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Effects of the Administration of a-Methyl-p-Tyrosine on the Striatal Dopamine Increase and the Behavioral Motor Disturbances in Rats Exposed to High Pressure[1]

J. H. ABRAINI AND J. C. ROSTAIN

Laboratoire de biologie des hautes pressions, CNRS URA 1330, Faculté de médecine nord boulevard Pierre Dramard, 13326 Marseille cédex 15, France

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ABRAINI, J. H. AND J. C. ROSTAIN. *Effects of the administration of a-methyl-p-tyrosine on the striatal dopamine increase and the behavioral motor disturbances in rats exposed to high pressure*. PHARMACOL BIOCHEM BEHAV **40**(2) 305–310, 1991.—When human divers and experimental animals are exposed to increased environmental pressure, they develop the high pressure neurological syndrome (HPNS). Moreover, it has been recently demonstrated that pressure exposure induced an increase in striatal dopamine (DA) release. In this study, the effects of intracerebroventricular administration of a-methyl-p-tyrosine on the pressure-induced striatal DA increase, and the behavioral motor disturbances of HPNS, including hyperlocomotor activity (HLA), tremor, and myoclonia were monitored in free-moving rats. Striatal DA release was monitored by in vivo voltammetry, and behavioral symptoms using piezoelectrical sensors. Results suggested that the pressure-induced striatal DA increase could be the consequence of a release in both newly synthesized and vesicular DA. Elsewhere, data also confirmed that the pressure-induced DA disturbances would be involved in the development of HLA.

Voltammetry Dopamine Striatum Motor behavior High pressure

HIGH pressure is a basic etiological factor underlying central nervous system changes referred to as the high pressure neurological syndrome (HPNS). This syndrome is observed when human divers or experimental animals are exposed to pressure greater than 20 bars in a helium-oxygen breathing mixture.

The principal symptoms of HPNS include EEG changes, sleep disturbances, tremor, and myoclonia. Animals also developed hyperlocomotor activity (HLA), and at higher pressures convulsions and epileptic seizures. These symptoms have been described by several authors (5, 8, 13), and reviewed elsewhere (15). In man, other effects include problems of motor coordination and loss of attention (20). Elsewhere, it has also been reported in divers schizophrenic disorders including paranoia, delirium and ambulatory activity (24, 25, 27). These mental disorders are generally considered according to the dopaminergic hypothesis as the result of a dopamine (DA) hyperactivity [for review see Lemoal (21)]. Accordingly, several authors have suggested that monoamine neurotransmitters could have a role in the occurrence of some of these symptoms. Indeed, recent in vitro and in vivo studies have demonstrated that high pressure induced an increase in extracellular striatal DA level (1, 12, 22, 23); and HLA has been found to be correlated with the pressure-induced striatal DA increase.

Thus we report here a neurochemical and behavioral study, performed during pressure experiments on free-moving rats, on the effects of the use of a-methyl-p-tyrosine (AMPT), a blocker of DA synthesis. In vivo measurements of extracellular DA level were performed using voltammetry and a multifiber carbon electrode developed by Forni (10), which has been demonstrated to be selective for DA, by in vitro and in vivo experiments (9,11). Behavioral disturbances including HLA, tremor, and myoclonia were monitored using piezoelectrical sensors as described previously by Tomei et al. (28).

METHOD

Animals

Male Sprague-Dawley rats (n = 22) weighing 300–350 g at time of surgery were used. Rats were housed at 21 ± 0.5°C in individual altuglass home cages under a 12–12-h light-dark cycle (lights on from 7 a.m. to 7 p.m.), with free access to food and water.

Drug Treatment

Saline experiments were performed in rats intracerebroventricularly (ICV) injected, 1 h before pressure exposure, with 10

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μl phosphate-buffered saline solution (PBS).

AMPT, an inhibitor of DA synthesis (Sigma, St. Louis, MO), was injected at 10^{-7} M (i.e., 50 μg/kg) in 10 μl PBS, 1 h or 3 h before pressure exposure (in each case: behavioral data, n = 4; neurochemical data, n = 4). This dose of AMPT, ICV injected, has been demonstrated in experiments performed under atmospheric pressure to decrease extracellular DA level over a period of time of more than 20 h with a maximal decrease of about 25% (2).

