Chapter

Features of Diagnostics and Differential Diagnostics of Chronic Heart Failure in Outpatient Clinics

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Abstract

The Chapter contains information about the prevalence of heart failure (HF) among patients in outpatient practice. The causal structure of HF, the prevalence of risk factors for HF, and the occurrence of a reduced ejection fraction are described. It describes the frequency of overdiagnosis of HF, the disease most often simulating its symptoms. The difficulties associated with laboratory and instrumental diagnostics of this syndrome are discussed. A pharmacological test for differential diagnosis of the causes of dyspnea in patients with suspected HF is described. Information is provided on the incidence of depressive and anxiety among the patients with this disease.

Keywords: heart failure, prevalence, outpatient, diagnostics, differential diagnostics, pharmacological test, torasemide, depressive, anxiety

1. Introduction

Chronic HF is a common complication of cardiovascular diseases. The widespread prevalence of this pathology in the world in recent years has taken on the scale of an epidemic. Tens of millions of people around the world suffer from this disease. It can be made with the growing burden of obesity-related diseases and with the aging of the population [1]. Probably, it is also important to increase the survival rate after acute forms of coronary artery disease and increase the life expectancy of patients with HF. Diagnosis of chronic HF is a major clinical problem, especially in patients with comorbidities [2]. This Chapter aims to highlight the features of the diagnosis of HF in an outpatient setting.

2. The prevalence of heart failure among patients in outpatient practice

The reasons for visiting a polyclinic by patients are often acute diseases, the need for a health examination, or dispensary monitoring for chronic diseases. The cohort of such visitors does not accurately reflect the state of the population, but these are the people that outpatient doctors have to deal with.

Many variants of criteria for the diagnosis of heart failure in epidemiological studies have been developed. Their diversity highlights the inferiority of diagnostic methods. There are Framingham, Gothenburg, Boston, European society of cardiology criteria and others. We used a questionnaire proposed by the Russian society of heart failure specialists [3]. The questionnaire includes a question about the presence of shortness of breath, confirmation of myocardial damage by instrumental methods, taking diuretics, a known diagnosis of heart failure, and a decrease in the left ventricular ejection fraction of less than 50%. A positive answer to the first question and any subsequent question makes it likely that the patient has HF.

In a survey of 3,000 adults who consecutively applied to the outpatient clinic, it was found that 543 patients were suspected of heart failure. The recruitment of people in the study was carried out by age and gender groups identical to the population living in the city. Among people who visit an outpatient department, the clinical suspicion of heart failure among men is 12.3%, among women it reaches 22.9% [4]. At the same time, it is possible to confirm the presence of heart failure in 11.7% of outpatient patients. In 36.2% of outpatient visitors with suspected HF, the disease is not confirmed during further examination. These cases were 57% more common among women compared to men.

The specificity of the questionnaire used was 63.8%. Thus, after clinical screening for possible HF, additional methods should be used that exclude the situation of overdiagnosis.

The vast majority of patients (87%) have HF with preserved ejection fraction (EF). However, among hospitalized patients with heart failure, the ratio between preserved and reduced ejection fraction is approximately one to one [5].

3. The prevalence of risk factors of heart failure among patients in outpatient practice

An analysis of a sample of 3,000 outpatient visitors found that the prevalence of obesity among adult outpatient patients is 20% (95% CI: 18.6–21.4%). Obesity increased the likelihood of shortness of breath in men and women almost equally (the relative risk was 2.39 and 2.49, respectively). The prevalence of diabetes mellitus, according to the survey, among adult outpatient visitors is 5.9%. The prevalence of smoking and alcohol abuse among adult outpatient visitors is 35% and 12.4%, respectively. The prevalence of MI among adult outpatient patients is 4.7%. According to the survey, the prevalence of hypertension among adult outpatient visitors was 37.9%.

Hence, among the risk factors, arterial hypertension has the greatest population contribution to the development of HF. It was found that not only the presence and severity of arterial hypertension affected the development of HF, but also the duration of increased blood pressure. Thus, in patients with confirmed HF, dyspnea developed on average only 8.4 years after the onset of hypertension, and in patients with excluded HF, dyspnea occurred only 2.8 years later. That is, when assessing the probability of a connection between the appearance of shortness of breath, the exposure to hypertension should be taken into account.

