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A study on related factors of hemodynamic depression in carotid artery stenting

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Abstract. – OBJECTIVE: To investigate the pathogenesis and related factors of carotid artery stenting (CAS) related to hemodynamic depression (HD).

PATIENTS AND METHODS: 433 CAS patients were admitted to our hospital and were selected from Jan 2013 to Jun 2016. We set up the HD diagnostic criteria for CAS and observed the whole morphology of vessels to classify carotid atherosclerotic plaques. We analyzed cerebral angiography and placed temporary cardiac pacemaker via femoral vein in order to prepare carotid artery stenting. We determined the general situation of HD after CAS according to the results.

RESULTS: 38 patients received temporary placement of cardiac temporary pacemakers, and 241 patients (59.80%) developed HD. 403 patients were divided into HD group and non HD group and results suggested there was no significant difference between the two groups except for diabetes (*p*>0.05). Also, bilateral carotid stenting was performed in 26 patients (6.45%), and the HD group was significantly higher than that in non HD group (*p*<0.05). HD group had 31 MACCE cases, and non HD group had 2 MACCE cases. There was no occurrence of adverse cardiovascular events in this study.

CONCLUSIONS: Cardiac temporary pacemaker may be one of the effective approaches to improve HD after CAS and reduce perioperative MACCE.

Key Words

Carotid artery stenting, Hemodynamic depression, Carotid extracranial atherosclerotic, Plague.

Introduction

Extracranial carotid atherosclerotic stenosis is one of the important causes of ischemic stroke. After 40 years of clinical practice, and through the verification of NASCET (North American Symptomatic Carotid Endarterectomy Trial), ECST (European Carotid Surgery Trial) and other tests, carotid endarterectomy (CEA) started the "golden standard" for the treatment of carotid stenosis. With the rapid development of imaging technology and neural interventional technology, the carotid artery stenting (CAS) has attracted much attention in the treatment of extracranial carotid artery stenosis for its minimally invasive, safe and effective advantages. In recent years, many clinical randomized controlled trials have confirmed that there is no significant difference between CAS and CEA in the major endpoint events. These suggest that both have the same efficacy in long-term carotid stroke prevention[1-4].

The most common complication of CAS is hemodynamic depression (HD) which changes by CAS induced hemodynamic. Compared with CEA, the occurrence of surgery is higher[5]. Studies have shown that[6] HD significantly increases the risk of major adverse events during the perioperative period of CAS. HD is an independent risk factor for major adverse cerebral and cardiovascular events (MACCE) during the perioperative CAS[7,8]. Moreover, angiographic evidence of reperfusion injury is often discovered after carotid artery stenting. In the situation of reperfusion after CEA, there are many risk factors for reperfusion syndrome such as stenosis, hypertension, and poor collaterals, which can be identified and after that prevented[9]. The purpose of this study was to investigate the pathogenesis and related factors of CAS related HD.

Patients and Methods

Patients

From January 2013 to June 2016, 433 CAS patients were admitted to the Department of Neurology of Daping Hospital of Third Military Medical University (Chongqing, China) and were selected in this study. Vascular stenosis was measured by NASCET[10]. CT angiography (CTA) was performed through the head and neck and confirmed by digital subtraction angiography (DSA). Patients and their families were informed about the purpose and process of this study, and they agreed and signed informed consent. This study was approved by the Ethical Committee of our hospital.

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Inclusion criteria were: 1- symptomatic carotid artery stetablenosis, stenosis rate ≥ 50%; 2- symptomatic carotid atherosclerotic stenosis, stenosis rate ≥ 60%.

Exclusion criteria: 1- severe neurological conditions, such as lesions, loss of function in the ipsilateral brain, and paralysis, with no benefit from CAS treatment; 2- the total occlusion of carotid artery is longer than 10 mm, DSA showed no blood flow at the distal end; 3- ipsilateral intracranial arteriovenous malformations or aneurysms with bleeding tendency; 4- intracranial hemorrhage occurred within 3 months or extensive cerebral infarction in 4 weeks or a history of acute myocardial infarction within 2 weeks; 5- severe heart, liver, and kidney dysfunction; 6- contrast agents, allergies, and other angiographic contraindications; 7- allergic to aspirin enteric-coated tablets, clopidogrel and other anti platelet drugs; 8- serial lesion combined with anterior circulation and posterior circulation combined with multi site cerebral vascular stent implantation; 9- severe sick sinus syndrome or atrioventricular block.

