Sleep-disordered breathing in ischemic cardiomyopathy and hypertensive heart failure patients

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Aims The aims of this study are to (a) detect the effect of different types of heart diseases [ischemic, cardiomyopathy, hypertensive heart failure (HF)] on the association with sleep disorders, and to (b) identify the relationship between Cheyne–Stokes respiration (CSR) and left ventricular dysfunction.

Materials and methods In a cross-sectional study involving 100 HF patients, we performed echocardiography and a full-night attended polysomnography for all patients.

Results In all, 47.9% of patients with ischemic heart disease had obstructive sleep apnea (OSA), whereas 37.5% had central sleep apnea (CSA). OSA was highly prevalent in patients with hypertensive heart disease (79.2%). On the other hand, 50.0% patients with dilated cardiomyopathy (DCM) had CSA, whereas 39.3% had OSA. Patients with DCM had a significant increase in the central apnea index (11.05±9.19 events/h), as well cycle length of CSR (68.14 ±13.26 s), as compared with other groups. There was an inverse increase of cycle length with reduction in left ventricular ejection fraction (LVEF) (LVEF≥50% had a cycle length of 41.55±10.84 s, whereas those with LVEF≤30% had a longer mean cycle length of 69.23±18.09 s).

Conclusion Sleep-disordered breathing is a common disorder in different groups of HF. OSA was prevalent in ischemic and hypertensive heart disease, whereas CSA was prevalent in DCM. There was a significant increase in cycle length of CSR with a reduction in LVEF.
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Background

Sleep apnea has been recognized as an important public health problem [1]. In patients with cardiac diseases, especially chronic heart failure (HF), the prevalence of sleep-disordered breathing (SDB) is remarkably high. Central sleep apnea (CSA), in particular Cheyne–Stokes respiration (CSR), has been found in up to 40% of patients with symptomatic HF (NYHA class<II) and impaired left ventricular pump function [left ventricular ejection fraction (LVEF)>40%]. SDB also has a high prevalence in hypertensive patients and an important prognostic impact on cardiac patients [2]. The objectives of the present study are to (a) evaluate the type of sleep apneas among ischemic cardiomyopathy and hypertensive HF patients, and to (b) determine the relationship between cycle length of CSR and left ventricular dysfunction.

Materials and methods

In this cross-sectional study, 100 patients with HF were referred to Chest and Cardiology Department, Faculty of Medicine, Assiut University Hospital, between April 2015 and March 2016. Patients were categorized as follows: 48 patients with ischemic heart diseases, 28 patients with dilated cardiomyopathy (DCM), and 24 patients with hypertensive heart disease.

An informed written consent was obtained from all the patients. The study design was approved by the Scientific Ethics Committee of Faculty of Medicine of Assiut University.

Exclusion criteria

Patients with recent unstable angina or myocardial infarction within 3 months of the study, history of chronic lung disease (i.e. obstructive pulmonary disease), pregnancy, history of stroke, or clinical signs of peripheral or central nervous system disorders were excluded.

All patients were subjected to a comprehensive clinical assessment, echocardiography, and an overnight attended polysomnogram.

Clinical assessment

Clinical data of patients were recorded, including age, sex, anthropometric measures (height, weight, BMI, neck and waist circumference), history of diabetes mellitus, and history of hypertension.

The standard HF therapy was noted for each patient (angiotensin-converting enzyme inhibitors, β-blockers, diuretics, amiodarone).

Echocardiography

All participants underwent transthoracic echocardiography (Vivid S5; GE Healthcare, United States), which was performed in the left lateral decubitus position. Images were obtained in different views (parasternal long-axis, as well as apical four-chamber, two-chamber, and three-chamber, views). The quantification of cardiac chamber size and function was performed according to the American Society of Echocardiography Guidelines [3].

Left ventricular internal dimension, left ventricular end-diastolic diameter, left ventricular end-systolic diameter, interventricular septal thickness, and posterior wall thickness were determined from M-mode measurements. In addition, LVEF was measured according to the modified Simpson's method using apical four-chamber and two-chamber views: EF (%)=(EDV–ESV)/EDV×100.

where EDV is end diastolic volume and ESV is end systolic volume.

