Mercury is toxic to almost every organ of body, including central nervous system. The aim of present study was to see the effect of mercury on the cerebellum which may explain the clinical manifestation of mercury neurotoxicity.

Material and Method
A total number of 20 adult albino rats of either sex were included in the present study, consisting of equal numbers in both control and experimental groups. Experimental group received mercuric chloride in distilled water for a period of 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Cerebellum was dissected out. 10µ thick sections, obtained by usual histological procedure, were stained with H&E. On light microscopic observation, cerebellum from experimental group revealed marked congestion of the blood capillaries with prominent perivascular fibrosis. Cerebellum of experimental group also showed spongiosis in the molecular layer and markedly increased cellular granular cell layer with pyknotic nucleus. Few Purkinje cells could be seen. It was concluded that mercury has toxic effects on the central nervous system including the cerebellum which may explain the clinical manifestation of mercury neurotoxicity.

ABSTRACT
Mercury is toxic to almost every organ of body, including central nervous system. The aim of present study is to observe the histopathological changes in the cerebellum of rat induced by oral administration of mercuric chloride in adult albino rats. A total number of 20 adult albino rats of either sex were included in the present study, consisting of equal numbers in both control and experimental groups. Experimental group received mercuric chloride in distilled water for a period of 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Cerebellum was dissected out. 10µ thick sections, obtained by usual histological procedure, were stained with H&E.

Introduction
Mercury intoxication can occur more through inhalation rather than ingestion of contaminated foods and drinks. Deposit of mercuric chloride in adult albino rats. A total number of 20 adult albino rats of either sex were included in the present study, consisting of equal numbers in both control and experimental groups. Experimental group received mercuric chloride in distilled water for a period of 15 days, then animals of both groups were anaesthetized with ether and perfused with 10% formalin. Cerebellum was dissected out. 10µ thick sections, obtained by usual histological procedure, were stained with H&E.

Material and Method
A total number of 20 adult albino rats (10 male and 10 female) weighing approximately 120g were used in the present study. 10 rats with equal number of either sex were treated with mercuric chloride (0.330 mg/kg body weight) while the remaining 10 rats (5 male and 5 female) served as control group and were given distilled water. Freshly prepared sterile solution of mercuric chloride in distilled water was used for oral administration as drinking water. This concentration was ascertained after a careful trial in order to find maximum survival days, which were 15 days. Then, rats were anaesthetized with ether and perfused with buffered 10% formalin. Brain was dissected out. 3mm thick coronally sliced pieces of cerebellum were removed and processed for paraffin embedding. Then, 10 µ thick sections were cut with rotary microtome. These sections were stained with H&E and observed under the light microscope.

Observations
On examination, under the light microscope, cerebellum (control group) shows normal 3 layers; Outer molecular, Inner extremely cellular granular cell layer and single layer of Purkinje cells in between the two layers. Haematoxylin and Eosin × 40X (Figure 1). The cerebellum of experimental group showed marked congestion of the blood capillaries with prominent perivascular fibrosis (40X (Figure 2)). Cerebellum of experimental group also showed spongiosis in the molecular layer and markedly increased cellular granular cell layer with pyknotic nucleus. Few Purkinje cells can be noted at 40X (Figure 3).

Figure 1: Photomicrograph of cerebellum (control group) shows normal 3 layers; Outer molecular, Inner extremely cellular granular cell layer and single layer of Purkinje cells in between the two layers. Haematoxylin and Eosin × 40X.

Figure 2: Photomicrograph of cerebrum (experimental group) shows marked congestion of the blood capillaries with prominent perivascular fibrosis. Haematoxylin and Eosin × 40X.
Discussion
In the present study, histological findings were suggestive of neurotoxic and degenerative effects of mercury on the cerebel-
num. These findings are in conformity with other neurohisto-
logical studies. In one of the studies, cerebellum showed pyk-
nosis of nuclei of granular layer cells, edema between granular
and molecular layer, degeneration of many purkinje cells (11),
these findings were found in the present study also. Earlier, It
was also reported that mercury has neurotoxic effects reflected
in its ability to penetrate and damage Blood Brain Barrier sys-
tem (12), (13). Another study, reported pathological lesions in
brain secondary to mercuric chloride poisoning (14). It has been
documented that treatment of rats with mercuric chloride re-
sulted in mild signs of neurotoxicity in the first two weeks of
treatment, like congestion of blood vessels perivascular reac-
tion in the cerebrum, cerebellar white matter showed multi-
ple well defined vacuoles with purkinje cells remaining intact
while in the third week, pericellular edema with ischemic injury
to some neurons and beginning of purkinje cells damage were
evident (15). It has also been reported that degeneration and ne-
crosis of purkinje cells and neuronal shrinkage was due to direct
toxic effect of the compound or the role of mercuric chloride as
anoxic agent(16). Reports are available, showing degeneration
and necrosis of purkinje cells and shrinkage of neurons in cer-
ebellum secondary to mercury poisoning (17). Mercury com-
ounds can induce cerebellar degeneration, sensorimotor and
gating disturbances, tremor, ataxia and depression (18,19,20).
The histological findings observed in our study confirmed the
cerebellar neurotoxicity following mercury poisoning and cor-
related very well with histological findings of the other studies.

Conclusion
Exposure of rat to mercury for 15 days produces demonstrable
microscopic alterations in the cerebellum.

REFERENCE