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CARDIAC ELECTRICAL DYSFUNCTION IN ACUTE BRAIN LESIONS

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ABSTRACT

This study briefly reviews the heart abnormalities in diverse intracranial pathologies, including strokes, spontaneous and traumatic subarachnoid hemorrhage and intracranial hemorrhage, and presents the results of a one-year prospective study of heart abnormalities in patients with moderate to severe head injuries and subarachnoid hemorrhage. Different abnormalities such as: QT-interval, T-waves, U-waves, QRS-complex, ST-Segment, arterial and ventricular flutter and PAC, heart arrhythmia, angina pectoris and blood pressure changes were recorded and analyzed. The most common electrocardiographic change was ST-segment depression (67%).

This report tries to identify a rational relationship among the severity of head injury, site of lesion and level of consciousness at the time of admission, different surgical procedures, site of operation, prognosis and cardiac abnormalities. The presence of the complete pathway for sympathetic outflow from the orbital-frontal cortex to the limbic system via stellate ganglia to the heart is also discussed.

In conclusion cardiac abnormalities can greatly increase the morbidity and mortality of patients with intracranial pathologies. Emphasis is made on timely prevention and treatment of cardiac abnormalities preferably by stabilization of homeostasis of the brain condition by medical and surgical techniques; anti-arrhythmic drugs should be avoided unless strongly indicated. Obviously ventricular flutter and fibrillation must be treated with countershock and anti-arrhythmic drugs.

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INTRODUCTION

Different cardiac arrhythmias such as fibrillation and PVCs that can cause functional changes of the brain are very common findings in heart diseases but there have been very few studies on the effect of different pathologies of the brain on functional and structural changes of heart rhythm. Cardiovascular changes especially in acute brain disorders are frequently seen and it is a mutual relationship, as heart effects brain function and brain effects heart rhythm. There is a close relationship between cerebrovascular accident, subdural hematomas and subarachnoid hemorrhage, intracerebral hemorrhage, thrombotic and embolic cerebral lesions, and cardiovascular malfunction, which have been seen for 100 years.[1,2,3]

Electrocardiographic (EKG) changes appear in intracranial space occupying lesions as a result of increased sympathetic discharge which is due to widespread connections between the cerebral cortex, hypothalamus and fronto-orbital cortex (frontal-hypothalamic) tract. The presence of a complete sympathetic pathway for conveying the discharges from the orbital frontal cortex via the stellate ganglion of the cervical sympathetic chain is hypothesized. These changes include rhythm abnormalities, changes in different waves of EKG and also changes in blood pressure and probably heart failure.

These disorders are not only observed in subarach-

noid hemorrhage but are also seen in all intracranial lesions. Especially in terminal stages of intracranial space occupying lesions these diseases are more frequently seen.[4,5] The more severe the brain lesion and the more involvement of the hypothalamus and medial portion of the brain, the more likely cardiovascular disorders will be. 90% of all victims of acute brain lesions including spontaneous and traumatic subarachnoid hemorrhage, and spontaneous and traumatic intracranial hemorrhage, develop electrocardiographic changes.[2] So proper and timely diagnosis and treatment of these are of utmost importance. This article presents 100 cases with acute brain lesions in a Neurosurgical Intensive Care Unit (NS.ICU) who were studied for cardiovascular dysfunction.[6,7]

MATERIAL AND METHODS

This study was an explanatory cross-sectional and a sequential random sampling of all head injured and SAH patients admitted to Mashhad NeuroTrauma Center for one year (1995-1996). One-hundred cases were selected and daily physical examinations and EKGs were done. Localization of the intracranial lesion was on the basis of CT scan findings. Information was also obtained by interviews and questionnaires of all in-patients.

All patients with previous cardiovascular diseases, hypertension, and older that 40 and younger than 18 years were excluded from this study. Systolic blood pressure less than 90 was considered as hypotension and systolic blood pressure above 160 and diastolic pressure above 95 were taken as hypertension. Patients with risk factors such as hyperlipidemia, diabetes, and cigarette smoking more than 20 per day were also excluded. The study was done in three groups having intracranial SOL due to head injury, cerebrovascular accident, and SAH, totally 100 cases. Electrocardiographic changes such as rhythm abnormalities, wave changes, changes in intervals and segments of QRS complexes in one cycle of electrical activity of the heart were studied. Also studied were changes in blood pressure, heart rate, angina, heart failure and myocardial lesions.

