Results

Results:

The present work was conducted on26 patients who were asked to particopate in this study. They were 11 males and 15 femals and their ages ranged from 6 to 41years (mean 21. 61 years).

Pre and postoperative audiometric evaluation included air conduction , bone conduction pure tone measurements, SRT, and speach discrimination tests. Comparison was made between the preoperative and postoperative audiograms of all patients . All patients had the below listed drugs perioperatively:.

- Morphine sulphate (0.08-0.1 mg/kg) .
- Midazolam (0.02-0.04 mg/kg) . Intravenously 10 minits befor operation.
- Fentanyl (25-75 ug/kg).
- Midazolam (0.1-0.2 mg/kg).
- Vecronium (0.07-0.1 mg/kg).
- 100% oxygen
- Intermittent halothane (as needed).
- Epineprine and / or dopamine during and after weaning from bypass 0.01: gm/kg/m).
- Dextrose 5% - 1/4 Ringer lactate 30 ml /k .
- Na HCO_3 5% .
- Mannitol 25 % (1/4 gm/k) .

- Heparin (300 Lu.1 liter of the prime.).

- Antibiotic . Cloforan 1 gm as a single does in the prim.

. Nebcin 80 m gm as a single dose in the prim.

- Lasix : when hypovolaemia occure.

- HCl : according to need.

The type of performed operation, age and sex of each patient are shown in table (2) :

Table (2)

Case No.	Age	Sex	Type of Operation
1	19 Years		Open mitral Vulvotomy
2	35 Years		Open mitral Vulvotoy + left atrial thrombus
3	15 Years		Mitral vulve replacement
4	17 Years		Open mitral Vulvotomy
5	28 Years		Aortic Vulve replacement
6	6 Years		Excisions of subaortic membrane
7	15 Years		Mitral Vulve replacement
8	19 Years		
9	19 Years		Mitral Vulve replacement
10	14 Years		Repaired V.S.D
11	22 Years		Mitral Vulve replacement
12	13 Years		Mitral Vulve replacement
13	14 Years		Subaortic membrane excision
14	24 Years		Aortic Vulve replacement
15	30 Years		Mitral Vulve replacement
16	28 Years		Mitral Vulve replacement
17	19 Years		Aortic Vulve replacement
18	20 Years		Mitral Vulve replacement
19	41 Years		Open mitral Vulvotomy
20	9 Years		Open pulmonary Vulvotomy
21	30 Years		Mitral Vulve replacement
22	40 Years		Mitral Vulve replacement
23	20 Years		Mitral Vulve repair
24	9 Years		A.S.D. repair
25	15 Years		M. Vulve repair
26	27 Years		A.S.D. repair

Three patients died during the operation . In two of them (case No.4 and Case No. 8) death was attributed to right ventricular faileur due to sever postoperative pulmorary hypertension. In the third case (Case No. 9), death was atlributed to massive air embolism.

The bypass time (perfusion time) and the aortic clamp time (Ischaemic time) for each patient are shown in table (3).

Table (3)

Case No.	Bypass time (perfusion time)	Aortic clamp time (Ischaemic time)
1	120 m	44 m
2	130 m	75 m
3	142 m	71 m
4	142 m	62 m
5	142 m	100 m
6	60 m	33 m
7	165 m	86 m
8	died	died
9	168 m	91 m
10	79 m	33 m
11	153 m	114 m
12	110 m	65 m
13	73 m	26 m
14	122 m	89 m
15	150 m	93 m
16	220 m	90 m
17	107m	75 m
18	91 m	63 m
19	200 m	106 m
20	63 m	15 m
21	217 m	89 m
22	202 m	116 m
23	89 m	49 m
24	58 m	28 m
25	130 m	83 m
26		

The length of time on bypass varied from 60 to 120 minutes, the mean being 130 . 54 .It exceeded 200 minutes in two patients and neither patients showed increase of hearing thresold more than 5 dB.

The aortic clamp time varied from 15 to 116 minutes,the mean being 70 . 67 minutes in two cases it was more than 100 minutes and non of them showed increase of hearing threshold.