4. The clinical diagnosis of heart failure

The diagnosis of chronic HF begins, of course, with the patient's complaints. Thanks to the existing complaints of the patient that he comes into our field of vision. However, the complaints are not specific. If the patient has a complex of

manifestations of heart failure such as oedema, orthopnea, complaints of dyspnea, heaviness in the right hypochondrium, fatigue, then we will not be difficult to diagnose HF. But if we consider each of the signs of heart failure separately, it turns out that their specificity is quite low. And just such a situation is observed in the initial manifestations of heart failure. These patients, who have 1–2 manifestations, are the cornerstone of the diagnosis of initial forms of heart failure.

The most common manifestation of chronic heart failure is a complaint of shortness of breath. The sensitivity of this sign is close to 100%, but the specificity is only 17% [6]. When dyspnea is difficult for the patient to inhale, it has mostly inspiratory character. The shortness of breath is quite stereotypical. It cannot be there for a while, then disappear and reappear spontaneously. Without a treatment it constantly progresses as a rule. In patients with limited mobility, dyspnea may not appear during exercise, but may debut at night in a horizontal position. Short-acting nitrates and diuretics can have a good effect on dyspnea of cardiac origin. The dyspnea increases as the intensity of the cause increases. For example, an increase of blood pressure or the appearance of paroxysms of atrial fibrillation or onset of angina pectoris usually cause an increase in dyspnea. When shortness of breath increases, a cough is added, first dry, then with foamy sputum and blood. This may occur during a period of inadequate physical activity of the patient. If shortness of breath manifests at rest, then there is more accurate sign of heart failure: shortness of breath becomes heavier in a horizontal position and is relieved when the head end of the trunk is raised. The patient's forced sitting position is called orthopnea. The analysis of these features of dyspnea allows for more accurate diagnosis of heart failure.

Bendopnea, described in recent years [7], is the occurrence of shortness of breath after bending the patient sitting in a chair and pressing the abdominal belt. Shortness of breath in a patient with CHF persists for at least 30 seconds. However, this sign is not specific enough and can be observed in lung diseases and obesity [8].

Another sign of heart failure is rapid fatigue during physical activity and longer recovery after exercise. Fatigue is observed in about 85% of patients with HF [9]. The appearance of this symptom is associated with a violation of nutrition and structural adjustment of muscle tissue in patients, but may be due to hypovolemia and hypokalemia due to the use of diuretics. The specificity of the sign is extremely low, but good performance quite accurately indicates the absence of heart failure in the patient. Weakness in heart failure cannot be reduced by short-term training. In the initial stages of the disease, the patient does not feel weak at rest. It occurs only during physical activity. The patient is able to withstand short-term quite intense physical activity and at the same time gets very tired with low-intensity, but prolonged exercise. But even with these features, fatigue is a low-specific sign of heart failure.

The appearance of heaviness in the right hypochondrium indicates an increase in the liver due to stagnation in the large circle of blood circulation. The widespread pathology of the gallbladder makes this clinical sign very non-specific.

Oedema in patients with heart failure starts from the lower part of the legs and gradually involves the upper parts. In bedridden patients, edema forms on the sacrum. Oedema has a dense consistency. Their prolonged existence leads to hyperpigmentation of the skin and trophic changes in the area of oedema.

The patient's complaints must be coordinated with the physical examination data. Low exercise tolerance should correlate with the appearance of signs of congestion. The process of congestion, as a rule, from the beginning involves a small circle of blood, and for a long time during auscultation of the lungs we do not find wheezing. As a rule, wheezing in the lungs appears when the patient has shortness of breath at rest or with minimal physical activity. With long-term heart failure, a patient with wheezing lungs may not have shortness of breath at rest. Wheezing

appears from the lower parts of the lungs. They are moist, small-bubbly, in the beginning not sound, but with the appearance of fibrosis of the lungs they become resounding and do not respond to treatment with diuretics.

The formation of congestion in a large circle of blood circulation is manifested by an increase and soreness of the liver. When pressing on the liver during deep breathing of the patient, you can see an increase in blood filling of the neck veins by >3 cm sustained during 10 s (hepatojugular reflux), with an abrupt fall after the pressure is released [10]. This proves the connection between an enlarged liver and increased pressure in the veins of the large circle of blood. The appearance of tricuspid insufficiency is accompanied by the occurrence of pulsation of the neck veins. Due to the decline in the contractility of the right ventricle, the patient has a decrease in shortness of breath, but an increase in weakness.