RESULTS

Table I shows the relationship of cardiac abnormalities and Glasgow coma scale (GCS).[19] A negative T wave appeared in 67% of patients, mostly with a GCS of 4-6; 27% of the cases developed life threatening rhythms, ventricular fibrillations or flutters (Table I) which needed electroshock conversion. Table II shows the cardiac changes in relation to localization of intracranial SOL as seen in CT scans. According to these results 88% of the patients with hypothalamic lesions, 80% with limbic lesions and 76% of frontal lesions had cardiac abnormalities. The highest changes were an increased heart rate, 95% increased blood pressure and the least changes were atrial fibrillation in 4% and ventricular fibrillation in 2%. Table III shows cardiac changes in SAH corresponding to Hess and Hunt's grading of SAH (Table IV).

Table III shows grade 4 of SAH had the greatest cardiac changes (82%), followed by grade 3. The most important finding in SAH was negative T waves and only one patient in grade 2 of Hess and Hunt had T inversion. The rest of the cases were in grade 3 and 4. One patient developed angina and two patients developed acute myocardial infarction, which recovered without sequelae.

DISCUSSION

This study shows close similarities between electrocardiac findings and cardiovascular changes in SAH and head injuries.[10] This similarity increases with the severity of the head injury and lesions approaching the hypothalamus and medial structures of the brain. Since in other slow growing intracranial SOL or cerebrovascular accidents such a similarity is not observed, it seems the rapidity of intracranial space occupying lesion formation, neighboring of the lesion to important frontal temporal tract (frontal basilar area of limbic system) or compressing the basal ganglia of the brain are the most important factors in producing such cardio-

Table I. Cardiac changes versus GCS.

GCS / Heart Rate Changes	13-15	10-12	7-9	4-6	Total
↑Q-T			3	4	7
↑-wave		1	15	12	28
↓T-wave		5	28	34	67
↑S-T			3	5	8
↓S-T			9	12	21
U-wave					
Q-wave					
↑BP	3	10	16	2	31
↓BP		2	8	23	33
↑HR		8	10	8	26
↓HR				3	3
SVT			15	4	19
A-FLT			5	5	
A-fib			2		2
PVC	2	4	8	12	26
PAC		3	9	11	23
V-FLT			3	4	7
V-F			8	12	20

Table II. Cardiac changes versus IC.SOL.

Site of lesion / Heart rhythm changes	Frontal Lobe	Frontobasal Orbitofrontal	Anterior frontal	Insular angular	Limbic lobe	Hypothalamus	Cerebellar	Total
↑Q-T						3	8	11
↑T-wave				3		6	11	20
↓T-wave	15	28	12	7		5		67
↑S-T						10	11	21
↓S-T						5	11	16
U-wave								
Q-wave								
↑BP	18	9	3	25	10	11	11	87
↓BP	2	3	1	4	11	10	9	40
↑HR	30	23	13		8	4	17	95
↓HR	2	5	1	4	5	10	11	36
SVT								
A-FLT							2	2
A-fib			1			3		4
PVC	10	3	3	8	10	10		44
PAC	1		2				3	6
V-FLT		3						3
V-F					1	1		2
LAD								1
TOTAL	76	74	37	51	80	88	53	

vascular abnormalities.[8,9]

Presence of widespread connections between hypothalamus and frontal orbital cortex and their relationship with sympathetic nerve cells of gray matter of dorsal spinal cord through the hypothalamic efferent tract in the human brain is well known. And the hypothesis of sympathetic efferent pathway which starts from the orbital frontal cortex and limbic system that goes to the heart through stellate ganglia seems reasonable.

Stretch or compression of delicate vessels at the base of the brain that nourishes the diencephalon due to any reason including aneurysmal rupture, hematoma formation, stroke, tumors, or any intracranial SOL might cause diencephalic ischemia and culminate in cardiovascular abnormalities such as different dysrthymias.[11] changes in consciousness or sudden death.[4,6] Electrocardiographic changes are the most usual cardiovascular abnormalities which are observed in acute brain lesions of different etiologies. 90% of the victims of acute brain lesions have electrocardiographic changes in rhythm and polarization;[2] in our study this number was 95%. Reported abnormalities including sinus bradycardia which can become dangerous because it may culminate to premature multi-focal atrial contractions, ventricular tachycardia and fibrillation, supraventricular tachycardia (SVTAC) and atrial fibrillation are also reported.[1,7,8,12]

T wave changes (Neurogenic T waves) that are deeply negative or become increasingly positive are an important finding. This wave is observed in 50% of frontal lobe hemorrhages especially in the left orbital frontal area (Broadman Area 13).[2,13] QT interval prolongation and disappearance of U and Q waves is another important finding.[14] Most researchers believe cardiovascular abnormalities in acute brain lesions are the result of in-

Table III. Cardiac changes v/s Hunt and Hess SAH grading.