The pre and postoperative hearing threshold level at 250,500,1000,2000,4000 and 8000 HZ frequencies, SRT, speach discrimination score, and tone decay pre and postoperatively were recorded in Table (4) , Table (5), Table (7) and Table (9). Table (5) and Table (6) showing changes in the threshold comparing the pre and postoperative pure tone audiograms in right ear and left ear respectively.

Table (4) : Right ear changes in the threshold comparing pre and postoperative PT audiograms

Case No.	Air Conduction						Bone conduction					
	250 Hz	500 Hz	1 KHz	2 KHz	4 KHz	8 KHz	250 Hz	500H Hz	1 KHz	2 KHz	4 KHz	8 KHz
1	+5	+5	+5	+5	+5	+5	-	+5	+5	+5	-	-
2	+5	+5	-	-	-	+5	-	-	-	+5	-	-
3	+5	+5	+5	+5	+5	+5	-	+5	+5	+5	+5	-
4	Died during operation											
5	-	-	-	-	-	-	-	-	-	-	-	-
6	-	-5	-	-5	-	-5	-	-5	-	-	-	-
7	+5	+10	+5	+5	+10	+10	-	+10	+5	+10	+5	-
8	Died during operation						-					-
9	Died during operation						-					-
10	-	+5	+5	-	-	+5	-	+5	-	-	-	-
11	-	-	-	-	-	-	-	-	-	-	-	-
12	+5	+10	+5	+5	+10	+5	-	+10	-	+5	+10	-
13	-	-	-	-	-	-	-	-	-	-	-	-
14	-	-	-	-5	-	-5	-	-5	-	-	-5	-
15	-	+5	-	+5	-	+5	-	-	-	+5	+5	-
16	-	-	-	-5	-5	-	-	-	-	-5	-5	-
17	-	+5	-	+5	+5	-	-	+5	+5	-	-	-
18	-	-5	-10	-10	-5	-5	-	-	-5	-	-10	-
19	-	-	-5	-5	-5	-5	-	-5	-	-5	-5	-
20	-	-	-5	-5	-	-	-	-	-	-	-	-
21	+5	-	+5	-	-	-	-	-	-	-	-	-
22	-	+5	+5	-	-	-	-	+5	+5	-	-	-
23	+5	-	-	-	+5	+5	-	-	-	+5	-	-
24	-	-5	-	-5	-	-	-	-5	-5	-	-	-
25	+5	+5	-	+5	+5	68	-	+5	-	-	+5	-
26	-	+5	-	-	-	+5	-	+5	-	-	-	-

Table (6) : Showing summary of changes of hearing threshold pre and postoperatively

changes in threshold	No of cases
No changes	3 cases
Decrease thresholdby (5 dB)	6 cases
Decrease threshold by (10 dB)	1 cases
Increase threshold by (5 dB)	11 case
Increase threshold by (10 dB)	2 cases
Total	23 cases

As shown in table (6) there was postoperative increase of threshold of hearing of 10 dB in 2 cases. In 11 cases there was an increase of threshold of 5 dB, and in 2 cases threre was an increase of threshold of 10 dB .Only one patient showed improvement of hearing (decreased threshold by 10 dB) postoperatively, and 6 patients showed decreased threshold of 5 dB postoperatively while 3 cases showed no changen in threshold of hearing .

Table (7) : showing changes in SRT comparing pre and post-operative

Case No.	Changes in SRT	
	Right ear	Left ear
1	+5 dB	+5 dB
2	-	-
3	+5 dB	+5 dB
4	Died during operation	
5	-5 dB	-5 dB
6	-5 dB	-
7	+10 dB	+10 dB
8	Deid during operation	
9	Died during operation	
10	+ 5 dB	-
11	+5 dB	-
12	+ 10 dB	+10 dB
13	-	-5 dB
14	-	+5 dB
15	+5 dB	-
16	-	-5 dB
17	-5 dB	-
18	-5 dB	-
19	-5 dB	-
20	+5dB	-
21	-	+5 dB
22	+5 dB	-
23	+5dB	+5 dB
24	+5 dB	-
25	-5 dB	-5 dB
26	+5dB	+5 dB

Table (8) : Showing summary of pre and postoperative changes in SRT

Changes in SRT	No of patients
* No changes	one case
* Less than 5 dB	8 cases
* More than 5 dB	12 cases
* More than 10 dB	2 cases
Total	32 cases

There was postoperative increase of speach reception threshold by + 10 dB in 2 cases, and by + 5 dB in 12 cases, while 8 cases showed decrease of SRT by 5 dB . No changes was recorded in only one case.