Oedema of the lower extremities sometimes precedes the appearance of hepatomegaly, but it also happens the other way around. This depends only on the innate predisposition to leg swelling and also from the patient's position. Oedema is dense in consistency, appearing first in the lower areas, then rising to the top. With long-term oedema in the area of their localization, skin atrophy occurs, hyperpigmentation appears, and the skin becomes easily vulnerable.

The third heart tone occurs when the pressure of filling the left ventricle in the diastole is increased. Listening to the third heart tone is specific for heart failure, but it is extremely rare among outpatients.

If there is a suspicion of heart failure in the patient, you need to go upstream. In other words, it is necessary to determine the probability of developing heart failure in the patient. When communicating with the patient, you need to pay attention to the coincidence of manifestations of heart disease and possible manifestations of heart failure. In addition, it is important to determine the dependence of manifestations on heart disease. Frequent provocateurs of heart failure are uncontrolled arterial hypertension, paroxysms of fibrillation or atrial flutter, the development of acute coronary syndrome, alcohol abuse, sodium or fluid overload and the addition of infection.

To sum up, it should be noted that the accuracy of the diagnosis of HF based on clinical manifestations and physical examination data depends on the number of detected signs and symptoms, as well as on the chronological relationship with the cause of occurrence. Data analysis requires the use of clinical thinking.

5. Issues of differential diagnosis of signs and symptoms of HF

To begin differential diagnosis of manifestations similar to SN, you should always ask whether there is a reason for its development and whether the patient has a disease that can explain the manifestations. For example, if a patient has atrial fibrillation, the occurrence of shortness of breath after the development of a rhythm disorder should be explained by heart failure. And if a patient without heart disease has anemia, then shortness of breath is probably due to this cause.

But it is not necessary for a patient with heart disease to have shortness of breath only of cardiac origin. It should be mentioned once again that in every third case among outpatient visitors, who had a cardiovascular disease, HF is falsely diagnosed. And among women, this is 2 times more common than among men.

We found that most of the patients with cardiovascular disease and misdiagnosed heart failure suffer from anxiety disorders (53%). Second place is taken by obesity (39%). Then among the causes are decrepitude, pathologies of the respiratory system and anemia. There is no doubt that the patient may have different causes of symptoms.

For example, a patient with arterial hypertension and obesity may have severe anxiety and lack of training. Such a patient has a pathological circle, when anxiety for their health leads to the fact that they move less and eat more, and the increasing weight further restricts their physical abilities. These patients often have shortness of breath, oedema, enlarged liver, and arterial hypertension as a possible cause of HF. In addition, their EF remains normal, and NT-proBNP increases slightly even in the presence of HF. The cause of edema in them may be a violation of venous outflow due to obesity, or, perhaps, taking calcium antagonists, hepatomegaly is caused by non-alcoholic steatohepatitis, and shortness of breath is associated with a large body weight and lack of training.

Outpatient patients with heart failure usually have several comorbidities. So, on average, there are an additional 2.5 diseases per patient with HF. It should be noted that anxiety and depression are frequent companions of patients with HF. According to our data, 22% of outpatient patients with HF have depression, and 53% have severe anxiety.

It is necessary to distinguish between dyspnea that occurs with myocardial ischemia and dyspnea caused by HF, because dyspnea caused by ischemia is treated by restoring myocardial nutrition, and dyspnea in HF requires treatment of HF itself. If there are reasonable suspicions of an ischemic origin of dyspnea, then CT-coronary angiography should be performed.

How, then, can we distinguish HF from other causes of symptoms? To do this, you need to start treating the identified suspected cause of symptoms and evaluate the dynamics. In any case, this should be done, because if we only deal with HF without correcting comorbidities, we will not get a good clinical dynamic. But if the symptoms were associated only with extra-cardiac pathology, then we will get a brilliant result without the use of medications for HF.

6. Controversies in the use of natriuretic peptides for the verification of HF

It is known that in HF, an increase in the level of natriuretic peptides is observed in the blood. They are natural antagonists of the renin-angiotensin-aldosterone system. This is a weapon with which the patient's body resists the onslaught of neurohumoral activity in HF. However, it is very difficult to interpret the increase in the activity of these substances in a patient 2–3 times in comparison with the upper limit of the norm.