LVEF severity was determined according to the American Society of Echocardiography Guidelines [3]. Regional wall motion can be assigned to each segment to calculate the left ventricular wall motion. Right (RT) ventricle diameter and pulmonary artery systolic pressure (PASP) were also measured. PASP can be determined by using the modified Bernoulli equation (PASP)=4×(v) 2+RAP, where (v) is the peak tricuspid valve velocity, measured by continuous-wave Doppler and added to the estimated right atrial pressure (RAP) [4].

Hypertensive heart disease defined by the presence of left ventricular hypertrophy increased left ventricular posterior wall thickness and interventricular septal thickness greater than 1.1 cm [5]. DCM was defined as dilatation of all the cardiac chambers and reduced ejection fraction of less than 40% [6]. Ischemic heart disease includes abnormalities of wall motion (hypokinetic, akinetic, dyskinetic) and abnormalities of overall left ventricular function [7].

Polysomnography

All patients underwent full-night attended polysomnography (Somnstar 4100; Sensor-Medics Co., Yorba Linda, California, USA) in the sleep laboratory of the Assiut University Hospital. The polysomnogram systematically monitors the electroencephalogram (C3-A2, C4-A1), electro-oculogram, electromyogram of the chin, ECG, body positions, nasal and oral airflow, thoracic and abdominal effort, limb movements, pulse oximetry, and snoring sound level. The polysomnography (PSG) was scored manually according to the American Academy of Sleep Medicine guidelines [8].

Cheyne–Stokes respiration

Three consecutive central apneas and/or central hypopneas were separated by a crescendo and decrescendo change in breathing amplitude with a cycle length of at least 40 s, and the central apnea index was at least 5/h [8]. Cycle length is the duration of the central apnea+the duration of a respiratory phase. If central hypopneas occur, the cycle length is defined as the time from the zenith in the respiratory phase preceding the central hypopnea to the zenith of the next respiratory phase [8]. Minimum and maximum heart rate was recorded. Brady/tachy index, which is the average number of bradycardia and tachycardia events/h, was also determined.

Statistical analysis

Data were analyzed using SPSS (Statistical Package for Social Science), version 16 (IBM Inc., Armonk, NewYork, USA). Results in this study were expressed as mean±SD or number and percentage. Comparison between two groups was done using *t*-test and one-way analysis of variance test for comparison between more than two groups. The difference was considered significant when *P* value was less than 0.05.

Results

Table 1 shows patient characteristics, anthropometric measures, and comorbidities among different groups of HF. Patients with DCM had a significantly higher age group as compared with those with hypertensive and ischemic heart disease. BMI was significantly increased in hypertensive heart disease when compared with ischemic heart disease and DCM.

Hypertensive heart disease had significantly higher prevalence of hypertension, as well as systolic, diastolic, and mean blood pressure. No significant differences were demonstrated between the three groups of HF regarding sex, neck, waist circumference, prevalence of diabetes, and serum cholesterol level.

On comparing arterial blood gases and ECG changes between different groups of HF as shown in Table 2. It was observed that patients with DCM had a significant increase in the mean pH and reduction in the mean value of $PaCO_2$ associated with marked atrial

Table 1 Patient characteristics, anthropometric measures, and comorbidities among different groups of heart failure

	Ischemic heart disease (n=48)	Dilated cardiomyopathy (n=28)	Hypertensive heart disease (n=24)	P_1	P_2	P_3
Age	55.25±6.53	58.64±8.55	52.63±4.85	0.091	0.161	0.015*
Sex [n (%)]						
Male	18 (56.3)	36 (67.9)	10 (66.7)	0.279	0.498	0.927
Female	14 (43.8)	17 (32.1)	5 (33.3)			
BMI (kg/m^2)	32.71±3.40	30.36±4.51	36.51±3.78	0.722	0.016*	0.001*
Neck circumference (cm)	41.46±2.42	42.39±2.66	43.92±2.87	0.084	0.455	0.054
Waist circumference (cm)	115.04±16.28	114.07±14.24	117.08±13.53	0.957	0.350	0.418
Hypertension [n (%)]	15 (31.25)	5 (17.85)	24 (100.0)	0.994	0.000*	0.000*
SBP (90–140 mmHg)	128.75±19.75	118.93±17.07	170.67±17.86	0.009*	0.001*	0.001*
DBP (60–90 mmHg)	87.08±11.29	83.21±10.56	95.42±10.21	0.106	0.001*	0.001*
Mean BP (70–110 mmHg)	90.97±13.82	85.12±12.49	130.83±12.17	0.013*	0.000*	0.000*
Diabetes mellitus [n (%)]	12 (25)	10 (35.7)	10 (41.6)	0.234	0.321	0.412
Cholesterol (0–200 mg/dl)	178.21±32.12	176.42±24.11	182.43±36.12	0.123	0.234	0.451

