 \pm 5,57$ mg/ml ; $p < 0,05$) oraz nie miała istotnego wpływu na rezystynę. W grupie kontrolnej stężenie wszystkich 3 biomarkerów uległo redukcji (adiponektyna $19,23 \pm 5,43$ vs $18,66 \pm 5,31$ $\mu\text{g/ml}$ [$p = 0,044$]), rezystyna $14,80 \pm 1,87$ vs $13,87 \pm 1,21$ [$p = 0,030$]), leptyna $14,34 \pm 4,74$ vs $13,50 \pm 3,98$ mg/ml [$p = 0,045$]). U chorych z HCM spoczynkowy i wysiłkowy gradient nie korelował ze stężeniem żadnej z adipokin.

WNIOSKI U chorych z HCM, w porównaniu z grupą kontrolną, umiarkowany wysiłek powodował zwiększenie stężenia adiponektyny i zmniejszenie stężenia leptyny niezależnie od zwiększenia gradientu LVOT. Potencjalna kardioprotekcyjna rola adiponektyny podczas obciążenia wysiłkowego w HCM wymaga dalszych badań.

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Uzyskasz dostęp do małego podręcznika „Choroby wewnętrzne” zawierającego informacje o: **objawach:** ataksja (niezbornosć), biegunka, ból brzucha, bóle głowy, ból w klatce piersiowej, ból ucha, chrypka, czkawka, zaburzenia czucia, drgawki, duszność, dysfagia, dyspepsja, ginekomastia, gorączka o nieznanej przyczynie (...); **chorobach:** choroby układu krążenia, choroby układu oddechowego, choroby przewodu pokarmowego, choroby trzustki, choroby pęcherzyka żółciowego (...); **pierwszej pomocy w urazach i innych stanach nagłych:** ogólne zasady udzielania pierwszej pomocy w nagłych wypadkach, utrata przytomności, omdlenia i inne krótkotrwałe utraty przytomności, śpiączka, zadławienie, rana i krwotok pourazowy (...); **zabiegach diagnostycznych i leczniczych:** wstrzyknięcia leków i innych środków, przygotowanie pola operacyjnego do drobnych zabiegów, znieczulenie miejscowe nasękowe, sedacja i krótkotrwałe dożylnie znieczulenie (...); **badaniach czynnościowych:** elektrokardiografia, pomiary ciśnienia tętniczego, pulsoksymetria, badania czynnościowe układu oddechowego; **badaniach endoskopowych:** bronchoskopia, endoskopia przewodu pokarmowego; **badaniach laboratoryjnych:** badania biochemiczne, hematologiczne i koagulologiczne, badanie ogólne płynu mózgowo-rdzeniowego, badanie ogólne moczu; **diagnostyce mikrobiologicznej:** diagnostyka zakażeń bakteryjnych, diagnostyka zakażeń wirusowych, diagnostyka zarażeń grzybiczych, diagnostyka zarażeń pasożytniczych; **stanach nagłych** (...)



Mały podręcznik „Choroby wewnętrzne” już dostępny na telefony i tablety z systemem Android, wkrótce na iPhone i iPady.



Podręczniki



Czasopisma



Leki



Gabinet

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