Methods

HD Diagnostic Criteria After CAS

HD after CAS included hypotension and bradycardia, or symptomatic or asymptomatic hypotension during or after CAS (systolic pressure < 90 mmHg), bradycardia (< 60 times/min)[6]. The pacing frequency was set as 60 times/min, if the intraoperative and postoperative ECG showed clear pacing signal, then HD was existed in patients.

Classification of Carotid Atherosclerotic Plaques

We used LightSpeed 64 CT scanner (GE, Boston, MA, USA). Moreover, to observe the whole morphology of vessels, we performed multiplanar reformation (MPR), maximum intensity projection (MIP), volume rendering (VR), vessel analysis (VA) etc. Also, the CT value of carotid atherosclerotic plaque was measured. CT value ≥ 130 Hu was defined as calcified plaque, the rest were non calcified plaques. The arbitrary level images of coronal, axial and sagittal showed the contrast medium diffused to the outside of the arterial lumen. The depth of the contrast agent entering the plaque along the plaque surface was >1 mm. The eccentricity of the plaque is determined by calculating the eccentricity index (EI). Maximum plaque thickness (A) and minimum thickness (B) were measured, EI formula =(A-B)/A. If EI > 0.7, it was eccentric plaque; if EI<0.7, it was concentric plaque[11].

Analysis of Cerebral Angiography

The instrument was Innova 3100 digital subtraction machine for American GE company (Boston, MA, USA). The 5FPIG and 5FVER contrast tubes were performed with an aortic arch (projection angle of 30-45° left forward) and an angiogram on the arch.

Aortic arch type: the measuring distance was within the diameter of the common carotid artery, which counted as Type I aortic arch. The distance was 1-2 times of the diameter of the common carotid artery, counted as Type II aortic arch. When 2 times of the diameter, it was Type III aortic arch[12].

Determination of the rate of carotid stenosis and the length of carotid stenosis: bilateral carotid stenting was recorded on the side with the greatest stenosis rate. The rate of carotid stenosis was calculated according to the NASCET standard. The degree of stenosis is divided into Mild stenosis (0-29%); moderate stenosis (30-69%); severe stenosis (70%); Occlusion (100%).

Extracranial carotid artery tortuosity: extracranial carotid artery tortuosity including "C" type (tortuosity), "S" type (kinking), "O" type (coiling). "C" type was characterized by vascular wavy lines. "S" type was characterized by vascular elongation, angle changed. "O" type showed excessive elongation of vascular tortuosity, as a "O" configuration[13,14].

Placement of Temporary Cardiac Pacemaker via Femoral Vein

Placement method: pacemaker implantation was performed by transvenous endocardial pacing. The pacing frequency was set as 60 times/min; CAS was then performed after pacemaker implantation success surgery.

Carotid Artery Stenting

Preoperative preparation: bayaspirin enteric-coated tablets (100 mg, 1 time/d) (Bayer Corporation, Leverkusen, Germany), clopidogrel (75 mg, 1 time/d) (Sanofi, Paris, France) were taken at least 3 days before the surgery. Blood routine, liver and kidney function, blood coagulation image, electrocardiogram, and chest X-ray, were operated. Preoperative intramuscular injection of phenobarbital (0.1 g) (Shanghai Haling, Shanghai, China) was operated 0.5 h before the surgery. 30 mg fasudil hydrochloride (Standard Med, Shenzhen, Guangdong, China) were added into 250 ml 0.9% saline intravenously during the operation, and ready for use at 3 d postoperatively.

Operation process: Seldinger technique was used to puncture the femoral artery, and the 8 F vascular sheath was successfully inserted into the femoral sheath. The 8FMPD (Cordis, Milpitas, CA, USA) leads were placed at the affected side of the common carotid artery and the distal protective device (DPD) was placed directly at the distal end of the carotid segment of the internal carotid artery. DPD included Angioguard (Cordis, Milpitas, CA, USA) and Spider RX (EV3, Minneapolis, MN, USA). Balloon dilatation or stenting was performed prior to implantation of DPD according to the stenosis of the lesion. After stent implantation, the stent location, vasospasm, vascular dissection and distal vascular embolization were confirmed by re-examination. Next, the DPD was recovered, the guide-wire, and catheter were withdrawn. ECG, blood pressure and pulse oxygen saturation were monitored during the operation. Blood pressure was monitored at 2 min before balloon and stent placement. The preoperative and postoperative rates and blood pressure changes were recorded. NIHSS (NIH Stroke Scale) score was assessed.