Values are expressed as mean±SD. BP, blood pressure; DBP, diastolic blood pressure; DCM, dilated cardiomyopathy; IHD, ischemic heart disease; SBP, systolic blood pressure. P_1 value (IHD vs. DCM). P_2 value (IHD vs. HTN). P_3 value (DCM vs. HTN). *Significant difference.

Table 2 Gasometeric parameter and ECG changes between different groups of heart failure

	Ischemic heart disease (n=48)	Dilated cardiomyopathy (n=28)	Hypertensive heart disease (n=24)	P_1	P_2	P_3
Arterial blood gases						
pH (7.35–7.45)	7.41±0.3	7.46±0.01	7.42±0.1	0.003*	0.175	0.01*
PaCO$_2$ (35–45 mmHg)	38.07±1.2	33.7±2.11	39.08±3.7	0.002*	0.534	0.02*
PaO$_2$ (95–98 mmHg)	78.54±2.3	79.42±3.11	79.01±4.1	0.732	0.570	0.451
HCO$_3^-$ (18–22 mmol/l)	18.04±3.2	20.31±1.41	19.03±2.11	0.073	0.521	0.052
SaO$_2$ (97–98%)	95.09±0.7	96.04±2.00	95.82±0.6	0.431	0.732	0.031
ECG changes [n (%)]						
Arrhythmia: AF	21 (43.7)	14 (50)	1 (4.1)	0.723	0.001*	0.002*
Vent. arrhythmia	7 (14.5)	5 (17.8)	6 (25)	0.321	0.571	0.621

AF, atrial fibrillation; PaCO$_2$, arterial carbon dioxide tension; PaO$_2$, arterial oxygen tension. *Significant difference.

fibrillation as compared with the other two groups of HF.

Echocardiographic parameters in different groups of HF patients are shown in Table 3. LVEF was significantly lower in patients with DCM as compared with ischemic and hypertensive heart diseases. All patients with DCM had a significantly reduced ejection fraction (HFrEF>45), whereas all patients with hypertensive heart disease presented with preserved ejection fraction (HFpEF).

As regards left ventricular dimension, patients with DCM had a significant increase in left ventricular end-diastolic diameter, left ventricular end-systolic diameter, left atrial, and right ventricle diameter when compared with ischemic and hypertensive groups. On the other hand, patients with hypertensive heart disease hypertension (HTN) had a significant increase in left ventricular posterior wall diameter and interventricular septal diameter as compared with the other groups.

PASP was significantly increased in patients with DCM when compared with other groups of HF.

Baseline polysomnographic data in different groups of HF are shown in Table 4. Patients with ischemic heart disease had 47.9% obstructive sleep apnea (OSA) versus 37.5% CSA. OSA was highly prevalent in patients with hypertensive heart disease (79.2%). While patients with DCM, 50.0% had CSA and 39.3% had OSA.

Patients with DCM had a significant increase in central apnea index as compared with other groups. CSR index, as well as cycle length, was significantly increased in patients with ischemic heart disease and DCM when compared with hypertensive heart disease.

It was observed that all groups of HF had moderate to severe apnea–hypopnea index. No significant differences were demonstrated between all groups in obstructive apnea index, mixed apnea index, and hypopnea index.

As regards oxygenation during sleep, it was observed that patients with ischemic heart disease and DCM had a significant increase in desaturation index when compared with hypertensive heart disease.