Exposure to Pressure

The unrestrained animals were placed in separate Altuglass cylinders, bored to enable an appropriate gas mixing. Cylinders were installed in a 50-liter pressure chamber (maximum pressure 200 bar) in which the 12–12-h light-dark regime was maintained. Rats were compressed to 80 bar of relative pressure (i.e., 81 bar absolute, equivalent of 800 m of sea water) with helium at a rate of 1 bar/min, 1 h after being injected with PBS or AMPT, or 3 h after being injected with AMPT. Oxygen was maintained at a constant partial pressure of 0.4 bar which is the partial pressure generally used in human dives. The CO_2 was less than 0.0003%. Humidity was controlled and temperature was progressively increased from 25 to 33°C to prevent hypothermia and to maintain the comfort of the animals, because of the important specific heat of helium as compared to air. The stay at the maximal depth lasted 4 h, and the decompression 24 h. Animals were decompressed at a rate of 0.06 bar/min from 80 bar to 12 bar and 0.04 bar/min from 12 bar to atmospheric pressure. During decompression partial pressure of oxygen was 0.5 bar. All the animals survived the hyperbaric experiments.

Neurochemistry

Differential pulse voltammetry was used according to the method developed by several authors (3, 14, 17, 19). Voltammetric measurements were performed on unrestrained awake animals using a PRG5 polarograph (Tacussel, France), and a classical 3-electrode potentiostatic system with reference, auxiliary, and working electrodes. Multifiber carbon electrodes were built as described previously by Forni (10), and electrochemically pretreated by applying a triangular wave potential of 0–3 V, 70 Hz, 20 s; 0–2 V, 70 Hz, 20 s; and 0–1 V, 70 Hz, 15 s (9,11).

Before being implanted the working carbon electrodes were calibrated in vitro to control their selectivity to DA in solutions from 10^{-8} M to 10^{-3} M of dihydroxyphenylacetic acid (DOPAC), ascorbic acid, uric acid, homovanillic acid, and norepinephrine. DA at 10^{-5} M was used to check that the DA oxidation peak was found at around 160 mV vs (range: 150 mV–170 mV) (9,11). Moreover, the implanted animals were injected with 10^{-7} M pargyline, a monoamine oxydase inhibitor which blocks DOPAC formation, to check in vivo that the electrochemical signal essentially resulted from DA oxydation.

Working carbon electrode and stainless steel cannula were stereotaxically implanted, according to the atlas of Konig and Klippel (18), in the caudate nucleus (A: 8.62; L: 2; H: 1.4) and the right lateral ventricle (A: 5.91; L: 1.4; H: 2) of the animals, under general anesthesia (pentobarbital sodium 30 mg/kg IP and ketamine 100 mg/kg IM). The reference and auxiliary electrodes (stainless steel screws) were fixed to the bone. The electrodes were attached to a miniconnector and electrodes, connector, and cannula were held in place with dental cement (resin cement, Ivoclar, Switzerland). After surgery, the animals were allowed to recover one week.

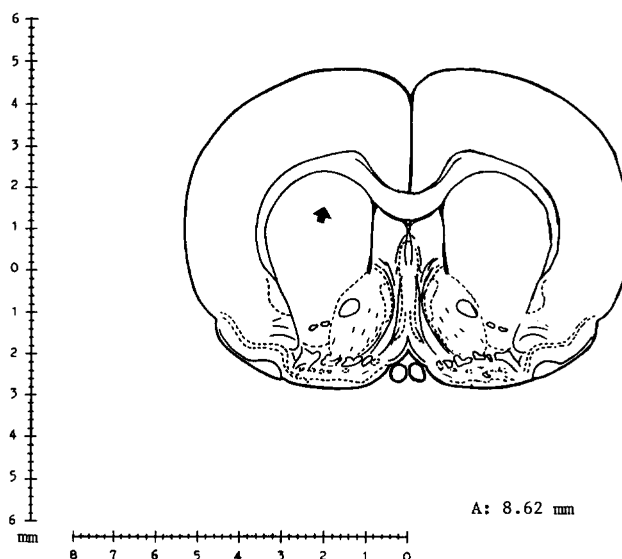

FIG. 1. Bilateral typical emplacement of working electrodes in the dorsal caudate-putamen of a Sprague-Dawley rat. Comparison with the stereotaxic coordinates indicated by the arrow (A: 8.62; L: 2; H: 1.4) can be seen according to the atlas of Konig and Klippel. Tissue sections were stained with cresyl violet.