Postoperative monitoring and treatment: the ECG, blood pressure, and pulse oxygen saturation were monitored for at least 24 h. The blood pressure was monitored every 5-30 min. All the patients with HD had to stop antihypertensive drugs and were definitely confined to bed for 24 h. If the heart rate was less than 60, 500 ml 0.9% hydrochloride were injected to elevate the heart rate. If the systolic pressure was lower than 90 mmHg, dopamine hydrochloride or dopamine hydrochloride + hydroxylamine micro pump were applied to regulate the blood pressure to 100-120/60-80 mmHg. If the heart rates of the patients stayed >60, the systolic pressure > 90 mmHg without other aids, and without symptoms of orthostatic hypotension persist for more than 3 h. After the operation, the patients were treated with clopidogrel (75 mg, 1/d, and aspirin enteric-coated tablets (100 mg, 1/d) for at least 3 months.

Perioperative observation of cardiac temporary pacemaker: patients with temporary cardiac pacemaker were followed by continuous ECG monitoring, and the pacing rhythm, autonomic rhythm and arrhythmia were observed carefully. The removal time of pacemaker catheter was recorded.

Follow-up: the occurrence of MACCE in 30 days after CAS was monitored, including TIA, ischemic and hemorrhagic strokes, acute myocardial infarction, and any cause of death. The severity of a stroke is divided into major stroke (mRS 4-5) or mini-stroke (mRs 2-3).

Statistical Analysis

SPSS 18.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. The difference of risk factors between HD group and non HD group was compared by x^2-test and independent sample *t*-test. The continuous variables were tested by independent *t*-test, and the categorical variables were checked by Pearson x^2-test or continuous correction x^2-test. Significant differences were included in the binary unconditional multivariate Logistic regression. When $p<0.05$, there was a statistically significant difference.

Results

General Situation of HD After CAS

In all patients, 16 cases of combined carotid and vertebral artery stenting (3.6%), 2 cases with operation failed (0.5%), 12 lost in follow-up (2.8%) were excluded. There were 403 patients (93.1%) with 30 days followed up were included in the study. In 403 CAS patients, 38 patients received temporary placement of cardiac temporary pacemakers, and 241 patients (59.80%) developed HD. 212 patients (87.97%) had bradycardia (36 only had bradycardia), 176 patients (73.03%) had bradycardia combined with decreased blood pressure, 191 (79.25%) had balloon dilatation during the operation, 21 (8.71%) had delayed ventricular tachycardia 1-5 d after the operation. 205 patients (85.06%) had blood pressure drops (29 only had blood drops), 127 (52.70%) had balloon dilatation during the operation. 78 (32.37%) had blood pressure drops after the operation. Within 241 patients, 184 patients (76.35%) needed vasopressor medication or heart rate enhancing medications. The longest duration of HD was 11 d (Table I).

Analysis of Related Factors of HD

403 patients were divided into HD group and non HD group. The baseline characteristics of the two groups were compared. The results showed that there was no significant difference between the two groups except for diabetes mellitus ($p>0.05$) (Table II). The imaging features of the two groups were compared. The results showed that the calcified plaques and eccentric plaques in the HD group were significantly higher than those in non HD in the patch type ($p>0.05$) (Table III).

Table I. General situation of HD occurrence after CAS.

	N=241
Bradycardia, n (%)	212 (87.97)
Simple bradycardia, n (%)	36 (14.94)
Blood pressure drop, n (%)	176 (73.03)
Intraoperative bradycardia, n (%)	191 (79.25)
Postoperative bradycardia, n (%)	21 (8.71)
Blood pressure drop, n (%)	205 (85.06)
Simple blood pressure drop, n (%)	29 (12.03)
Bradycardia, n (%)	176 (73.03)
Intraoperative blood pressure drop, n (%)	127 (52.70)
Postoperative blood pressure drop, n (%)	78 (32.37)

The Comparison of CAS Operation Process Between HD Group with non HD Group

In 403 patients, bilateral carotid stenting was performed in 26 patients (6.45%), and the HD group was significantly higher than that in non HD group ($p<0.05$). Stents were placed in 342 patients (84.86%). In 61 patients (15.14%), the conical stent was placed due to the difference between the diameter of the common carotid artery and the internal carotid artery. The proportion of tapered stents in group HD was significantly lower than that in non HD group ($p<0.05$). Balloon dilatation was performed in 55 patients (13.65%) prior to stenting. The balloon dilation rate in the HD group was significantly higher than that in the non HD group ($p<0.001$) (Table IV).