Thus, the level of brain natriuretic peptide (BNP) in obese patients is one and a half times lower compared to people with normal body weight despite a similar severity of HF [11]. In patients with liver cirrhosis, N-terminal precursor of brain natriuretic peptide (NT-proBNP) plasma level may increase five times in comparison with the control [12]. In patients with renal insufficiency, standards for the content of NT-proBNP have been developed depending on age [13].

The Association between NT-proBNP and the probability of developing atrial fibrillation [14] can be interpreted as an increase in the level of NT-proBNP in patients after silent paroxysms of atrial fibrillation. That is, the NT-proBNP level will indicate that the patient has suffered acute cardiac dysfunction, and not the presence of HF.

Thus, the abundance of parameters that affect the concentration of NT-proBNP does not allow us to make a decision about the presence or absence of HF with a small increase in its level. NTproBNP provided a higher negative predictive values (0.97) than BNP (0.87), but at lower positive predictive values (0.44 versus 0.59) [15].

In summary, it can be argued that the absence of an increase in NTproBNP can be used to exclude the diagnosis of HF, but an increase within 3 HGN should be interpreted very carefully.

Soluble ST2 receptor is a marker of the severity of fibrosis, remodeling, inflammation, and volume load on the heart. It can claim to be a more accurate marker of CH dynamics in comparison with NTproBNP [16].

On the other hand, the practitioner does not need a predictor of the developing deterioration of the patient's condition. The dynamics of body weight indicates an occurring fluid retention and the need to increase the dose of diuretics and search for the cause. And life-saving medications, such as renin-angiotensin-aldosterone system blockers, should be given at targeted doses that are independent of such markers.

7. Instrumental methods for diagnosing HF

Echocardiography is most important for confirming the diagnosis of HF as a screening method, since if EF ≤40%, the diagnosis can be considered proven. But there are few such patients in outpatient practice. Thus, most outpatient patients have diastolic heart failure. The diastolic insufficiency is the inability of the heart to accept all the blood that flows to it, while there is stagnation (increased pressure) in front of the heart chambers. This situation can occur only with physical activity, and in advanced cases also at rest.

Global longitudinal strain (GLS) is a more reproducible parameter regardless of echocardiographic training and image quality compared to EF [17]. As GLS ordinarily varies with age, sex, and LV loading conditions, defining abnormal GLS is not uncomplicated. However, in adults, GLS <16% is abnormal, GLS >18% is normal, and GLS 16–18% is borderline [18].

Traditionally, diastolic insufficiency is assessed by the ratio of the velocity of early transmitral filling (e) and the average early velocity of diastolic movement of the base of the mitral valve ring (e') [19]. It is an accurate, reliable and easily reproducible method for evaluating left ventricular diastolic function. This method does not require a sinus rhythm. In normal persons the E/e' ratio is <8. Values >14 have high specificity for increased LV filling pressures. In the range e/e' from 8 to 14, it is not possible to determine definitely the presence of HF. The accuracy of the indicator is reduced in severe mitral valve calcification, mitral valve defects, pericarditis and the presence of violations of regional contractility of the left ventricle.

Since the left atrium is also overloaded when the filling pressure of the left ventricle increases, it is important to detect the enlargement of the left atrium for the diagnosis of HF. However, it should be taken into account that the atrium can be expanded in athletes and with atrial fibrillation and flutter.

In symptomatic patients with normal levels of NTproBNP, no reduction in EF and signs of stagnation, as well as normal cardiac filling pressures at rest may have markedly abnormal hemodynamic responses during exercise [20]. An exercise pulmonary artery systolic pressure ≥ 45 mm Hg identified HF with 96% sensitivity and 95% specificity. However, such invasive tests are not acceptable for outpatient clinics.

If the etiology of HF is unclear, the need to assess the structures of the heart and surrounding tissues, assess the severity of fibrosis, differential diagnosis of inflammatory, accumulative and ischemic causes, the patient should perform cardiovascular magnetic resonance (CMR) tracking with or without an assessment of late gadolinium enhancement (LGE). A fast long-axis strain (FLAS) at end-systole of 12.3% and less predict the presence of HF [21]. The sensitivity and specificity of the method is 93% and 86%, respectively. This method is reproducible, reliable, and effective, but expensive.