Table (9) : showing pre and postoperative changes in discrimination score

Case No.	changes in discrimination Score	
	Rt ear	Lt ear
1	- 2 %	- 2 %
2	-	-
3	- 2 %	- 4 %
4	Died during operation	
5	- 2 %	-
6	-	
7	- 4%	-4 %
8	Died during operation	
9	Died during operation	
10	-	
11	-	-
12	- 2 %	-
13	-	- 2 %
14	-	-
15	-	- 2 %
16	- 4 %	- 2 %
17	-	-
18	+ 2 %	- 2 %
19	- 2 %	+ 2 %
20	-	-
21	-	-
22	-	-
23	-	-
24	-	-
25	- 2 %	-
26	-	- 2 %
		+ 2 %

Table (9) : showing pre and postoperative changes in discrimination score

Case No.	changes in discrimination Score	
	Rt ear	Lt ear
1	- 2 %	- 2 %
2	-	-
3	- 2 %	- 4 %
4	Died during operation	
5	- 2 %	-
6	-	
7	- 4%	-4 %
8	Died during operation	
9	Died during operation	
10	-	
11	-	-
12	- 2 %	-
13	-	- 2 %
14	-	-
15	-	- 2 %
16	- 4 %	- 2 %
17	-	-
18	+ 2 %	- 2 %
19	- 2 %	+ 2 %
20	-	-
21	-	-
22	-	-
23	-	-
24	-	-
25	- 2 %	-
26	-	- 2 %
		+ 2 %

Table (10) : Showing summary of pre and postoperative changes of discrimination score

Changes of discrimination score	No of cases
* No changes	9 Cases
* + 2 %	3 cases
* - 2 %	8 cases
* - 4 %	3 cases
Total	23 cases

Discussion:

The developement of the extracorporeal cardiopulonary bypass apparotus had led to marked advances in the salvege of patients with previously incurable lesious. One of the complications of the use of this apparatus is the occasional reports of decreased hearing postoperatively.

In 1972 a case report was published by Arenberg. Since then, several case reports have followed, including one reported by Wright and Saunders (1975) , seven by Plasse et al., (1980) , two by Shapiro et al., (1981) and two by Millen et al., (1982).

Numerous aetiologies have been proposed for sudden loss of hearing . The most often cited are viral, viral-vascular, vasospasm from autonomic imbalance, intracochlear membrane breaks and purely vascular.

Javid et al., (1969), in a study on 100 patients after cardiac operations found a 23 % incidence of neurologic abnormalities .Lee and associates (1970) found central nervous system distrbance in 31% of patients undergoing cardiopulmonary bypass. The neurologic deficits and more precisely the sudden hearing loss in cardiopulmonary bypass is most probably a vascular insult (Millen et al., 1982). A vascular etiology could be on the basis of haemorrhage, thrombosis, vasospasm or emboli.

All the collected data on the reported cases of sudden hearing loss following the use of cardiopulmonary bypass apparatus are consistent with the hypothesis that emboli are the most likely cause of hearing loss . Such emboli occlude

part of the blood supply to one cochlea and as a result the hearing loss occured . Particulate matter from heavily calcified valve or generated by the cardiopulmonary byoass pump can cause emboli .

Dutton and Emnunds (1973) were able to detect the number , size , shape, and composition of the emboli produced during partial or total cardiopulmonary bypass . They designed a reliable method to measure the platelet aggregate emboli which occure continuously throughout bypass procedures . They found emboli at a rate of 2 - 3 per ml of circulated blood , ranging in size from 40 to 200μ in diameter . Study through electron mocrographs showed that such platelets , were undergoing intensive alteration in ultrastructure with a few leucocytes attached to the surface of the aggregates .

Millen et al . (1982) adopted the same hypothesis and attributed the hearing impairement to microemboli from the increased platelet numbers and adhesiveness present postoperatively .