Table II. Comparison of general baseline characteristics between HD group and non HD group after CAS.

	HD (n=241)	Non HD (n=162)	*p*-value
Age ≥70, n (%)	101 (41.91)	53 (32.72)	0.063
Gender (m), n (%)	186 (77.18)	112 (69.14)	0.071
HR (time/min, ±s)	66.62±4.85	67.16±4.71	0.267
Hypertension, n (%)	168 (69.71)	98 (60.49)	0.056
Diabetes, n (%)	77 (31.95)	31 (19.14)	0.004*
Smoking, n (%)	87 (36.10)	72 (44.44)	0.093
Hypercholesterolemia, n (%)	46 (19.09)	42 (25.93)	0.103
Coronary heart disease, n (%)	69 (28.63)	33 (20.37)	0.061
Symptomatic carotid stenosis, n (%)	159 (65.98)	106 (65.43)	0.910

*$p<0.05$.

Table III. Comparison of imaging features between HD group and non HD group after CAS.

	HD (n=241)	Non HD (n=162)	*p*-value
Calcification of aortic arch, n (%)	54 (22.41)	32 (19.75)	0.524
Vulnerable plaque			
Calcified plaque, n (%)	87 (36.10)	7 (4.32)	<0.001*
Ulcerative plaques, n (%)	56 (23.24)	46 (28.40)	0.243
Eccentric plaque, n (%)	141 (58.51)	57 (35.19)	<0.001*
Classification of aortic arch			0.674
I arch, n (%)	72 (29.88)	42 (25.93)	
II arch, n (%)	141 (58.51)	99 (61.11)	
III arch, n (%)	28 (11.62)	21 (12.96)	
Carotid artery Tortuosity, n (%)	135 (56.02)	80 (49.38)	0.191
Carotid artery stenosis			0.003*
≥ 70%, n (%)	178 (73.86)	97 (59.88)	
< 70%, n (%)	63 (26.14)	65 (40.12)	
Contralateral carotid Occlusion, n (%)	13 (5.39)	3 (1.85)	0.074
Length			
≥15 mm, n (%)	108 (44.81)	13 (8.02)	<0.001*
<15 mm, n (%)	133 (55.19)	149 (91.98)	

*$p<0.05$.

Table IV. The operation process of HD group and non HD group were compared.

	HD (n=241)	Non HD (n=162)	Statistics	*p*-value
Bilateral carotid stent, n (%)	24 (9.96)	2 (1.23)	χ^2=12.217	<0.001*
Shapes				
Tapered, n (%)	26 (10.79)	35 (21.60)	χ^2=8.824	0.003*
Vertical, n (%)	215 (89.21)	127 (78.40)		
Balloon dilation				
Pre, n (%)	52 (21.58)	3 (1.85)	χ^2=31.983	<0.001*
Post, n (%)	5 (2.07)	6 (3.70)	χ^2=0.968	0.325

*$p<0.05$.

Multivariate Analysis of CAS Correlated With HD

Baseline characteristics, radiographic features, and intraoperative procedures were compared between the HD group and non HD group. All the significant differences were analyzed by the binary unconditional Logistic regression analysis. The results showed the following 7 factors: calcified plaque ($p<0.001$), eccentric plaque ($p<0.001$), severe stenosis ($p=0.048$), long lesion ($p<0.001$), bilateral carotid artery stenting ($p=0.021$), tapered support (p=0.007), balloon pre dilation ($p=0.015$) (Table V).

Comparison of the Occurrence of MACCE in Group HD and Non HD Group

CAS total MACCE occurred in 33 patients during the perioperative period. There were 31 MACCE cases, mainly in TIA (Transient Ischemic Attack) and minor stroke. There were 2 cases of death due to cerebral hemorrhage and acute myocardial infarction. There were also 2 MACCE cases, 1 TIA and 1 cerebral hemorrhage (death) during the perioperative period. The occurrence of MACCE in group HD and non HD group was compared. The results showed that the total occurrence of MACCE in group HD was significantly higher than that in non HD group. ($p<0.001$) (Table VI).