Table 3 Echocardiographic parameters in different groups of heart failure

	Ischemic heart disease (n=48)	Dilated cardiomyopathy (n=28)	Hypertensive heart disease (n=24)	P_1	P_2	P_3
LVEF (%) (52–72%)	40.96±12.28	33.82±8.02	55.50±5.15	0.002*	0.000*	0.000*
LVEF [n (%)]						
Reduced (EF≤45)	38 (79.2)	28 (100.0)	0 (0)			
Preserved (EF<45)	10 (20.8)	0 (0)	24 (100.0)	0.000*	0.004*	0.000*
LVEDD (4.2–5.8 cm)	6.10±0.31	6.82±0.54	4.30±0.23	0.000*	0.000*	0.000*
LVESD (2.5–3.9 cm)	4.46±0.42	5.25±0.63	2.85±0.20	0.000*	0.000*	0.000*
PWd (0.6–1.0 cm)	0.93±0.10	0.89±0.11	1.37±0.30	0.137	0.000*	0.000*
IVSD (0.6–1.0 cm)	1.09±0.09	1.13±0.06	1.30±0.08	0.121	0.000*	0.000*
LVFS (≥30%)	23.35±3.50	21.43±2.78	40.88±0.90	0.104	0.000*	0.000*
LT atrial diameter (3–4 cm)	3.71±0.13	4.81±0.56	3.78±0.39	0.000*	0.162	0.000*
RT vent. diameter (2–2.8 cm)	1.94±0.25	2.07±0.27	1.55±0.29	0.060	0.000*	0.000*
PASP (15–30 mmHg)	45.23±5.87	49.64±11.74	41.83±7.70	0.221	0.026*	0.010*

Values are expressed as mean±SD. DCM, dilated cardiomyopathy; EF, ejection fraction; IHD, ischemic heart disease; IVSD, interventricular septal diameter; LVFS, fraction of shortening; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; PASP, pulmonary artery systolic pressure; PWd, left ventricular posterior wall diameter. P_1 value (IHD vs. DCM). P_2 value (IHD vs. HTN). P_3 value (DCM vs. HTN). *Significant difference.

Table 4 Baseline polysomnographic data in different groups of heart failure

	Ischemic heart disease (n=48)	Dilated cardiomyopathy (n=28)	Hypertensive heart disease (n=24)	P_1	P_2	P_3
Type of apnea [n (%)]						
CSA	18 (37.5)	14 (50.0)	0 (0.0)	0.562	0.002*	0.000*
OSA	23 (47.9)	11 (39.3)	19 (79.2)			
No SDB	7 (14.6)	3 (10.7)	5 (20.8)			
Respiratory events						
Obstructive AI (n/h)	12.12±10.71	10.24±10.88	14.90±9.13	0.940	0.254	0.463
Mixed apnea index (n/h)	1.05±1.38	0.71±0.94	0.78±1.13	0.778	0.228	0.305
Central AI (n/h)	8.11±9.94	11.05±9.19	2.76±3.53	0.109	0.072	0.000*
Hypopnea index (n/h)	11.18±12.99	10.18±8.03	13.70±8.50	0.948	0.891	0.614
AHI (n/h)	32.07±20.81	33.01±14.28	30.76±17.39	0.401	0.280	0.114
CSR (index/h)	44.14±9.29	42.97±11.20	5.77±2.65	0.001*	0.039*	0.000*
Cycle length (s)	62.12±14.11	68.14±13.26	45.03±12.33	0.003*	0.031*	0.000*
Oxygenation						
Desaturation index	57.15±23.99	61.21±20.43	44.63±16.94	0.365	0.041*	0.004*
Basal SaO₂ asleep (%)	96.04±1.24	95.39±3.74	97.04±1.27	0.843	0.001*	0.001*
Minimum SaO₂ asleep (%)	64.23±16.09	58.36±11.93	71.38±17.63	0.213	0.124	0.009*
Average SaO₂ (%)	81.13±8.38	75.75±6.88	85.29±8.49	0.005*	0.040*	0.000*
SaO₂>90% (min)	59.06±46.56	72.71±38.00	49.63±39.02	0.002*	0.002*	0.005*
Heart rate						
Basal HR (60–90 beats/min)	69.02±3.21	65.18±2.33	75.17±3.31	0.000*	0.000*	0.000*
Minimum HR (beats/min)	49.17±6.04	46.39±8.51	48.92±6.01	0.061	0.614	0.217
Maximum HR (beats/min)	118.94±11.15	114.50±12.17	130.21±12.29	0.377	0.003*	0.002*
Brady/tachy index	32.56±13.67	29.27±15.05	40.35±12.90	0.542	0.001*	0.005*
Arousal index						
Arousal index (n/h)	22.99±10.23	24.30±8.67	19.41±10.83	0.686	0.195	0.110