For voltammetric recordings, the animals were connected to the polarograph through a flexible cable and a swivel connector and the polarograph was set to the following parameters: scan rate 20 mV/s; voltage range 0 to 1000 mV vs; pulse modulation amplitude 50 mV; pulse modulation duration 48 ms; pulse period 0.2 s. Voltammograms were recorded every 3 min. Extracellular DA release was quantified automatically by measuring the height of the DA oxidation peak, using a computerized device. Results from PBS experiments or AMPT experiments were compared to the mean of the DA peaks recorded during a 2-h control period before injection, taken as the 100% value (DA control value).

In some animals, the location in the caudate-putamen of the mulfiber carbon working electrodes was histologically checked after pressure exposure, referred to the atlas of Konig and Klippel (18) (Fig. 1).

Behavioral Analysis

Behavioral analysis was performed as described previously by Tomei et al. (28). Behavioral symptoms of HPNS were deduced

from piezoelectrical sensors fixed under the floor of each altuglass cylinder; the signals were quantitatively analysed on a PC-AT compatible computer and decomposed on line into HLA, tremor and myoclonia. Tremor was calculated from the signals recorded on the 10–16 Hz band frequency (4,29); myoclonia were detected as signals of unusually high amplitude (with a threshold of detection adjustable of each rat); and HLA, as the whole signal minus myoclonia and tremor. HLA, tremor, and myoclonia were expressed in arbitrary units (U).

Statistical Methods

Nonparametric statistics were used such as the Wilcoxon sign-rank paired *t*-test (W-test), the Mann-Whitney U-test (U-test) and median value ± the 25th–75th percentiles. W-test was used for control experiments to analyze the effects of pressure on DA level and rat's behavior (before and after pressure exposure); U-test was used to compare data obtained in injected animals during drug experiments to the corresponding data (same pressure during compression and decompression, and same time during the 4-h stay at 80 bar) recorded in noninjected animals during control experiments (26).

RESULTS

PBS Experiments

Compression to 80 bar was found to induce an 18% increase (25th–75th percentiles: +10%, +26%; W-test, $p<0.05$) in the amplitude of the DA electrochemical response. During the 4-h stay at 80 bar, no further change in signal size was seen. During decompression the DA signal progressively declined, reaching a decrease of −4% of the control value when the pressure had fallen to atmospheric pressure (Fig. 2A).

Compression led to a sustained increase of HLA (W-test: $p<0.05$) (Fig. 3A), tremor (W-test: $p<0.05$) (Fig. 4A) and myoclonia (W-test: $p<0.05$) (Fig. 5A). During the 4-h stay and decompression phases, all of the symptoms disappeared progressively.

Effects of AMPT Injected 1 h Before Pressure Exposure

During the precompression phase at atmospheric pressure, administration of AMPT 1 h before pressure exposure resulted in a striatal DA decrease of −5% at time of compression (25th–75th percentiles: −4%, and −7%; U-test: $p<0.05$). During compression, no further change in signal size was seen as compared to the DA control value. During the stay and decompression phases, striatal DA release progressively decreased to a value of −13% of control when the pressure had fallen to atmospheric pressure (25th–75th percentiles: −8%, and −20%) (Fig. 2B).

At a behavioral level, as compared to PBS experiments, administration of AMPT 1 h before pressure exposure was found to temporarily decrease HLA during compression at around 60–70 bar (U-test: $p<0.05$) (Fig. 3B), to decrease tremor (U-test: $p<0.05$) (Fig.4B), and to enhance myoclonia (U-test: n.s.) (Fig. 5B).

Effects of AMPT Injected 3 h Before Pressure Exposure

During the precompression phase at atmospheric pressure, administration of AMPT 3 h before pressure exposure resulted in a striatal DA decrease of −12% at time of compression (25th–75th percentiles: −8%, and −23%; U-test: $p<0.05$). Compression slightly increased striatal DA levels from −12% (25th–75th percentiles: −8%, and −23%) to −6% (25th–75th percentiles: −4%, and −9%) of the DA control value. During the 4-h stay at 80 bar, striatal DA level showed no major change. During the decompression, the striatal DA level progressively declined, reaching a decrease of −22% (25th–75th per-