Finding \ Hunt & Hess Grading	I	II	III	IV	V	Total
Acute M.I	1				1	2
ANGINA				11	1	1
E.V.TAC		2	8		4	25
V.FIB		2				
V.FLT					1	
PAS	1		2	9		4
PVC		4	3	3	1	9
A. FIB		4	1		1	8
A.FLT			1	4		2
SVT		5	3	17		12
↑HR		3	12	3		32
↓HR		17	8			28
↑BP		2	1	5		3
↓BP		3	8			16
Q-wave	1				1	2
U-wave					2	
ST segment depression			1	3		3
ST segment elevation	1	2	4	10		10
Negative T-wave	1	3	21	4		35
Positive T-wave		2	1	4	2	9
Q-T		1	7		8	20

Table IV. Hunt-Hess SAH classification.

Grade	Description
0	unruptured aneurysm
1	asymptomatic or mild H/A and slight nuchal rigidity
1a	no acute meningeal/brain reaction but with fixed neurodeficit
2	Cr. N. palsy (e.g.III, IV), moderate to severe H/A, nuchal rigidity
3	mild focal deficit, lethargy or confusion
4	stupor, moderate to severe hemiparesis, early decerebrate
5	rigidity, deep coma, decerebrate rigidity, moribund appearance

Add one grade for serious systemic disease (e.g. HTN, DM, severe atherosclerosis, COPD) or severe vasospasm on arteriography.

creased catecholamine secretion in blood (catecholamine storm) that cause such a serious cardiac change,[15,16] also microscopic studies have shown myofibrial degeneration[17,18] On the other hand steroids released in parallel to catecholamines strengthen the myocardiac lesion from such a stressful condition.[18] Premature ventricular contractions and more than 30 degrees of heart axis deviation in frontotemporal lobe lesions is another finding.[19] In our patients 11% had this (10% in frontal lobe lesion, 1% due to anterior frontal lobe lesion). Irritation of frontal lobe, orbital cortex, anterior temporal lobe area, insular gyrus and angular gyrus almost always cause changes in the S-T wave segment. Lesions in the limbic system, hypothalamus and central gray matter cause ectopic ventricular contractions (EVC) which are due to passage of discharges through unusual pathways to the ventricle.[2,20] In our patients this abnormality was seen in grade 3 of Hunt and Hess of SAH (Table IV). According to Torner 22% of SAH patients develop such electrocardiographic changes.[12] In Estanol and collegue's report 20% of SAH patients had developed tachyarrhythmia, increased Q-T intervals, and malignant ventricular tachycardia.[4] Page and colleagues found 34% negative T waves in SAH, and bradycardia in 32%.[13] Diencephalic vascular rupture is always associated with obvious cardiovascular changes,[15] and increased systolic blood pressure is another finding.[15] Harvey Cushing's studies showed increased intracranial pressure is always accompanied by systemic vascular resistance which may cause a severe decrease in cardiac output, pulmonary vein congestion and pulmonary hemorrhagic edema.[21]

CONCLUSION

Treatment of cardiovascular abnormalities following acute brain lesions is of utmost importance and should preferably be done without use of drugs acting on the heart to regulate its rhythm, but by restoration of intracranial structures and functions to a normal millieu by medical or surgical techniques. For dangerous arrhythmias such as ventricular flutter or fibrillation, use of electrocardioshock is recommended. Lidocaine is the drug of choice for prevention of ventricular tachyarrhythmias and ventricular extrasystoles. The dosage is 1-1.5 mg/kg body weight as an intravenous bolus and in case of unrelenting tachycardia an extra dose of 0.5mg/kg body weight is given. And the maintenance dose is 1-4mg/minute (20-60 microgram/kg/minute).[21] The drug is stopped as soon as the arrhythmia disappears. Beta-blockers are also indicated for prevention of deleterious effects of catecholamines on the heart.

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