Values are expressed as mean±SD. AHI, apnea–hypopnea index (n/h sleep); Average SaO₂, average arterial oxygen saturation; Brady/tachy index, average number of bradycardia and tachycardia events/h; CSA, central sleep apnea; CSR index, number of CSR events/total sleep time; CSR, Cheyne–Stokes respiration; Cycle length, the length of one CSR cycle spent in seconds; DCM, dilated cardiomyopathy; HR, heart rate; IHD, ischemic heart disease; Oxygen desaturation index (events/h), min; Max HR, maximum heart rate; Min HR, minimum heart rate; No SDB, no sleep-disordered breathing; OSA, obstructive sleep apnea; SaO₂>90% min, time in bed with oxygen saturation<90% in minutes; SaO₂%, minimum arterial oxygen saturation. P_1 value (IHD vs. DCM). P_2 value (IHD vs. HTN). P_3 value (DCM vs. HTN). *Significant difference.

A significant reduction in minimum and average oxygen saturation during sleep was demonstrated in those patients with ischemic heart disease and DCM as compared with hypertensive heart disease. In addition, time spent with oxygen saturation below 90% was increased in ischemic heart disease and DCM.

There was a significant decrease in basal heart rate in those patients with DCM as compared with the

groups of HF. Maximum heart rate and brady/tachy index were significantly increased in hypertensive heart disease when compared with ischemic heart disease and DCM. No significant difference was observed in arousal index between all groups of HF.

As shown in Table 5, The variation in cycle length in Cheyne–Stokes breathing with different severities of HF. It was found that there was a considerable variation in the cycle length with different degrees in left ventricular dysfunction. It was observed that patients with LVEF at least 50% exhibited a mean cycle length of 41.55±10.84 s, whereas those with LVEF of 30% or less had a longer mean cycle length of 69.23±18.09 s.

Discussion

In this study, we have demonstrated that SDB was a common disorder among HF patients. On the basis of polysomnography, we found that 47.9% of patients with ischemic heart disease had OSA, whereas 37.5% had CSA. Similarly, Wali et al. [9] investigated the prevalence of OSA among 156 patients with ischemic HF and demonstrated that 56.4% were found to have OSA. In addition, Prinz et al. [10] studied 275 patients with ischemic heart disease who underwent polysomnography to demonstrate the prevalence of SDB and found that 51% had OSA and 23% had CSA. Another study by Yumino et al. [11] studied 193 patients with ischemic and nonischemic HF to evaluate the prevalence of sleep apnea and possible risk factors, and found that the prevalence of sleep apnea (apnea–hypopnea index≥15) was found in 73% (OSA in 50% and CSA in 25%) of those with ischemic HF.

Our study revealed that CSA was a prevalent disorder among DCM and estimated for 50% as compared with 39.3% who had OSA. Similarly, Banno et al. [12] investigated the prevalence of SDB among 35 patients with DCM and found that 50% of patients had CSA and 30% had OSA. In another study by Javaheri et al. [13], 81 patients with HF were studied, and it was found that CSA was common in patients with DCM (45%). In addition, Wilcox et al. [14] showed that 56% of patients with DCM with severe left ventricular dysfunction had CSA. In another study by Tkacova and colleagues, 316 patients with HF secondary to DCM were studied, and it was found that 42% of them had CSR-CSA. Thus, elevated left ventricular volume might lead to pulmonary congestion and hypocapnia, which would create the predisposition to CSR-CSA [15].

Our results showed that patients with DCM had a significant increase in the mean pH and reduction in the mean value of $PaCO_2$ (34.7±2.11 mmHg) as compared with the other groups. In addition, Wilcox et al. [14] found that the mean value of $PaCO_2$ was significantly reduced among patients with DCM (34.3± 3.14 mmHg); the authors postulated that a lower $PaCO_2$ was a key predisposing factor for CSA in DCM. Elevated left ventricular filling pressures and pulmonary congestion causes hyperventilation through stimulation of pulmonary vagal irritant receptors causing hypocapnia with $PaCO_2$ closer to the apnea threshold than normal [16].