Clamping of the aorta can dislodge atherosclerotic plaques . In over half of the patients reported with sudden unilateral hearing loss , pump times were longer than 150 min . In our series 8 patients had pump times of longer than 150 min . and only one of them showed , change in hearing threshold . However , there are other possible sources of emboli such as air bubbles induced by air entering the circulation at surgery , silicon antifoaming agents which was proved by both animal experiments and autopsy to be present intravaculaly (Lindberg , et al ., 1961) or fat which was demonstrated at autopsy in the lung , kideny or brain of 50 - 100% of patients who died following extracotporeal circultation (Miller , et al ., 1962) .

On the other hand , Bronson et al ., (1971) performed audiograms on 50 patients befor and after cardiopulmonary bypass , and non of the patients showed

any postoperative hearing loss.The main drawaback in their series was that patients were tested by different audiologists and audiometers making their study rather uncontrolled.

In the present work every effort was made to obtain a rigidly controlled study. The patients were always tested by the same audiologist using the same audiometer. In case where the schedule did not allow time for testing prior to surgery or when the patient was discharged before the postoperative test could be acomplished , the patient was dropped out of the study even though non of such patients complained of tinnitus or decreased hearing postoperativelly.

The hypothesis of emboli causing hearing impairement is challenged in our study by the fact that one could suspect that if microemboli were generated to a significant degree during bypass, this might be reflected in a marked change in the audiogram postoperatively. However , the result of the study do not indicate this. Moreover the very mild hearing loss mostly 5 dB or less reported in our series in 11 patients and of 10 dB in 2 patients occured almost bilaterally while emboli would be expected to cause unilateral loss as was reported previously by Arenberg (1972), Saunders (1975), Plasse et al., (1980) , Sapiro et al., (1981), and Millen et al., (1982).

Although the support for an embolic etiology appears strong, doubt is cast on this causation due to several factors : First, cochlear artery embolism has not yet been demonstrated histologically. Second , in most centers embolism has been less a problem with the use of membrane oxygenators, aortic venting , better fitteration and droppters (Shapiro et al., 1981) Third, Belal in 1980 found that total occlusion of the internal auditory artery resulted in total loss of cochlear

function. However, total loss of hearing has not been reported as would be expected with at least some embolic obstruction . Finally, and even more important, complexity of the cochlear blood supply(which was shown in the review) would make terminal branches an unlikely locale for the lodjement of an emboli.

As an alternative embolic hypothesis Shapiro et al., (1982) postulated a more general perfusion failure as an etiology. They theorized that atherosclerosis in the basilar artery system in combination with perfusion pressure which, on bypass, are maintained at levels less than diastolic pressure, resulted in a localized perfusion deficit. This phenomenon would be enhanced by the hypocapnia and its resultant vasoconstrictive effect in cardiac surgery.

The strongest support for the perfusion deficits is presented by Javid et al., (1969) who recorded a twofold increase in the neurologic deficits when the mean perfusion pressures fell below 50 mm of mercury . In all aur cases including the 2 cases who exhibited postoperative hearing loss of 10 dB perfusion pressure was above so mm of murcury .

It is worth noty that most of the potients in our series are young and they were subjected to valve replacement operation in contradistinction to the previously performed studies where the patients were subjected to coronary bypass and most patients in such studies were of old age . So a distinct difference exists our study and the previous studies . In the later, sudden hearing loss occured mostly in older age group with known pre- existing cardiovascular diseases (Millen 1982). This support the hyposthesis of emboli causing hearing loss in such series

In the present work, hearing loss of 5dB occured in 11 cases and of 10 dB in 2

patients. Any change greater than 5 dB is considered by most authorities as a signeficant change in pure tone audiometry (Brownson, et al .,1971) . So we have only 2 cases of significant hearing loss . In both cases the hearing loss was bilateral . This can be explained on the basis of perfusion failure theory. However the ototoxic drugs can not be denied as a cause of hering loss since the hallmark of these drugs is usually bilateral (Shapiro 1981) .

The embolic theory can not be judged in our study since all the included candidates were young with no history of arteriosclerotic cerebrovascular disease and no coronary byoass operations were available during the period of the study. The low possibility - if ever present - of artariosclerosis in young patients denies the emboli as an aetiology of postoperative hearing loss . In our zeal to find simple solutions to complex proplems , it may be that we are overlooking multi - factorial etiology encompassing both the embolic phenomenon and perfusion deficits theory .