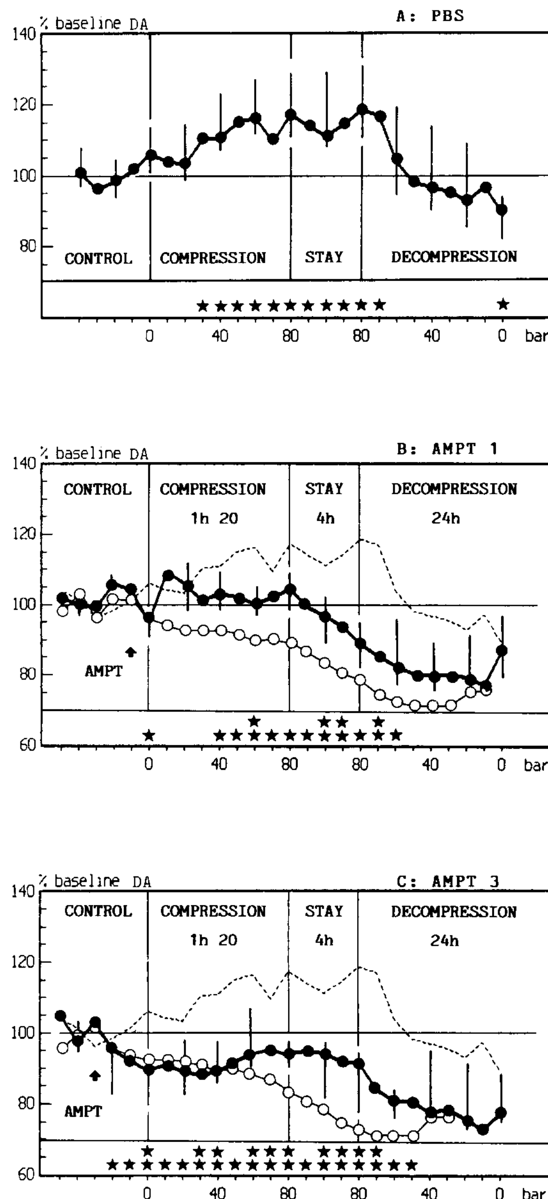

FIG. 2. Development of the extracellular DA concentration recorded from the caudate-putamen of free-moving rats exposed to 80 bars of high pressure. Left: compression up to 80 bar; duration 1 h 20 min. Middle: stay at 80 bar; duration 4 h. Right: decompression from 80 bar to atmospheric pressure; duration 24 h. Y-axis: extracellular DA level expressed as a percentage from control value (120 min); median values ± 25th–75th percentiles. X-axis: pressure expressed in bar, 1 bar = 10^5 P; representation is not proportional to time. (A) during PBS experiments, W-test: *$p<0.05$ vs. control value; (B) in rats injected with AMPT 1 h before pressure exposure, U-test: **$p<0.02$, *$p<0.05$ vs. PBS experiments (dotted line); (C) in rats injected with AMPT 3 h before pressure exposure, U-test: **$p<0.02$, *$p<0.05$ vs. PBS experiments (dotted line). Line with open circles represents the effects of AMPT administration at atmospheric pressure.