In a study by Oldenburg et al. [17], 647 stable HF patients with NYHA class at least II and with impaired systolic left ventricular function (ejection fraction≤40%) were studied, and 342 patients with ischemic heart disease and 305 patients with DCM were all screened by cardiorespiratory polygraphy for the presence and type of SDB, and it was found that 46% of patients with ischemic heart disease had OSA and 36% had CSA; on the other hand, 38% of patients with DCM had CSA and 32% had OSA.

Similarly, Paulino et al. [18] investigated 316 French patients with different etiologies of HF (ischemic heart disease, cardiomyopathy) and found that the prevalence of SDB was high (52%) in ischemic heart disease; of them, 38% had OSA and 30% had CSA, whereas in patients with DCM 40% had CSA and 36% had OSA.

We observed that OSA was highly prevalent in hypertensive heart disease (79.2%). Our findings are in agreement with those of Logan et al. [19], who observed that the prevalence of OSA was 83% in patients with hypertensive heart disease hypertension (HTN), with a significant increase in daytime systolic

Table 5 Variation in cycle length in Cheyne–Stokes breathing with different severities of heart failure

LVEF (%)	Normal range (≥50%)	Mildly abnormal (40–50%)	Moderately abnormal (30–40%)	Severely abnormal (≤30%)	P value
Number of patients [n (%)]	20 (20)	24 (24)	40 (40)	16 (16)	0.0001[*]
Cycle length(s) (mean±SD)	41.55±10.84	51.99±12.44	56.32±13.24	69.23±18.09	

CSR, Cheyne–Stokes respiration; LVEF, left ventricular ejection fraction; Cycle length, length of one CSR cycle spent in seconds.
*Significant difference.

blood pressure (190.43±43.12 vs. 140.54±23.11 mmHg in no SDB).

In addition, Labib *et al.* [20] investigated 40 patients suffering from uncontrolled HTN to study the prevalence of SDB and found that 70% of HTN patients suffered from OSA.

OSA plays a linking role between hypertension and HF. This could contribute to an increase in sympathetic nerve activity. Intermittent apnea-related hypoxia, as well as the intrathoracic pressure swings and arousals associated with respiratory events, augment sympathetic nervous system activity. Thus, increased sympathetic activity results in a concomitant increase in heart rate, cardiac output, peripheral vascular resistance, and enhanced fluid retention – mechanisms that may contribute to elevated blood pressure [21].

Another striking finding of our study was that there was a considerable variation in the cycle length with different degrees of left ventricular dysfunction among HF patients; patients with LVEF at least 50% had a mean cycle length of 41.55±10.84 s, whereas those with lower LVEF of 30% or less had a longer mean cycle length of 69.23±18.09 s. Similarly, Wedewardt and colleagues studied 104 HF patients who underwent polysomnography and echocardiographic evaluation; patients were stratified into five groups according to their LVEF (<20% up to ≥50%). Comparing the groups of the best LVEF (>50%) with the worst LVEF (<20%), the authors demonstrated that there was a significant increase in cycle length of CSR between the five groups of HF (cycle length 49±17 s in LVEF≥50% and 86±23 s in LVEF>20%) [22]. In addition, Nopmaneejumruslers and colleagues compared cycle length of CSR in HF patients with LVEF greater than 40% and those with impaired left ventricular systolic function (LVEF 40%). The authors found that those patients with LVEF of 40% or less had a longer mean cycle duration than those with an LVEF of greater than 40% (66.6±5.6 vs. 46.6±2.9 s). Moreover, they postulated that prolonged cycle length of CSR and hyperpnea durations typical of CSR-CSA are a consequence of left ventricular systolic dysfunction, low cardiac output, and prolonged circulation time. Thus, it is likely that impaired cardiac function was a major contributor to the development of CSR [23].

Conclusion
SDB is a common disorder in different groups of HF. OSA was prevalent in ischemic and hypertensive heart disease, whereas CSA was prevalent in DCM. There was a significant increase in cycle length of CSR with reduction in LVEF.

Conflicts of interest
There are no conflicts of interest.

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