Discussion

Carotid atherosclerosis stenosis is one of the main causes of ischemic stroke; 20-25% ischemic stroke is caused by extracranial carotid atherosclerotic stenosis[15,16]. Optimizing antithrombotic therapy is still insufficient to control the occurrence of stroke due to carotid stenosis. CEA has long been regarded as the gold standard for the treatment of carotid stenosis. A number of studies have also shown that optimized drug therapy with CEA can reduce stroke occurrence and mortality[17-19]. With the continuous improvement of neuroimaging and the rapid development of brain protection devices, CAS becomes popular in the treatment of extracranial carotid artery stenosis. In recent years, a number of studies have compared two surgical procedures[20-22]. Since 2010, a series of studies on Carotid Revascularization Endarterectomy *vs*. Stenting Trial (CREST) have been published[2,22], which were considered as a milestone in CAS's progress. The trial showed no significant difference in stroke occurrence between the symptomatic and asymptomatic patients during the primary endpoint of the perioperative period (stroke, myocardial infarction, death) and during the 10-year follow-up. Another asymptomatic carotid artery disease trial (ACT-1) by published

Table V. Multivariate logistic regression analysis of HD after CAS.

Risk factor	B	S.E.	Wals	OR (95%CI)	*p*-value
Calcified plaque	2.062	0.455	20.503	7.863 (3.221-19.199)	<0.001*
Eccentric plaque	1.009	0.257	15.460	2.744 (1.659-4.538)	<0.001*
Severe stenosis	0.531	0.268	3.924	1.701 (1.006-2.878)	0.048*
Length of lesion (15 mm)	1.699	0.350	23.534	5.469 (2.753-10.866)	<0.001*
Bilateral carotid stent	1.935	0.838	5.324	6.921 (1.338-35.795)	0.021*
Tapered support	-0.945	0.351	7.240	0.389 (0.195-0.774)	0.007*
Balloon dilation	1.606	0.659	5.948	4.985 (1.371-18.126)	0.015*

*$p<0.05$. B: ratio; S.E.: Standard error; Wals: Wald statistic.

Table VI. MACCE occurrence at CAS of HD group compared with the non HD group.

	HD (n=241)	Non HD (n=162)
MACCE, n (%)	31 (12.86)*	2 (1.24)
TIA, n (%)	16 (6.64)	1 (0.62)
Stroke, n (%)	14 (5.81)	1 (0.62)
Major stroke, n (%)	4 (1.66)	1 (0.62)
Minor stroke, n (%)	10 (4.15)	0 (0)
Myocardial infarction, n (%)	1 (0.41)	0 (0)
Death, n (%)	2 (0.83)	1 (0.62)

*compared with non HD group, $p<0.001$.

results over the same period confirmed that the effect of carotid stenting was not inferior to endarterectomy[4]. Moreover, the occurrence of severe stroke and death during the perioperative period and the occurrence of stroke during the 5-year follow-up were not significantly different. We further analyzed the results of two clinical randomized controlled trials, which showed that perioperative adverse events (mostly mild strokes) were significantly higher in CAS than in CEA. Therefore, the reduction of perioperative cerebrovascular events in CAS is still the focus of current research.

Hemodynamic changes (HD) caused by CAS are the most common perioperative complications, and have a higher occurrence compared to that caused by CEA[5]. The occurrence rate of HD caused by CAS is 72-80%[6]. HD is an important risk factor for perioperative ischemic stroke in CAS[23]. Balloon dilation, stent expansion and a mechanical stretch of carotid sinus caused by surface tearing intima and atherosclerotic plaque in CAS operation, adjust the carotid arterial wall compliance, change the sensitivity of carotid artery carotid sinus receptors and resulting in receptor dysfunction. Previous researches explored the factors related to the occurrence of HD. They found that old age[23], history of cardiovascular disease[24], eccentric calcified plaque near the bifurcation of carotid artery[25], balloon dilation[26], may be the risk factors of HD. However, smoking and diabetes may be the protective factors for HD.