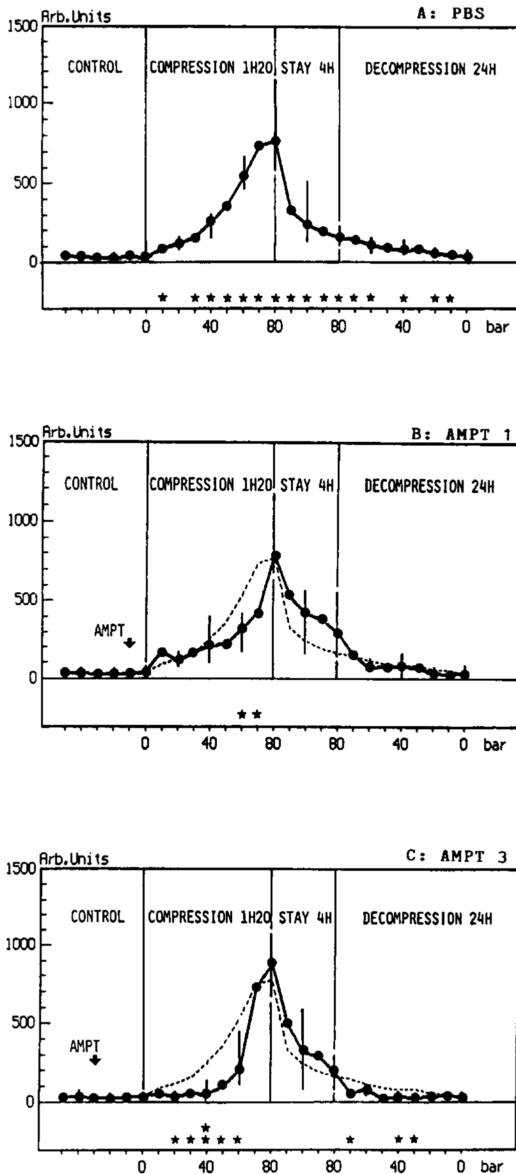

FIG. 3. Development of hyperlocomotor activity in free-moving rats exposed to 80 bar of high pressure. Left: compression up to 80 bar; duration 1 h 20. Middle: stay at 80 bar; duration 4 h. Right: decompression from 80 bar to atmospheric pressure; duration 24 h. Y-axis: hyperlocomotor activity expressed in arbitrary units; median values ± 25th–75th percentiles. X-axis: pressure expressed in bar, 1 bar = 10^5 P; representation is not proportional to time. (A): during PBS experiments, W-test: $*p<0.05$; (B): in rats injected with AMPT 1 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line); (C): in rats injected with AMPT 3 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line).

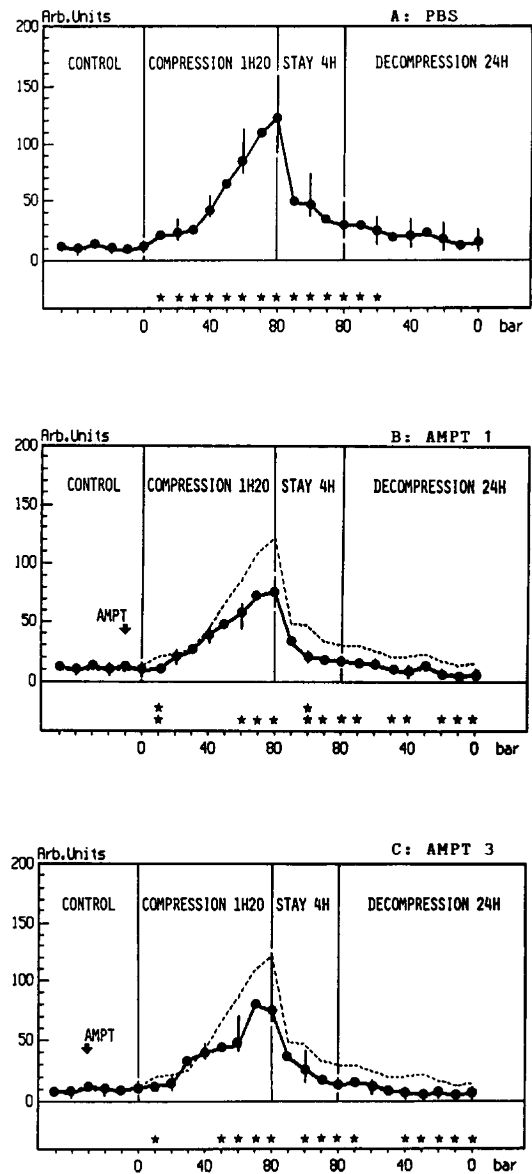

FIG. 4. Development of tremor in free-moving rats exposed to 80 bar of high pressure. Left: compression up to 80 bar; duration 1 h 20 min. Middle: stay at 80 bar; duration 4 h. Right: decompression from 80 bar to atmospheric pressure; duration 24 h. Y-axis: tremor expressed in arbitrary units; median values ± 25th–75th percentiles. X-axis: pressure expressed in bar, 1 bar = 10^5 P; representation is not proportional to time. (A): during PBS experiments, W-test: $*p<0.05$ vs. control value; (B): in rats injected with AMPT 1 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line); (C): in rats injected with AMPT 3 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line).

centiles: −13%, and −25%) of the control value when the pressure had fallen to atmospheric pressure (Fig. 2C).

At a behavioral level, HLA decreased as compared to experiments performed in animals which received PBS or AMPT injections 1 h before pressure exposure (U-test: $p<0.05$) (Fig. 3C). Nevertheless, HLA occurred suddenly at 60–70 bars. As compared to PBS experiments, administration of AMPT 3 h before pressure exposure was found to decrease tremor (U-test: $p<0.05$) (Fig. 4C), and to enhance myoclonia (U-test: n.s.) (Fig. 5C).