8. Use of acute medication tests for the diagnosis of HF

It is known that the manifestation of congestion is best reversed against the background of the use of diuretics, while congestion in the lungs is quickly stopped by nitroglycerin. However, nitroglycerin is poorly tolerated by some patients, and it can also be effective for spasms of any origin, including bronchial asthma. Thus, the specificity of reducing shortness of breath after using nitroglycerin is not great. The use of diuretics seems to be a more effective way to detect the relationship of low exercise tolerance with fluid retention in the patient's body.

A sample with torasemide was developed for use in outpatient clinics for the differential diagnosis of dyspnea of cardiac and non-cardiac origin with a sensitivity of 89% and specificity of 82%.

Method of performing the test [22]. In the morning, all patients undergo a 6-minute walking test. Then the patient should take torasemide 5 mg. 6-minute walking test is repeated every other day, all other things being equal. The increase in walking distance should be at least 15 m.

The test with torasemide is not expensive and can be performed by any medical professional without special training. The test can be used as the first stage of differential diagnosis in an outpatient environment in a patient with cardiovascular disease and dyspnea of unknown origin.

9. Complex systems for assessing the probability of having HF

The newly created H2FPEF score allows you to accurately and quickly assess the probability of having HF in a patient [23]. Obesity (body mass index >30 kg/m^2), atrial fibrillation, age > 60 years, treatment with ≥2 antihypertensive drugs, E/e' > 9, and pulmonary artery systolic pressure > 35 mm Hg were associated with HF. Atrial fibrillation gives 3 points, obesity - 2 points, other signs-1 point each. If the scale value is 0–1, it is very likely to exclude SN, if it is 6 or more points, it confirms it. Values from 2 to 5 require the use of clarifying methods (**Figure 1**).

The 'HFA-PEFF diagnostic algorithm' is also proposed [24]. In accordance with this algorithm, a pre-test assessment of the probability of CH is first performed. For this purpose, the presence of such risk factors for HF as obesity, hypertension, diabetes mellitus, the elderly, and atrial fibrillation is evaluated. Routine laboratory tests (sodium, potassium, urea, and creatinine, liver function tests, HbA1c, thyroid stimulating hormone, full blood count, ferritin, transferrin saturation, and hemoglobin), electrocardiogram and echocardiography are performed. In the absence of an obvious extracardial cause of symptoms and the presence of risk factors for heart failure, even at normal levels of natriuretic peptides, HF should be suspected.

The next step is an in-depth echocardiographic study, including E/e', left atrial volume index, LV mass index, relative LV wall thickness, tricuspid regurgitation rate, global longitudinal LV systolic strain, and serum natriuretic peptide levels if not already done (**Table 1**). If the sum of points of the HFA–PEFF Score is 1 and less than points, the diagnosis of CH is considered unlikely, if 5 the diagnosis of CH is proven. Sum 2–4 requires a transition to the next stage of diagnostics.

At Step 3 is recommended an echocardiographic or invasive hemodynamic exercise stress tests. The HF criteria for performing stress echocardiography are the average E/e 'ratio at peak stress increases to ≥15, with or without a peak tricuspid regurgitation velocity of >3.4 m/s. If the above-mentioned signs are not detected during the exercise echocardiography, then an invasive test should have performed. An elevated LV filling pressures at rest (LVEDP ≥16 mmHg) or/and a high mean pulmonary capillary wedge pressure (mPCWP≥15 mmHg) at rest is confirmed HF.

	Clinical Variable	Values	Points
H_2	Heavy	Body mass index > 30 kg/m^2	2
	Hypertensive	2 or more antihypertensive medicines	1
F	Atrial Fibrillation	Paroxysmal or Persistent	3
P	Pulmonary Hypertension	Doppler Echocardiographic estimated Pulmonary Artery Systolic Pressure > 35 mmHg	1
E	Elder	Age > 60 years	1
F	Filling Pressure	Doppler Echocardiographic E/e' > 9	1
H_2FPEF score			Sum (0-9)

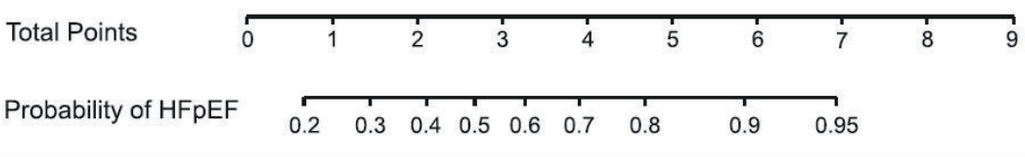

Figure 1.
The H2FPEF score with associated probability of having heart failure [23].