In this study, the prevalence of HD after CAS was 59.8%. The analysis of related factors of HD showed that the occurrence of HD was mainly related to the characteristics of the lesion and the mode of operation, which suggested that HD was mainly caused by the carotid sinus baroreceptor during mechanical stimulation after CAS. A long lesion, calcified plaque, eccentric plaque, bilateral carotid stent implantation and balloon dilatation were positively correlated with HD after CAS. The reason of why long plaque is more susceptible to HD may be related to balloon length and expansion pressure, and the operation time and postoperative expansion of stents slowly increase the contact area of a baroreceptor. The reason that HD is more likely caused by calcified plaque, on one hand is due to calcification caused local vascular wall expansion ability decreased baroreceptor sensitivity decreased, and decreasing the short-term blood pressure regulation. On the other hand, it may due to increased plaque hardness, the need for greater expansion pressure balloon dilatation and the enhancement on carotid sinus baroreceptor stimulation. Eccentric plaques may exacerbate HD by balloon dilatation, unstable mechanical conditions and elastic retraction during stent release. Bilateral carotid artery stenting due to stimulation of balloon and stent on carotid sinus baroreceptor increase the occurrence of HD. Another reason may be the improvement in blood supply to the brain as the bilateral carotid stent is released. The blood pressure regulating central insular blood supply has also improved, to enhance bilateral insula on carotid sinus baroreceptor sensitivity adjustment function. The regulation of blood pressure and heart rate are all increased by mechanical stimulation of the baroreceptor[27]. This study also confirmed that the cone stent is a negative factor associated with HD. HD is associated with adverse cardiovascular events after CAS. 500 CAS cases study by Gupta et al[6] showed that the major adverse cardiovascular events (OR 3.05, 95% CI, 1.35-5.23, $p<0.02$), or stroke (OR 3.34, 95% CI, 1.13-9.90, $p<0.03$) increased significantly during the perioperative period in patients with persistent HD. Ito et al[28] studied the comparison of 128 cases of CAS patients before and after MRI DWI (diffusion weighted imaging); the results showed that in HD group at 1 d after surgery, the occurrence of new acute ischemic lesions on MRI DWI were significantly higher than non HD group (32.2% *vs.* 15.5%, $p=0.04$). Studies have shown that new ischemic lesions of DWI after CAS are related to long-term postoperative cognitive impairment[29]. We investigated the mechanism of stroke led by HD. During CAS, due to endovascular procedures such as balloon dilation and stent release, it causes stent thrombosis. The lipid fragmentation and vulnerable plaque spontaneous reaches the downstream vascular bed. The low flow mechanics slows the removal of emboli and plaque debris by cerebral blood flow, resulting in vascular occlusion, plaques, the release of inflammatory mediators, vasospasm, etc.. When the intracranial arterial stenosis occurs, the mechanical changes in the low blood flow weaken the collateral circulation of the narrow blood supply area, leading to the occurrence of stroke[30]. Therefore, we hypothesized that the intervention therapy for HD might be one of the active ways to reduce the occurrence of CAS during the perioperative stroke. During AS surgery, due to mechanical stimulation of carotid sinus baroreceptor, depressor reflex is evoked. The heart rate is slowing down, and cardiac output decreased, vasodilation, the peripheral resistance reduced, followed by lower blood pressure. Due to the decline of the sensitivity of carotid atherosclerosis in patients with carotid sinus baroreceptor, it is difficult to receive a negative feedback mechanism under a physiological condition of depressor reflex. Therefore, interventional therapy is required. Atropine is the most common drug used to correct bradycardia during CAS. Atropine can also promote blood pressure to a certain extent[31,32]. Isoproterenol is often used to correct bradycardia after surgery. However, there are a few limits of drug therapy for bradycardia: 1- there are inevitable delays; 2- the effect of atropine is not efficient; 3- in elderly patients with coronary artery disease, the use of atropine may induce disturbance of consciousness, urinary retention, and arrhythmias[33]; 4- Long-term use of isoproterenol may increase myocardial contractility and myocardial oxygen consumption, resulting in angina pectoris, myocardial infarction, and rapid arrhythmia. These factors may be the reasons for the perioperative stroke rate in CAS, especially in patients with higher HD high-risk than those in the CEA group. The application of temporary cardiac pacing in the surgical treatment of patients with slow arrhythmia is relatively mature. However, there are no consistent criteria for the indications for CAS. Most of the observations were still from clinical experiences. Because of the particularity of the CAS technology, the application of temporary cardiac pacing in CAS is not only suitable for slow arrhythmia patients, but also for those patients with a high-risk occurrence of HD. We applied protective cardiac temporary pacemaker to 38 patients with bradycardia or high-risk HD. Results came back that no occurrence of adverse cardiovascular events. A temporary cardiac pacemaker may be one of the practical approaches to improve HD after CAS and reduce perioperative MACCE.

Conclusions

We showed that cardiac temporary pacemaker may be one of the effective approaches to improve HD after CAS and reduce perioperative MACCE.

Conflict of Interest:

The Authors declare that they have no conflict of interests.

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