Measurement	Criterion	Points
A. Functional Measurement Domain		
e'	Age < 75 years:	
Peak early diastolic velocity of mitral annular motion (cm/s)	Septal e' < 7 or lateral e' < 10	2
	Age ≥ 75 years:	
	Septal e' < 5 or lateral e' < 7	2
E/e' as major criterion		
Peak early diastolic velocity of mitral inflow, divided by the mean value of e' recorded at the septal and lateral mitral annulus	E/e' ratio ≥ 15	2
Tricuspid regurgitation velocity (m/s)	Peak velocity > 2.8	2
E/e' as minor criterion	E/e' ratio 9–14	1
Global longitudinal strain (GLS) of the LV in systole (%, as positive value)	GLS < 16	1
B. Morphological Measurement Domain		
Left atrial volume index (ml/m2) as major criterion	Patient in sinus rhythm:	
	> 34	2
	Patient in atrial fibrillation: > 40	2
Left ventricular hypertrophy (major)		
LV mass index (LVMI) in g/m^2	Male patient:	
LV relative wall thickness (RWT)	LVMI ≥149 and RWT > 0.42	2
	Female patient:	

Measurement	Criterion	Points
	LVMI ≥122 and RWT > 0.42	2
Left atrial volume index (ml/m2) as minor criterion	Patient in sinus rhythm:	
	29–34	1
	Patient in atrial fibrillation:	
	34–40	1
Left ventricular hypertrophy	Male patient:	
Minor criteria:	LVMI ≥115	
LV mass index (LVMI) in g/m2	RWT > 0.42	
LV relative wall thickness (RWT)	LV wall thickness ≥ 12	
LV wall thickness (mm)	Any 1 criterion positive	1
	2 or 3 positive	1
	Female patient:	
	LVMI ≥95	
	RWT > 0.42	
	LV wall thickness ≥ 12	
	Any 1 criterion positive	1
	2 or 3 positive	1
C. Natriuretic Peptide Domain		
Serum concentration of brain natriuretic peptide (BNP) or N-terminal proBNP (NT-proBNP) (pg/ml or ng/L)	Patient in sinus rhythm:	
	BNP > 80	2
	BNP 35–80	1
	NT-proBNP >220	2
	NT-proBNP 125–220	1
	Patient in atrial fibrillation:	
	BNP > 240	2
	BNP 105–240	1
	NT-proBNP >660	2
	NT-proBNP 375–660	1
From 0 to maximal 2 points per domain		
Select only 1 score from each domain (A, B, C) TOTAL		
Unlikely ≤1		
Intermediate 2–4		
Probable ≥5		

Table 1.
The HFA–PEFF score (step 2).

In the absence of detection of these indicators, a stress test is required. An increase in peak exercise PCWP ≥25 mmHg is interpreted as proof of the diagnosis of HF.

Step 4 involves clarifying the cause of HF. Sometimes it is necessary to perform cardiac magnetic resonance imaging, computed tomography, positron emission tomography, myocardial biopsy, genetic and special laboratory tests.

Performing the algorithm further in step 2 is not appropriate for routine practice due to the need for high-level specialists and the need for invasive diagnostics, which increases the risk for the patient. Moreover, difficulties may arise even when evaluating E/e' [25].

10. Conclusions

The diagnosis of HF in an outpatient setting is a difficult task. First, it is necessary to determine the clinical probability of HF by the features of manifestations, their dynamics, and risk factors. Then, to clarify the presence of diseases that could explain the symptoms, to assess the EF. In the absence of a reduction in EF ≤40%, it is required to calculate the HFA-PEFF Score (a more complex and expensive variant) or H2FPEF score (a cheaper and more available option, but less precise). If tissue Doppler imaging is not available, a torasemide test should be used for differential diagnosis of dyspnea.

In any case, it is impossible to allow the risk of performing a patient's examination to exceed the benefit of the information received. The examination should be sufficient to make an adequate decision about the treatment of the patient, and not be the goal itself.

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Conflict of interest

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