DISCUSSION

During PBS experiments, compression was found to increase striatal DA levels in the striatum of the pressurized animals. During the 4-h stay at 80 bars, no change was seen in DA re-

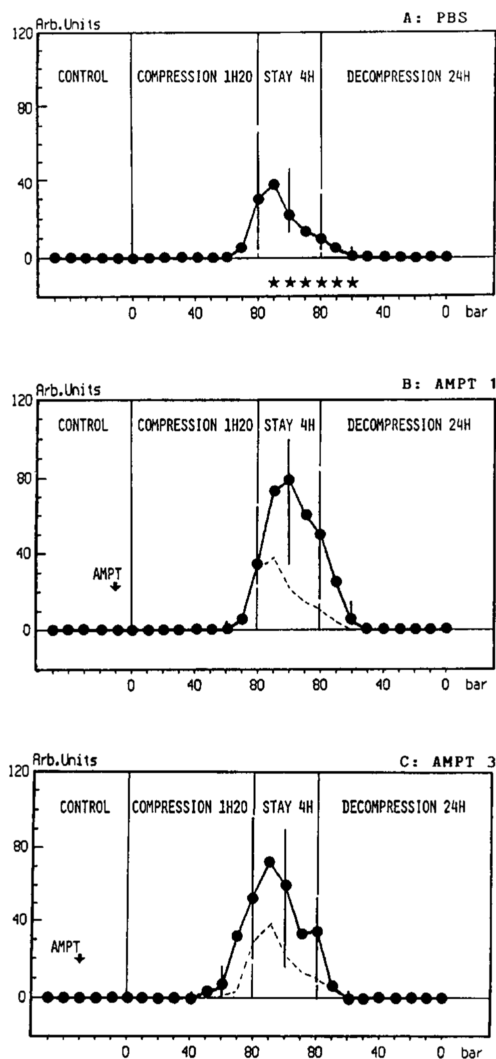

FIG. 5. Development of myoclonia in free-moving rats exposed to 80 bar of high pressure. Left: compression up to 80 bar; duration 1 h 20 min. Middle: stay at 80 bar; duration 4 h. Right: decompression from 80 bar to atmospheric pressure; duration 24 h. Y-axis: tremor expressed in arbitrary units; median values ± 25th–75th percentiles. X-axis: pressure expressed in bar, 1 bar = 10^5 P; representation is not proportional to time. (A): during PBS experiments, W-test: $*p<0.05$ vs. control value; (B): in rats injected with AMPT 1 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line); (C): in rats injected with AMPT 3 h before pressure exposure, U-test: $**p<0.02$, $*p<0.05$ vs. PBS experiments (dotted line).

lease, then during decompression DA levels decreased. Consequently, since previous experiments have shown: (a) in vitro, in a constant DA concentration, that the electrochemical signal was unchanged as pressure increased (12); (b) in vivo, that increasing temperature and oxygen partial pressure had no influence in the release of DA (12), our results confirm that high pressure increased striatal DA release (1, 12, 22, 23). Moreover, these data also confirm that the pressure-induced striatal DA release would be pressure dependent (1). As described previously (1,28), compression led to a sustained increase of HLA, tremor, and myoclonia. All of these motor disturbances progressively decreased during the stay and decompression phases.

Administration of AMPT 1 or 3 h before pressure exposure was found to decrease striatal DA release, during the precompression phase at atmospheric pressure, by respectively −5% and −12% as compared to the DA control value. These data are in agreement with the effects and time-course of ICV injection of AMPT at atmospheric pressure that we recently described in a neurochemical study on the long-term effects of drug administration (2). Administration of AMPT before compression was found to counteract the effect of pressure on DA release. This suggests, in agreement with previous data performed in striatal brain slices (23), that the pressure-induced striatal DA increase could be the consequence of a release in de novo synthesized DA. Nevertheless, if one compares the effects of AMPT under high pressure to the effects of AMPT at normal pressure (data obtained from previous experiments) (2), our results clearly show that the inhibition of DA synthesis by the use of AMPT had no ability to totally block the pressure-induced DA release. This epiphenomenon of the falling baseline could reflect a release in vesicular DA, since bimodal time-courses in striatal DA levels have been reported, during pressure experiments (1) and AMPT experiments performed at normal pressure (16), as being the consequence of successive releases in newly synthesized DA and vesicular DA. During decompression the extracellular striatal DA levels progressively declined, reaching, when the pressure had fallen to 1 bar, similar values than those recorded in AMPT experiments performed at atmospheric pressure (2). These data also support the hypothesis of a release in vesicular DA. Consequently, our results suggest that the pressure-induced striatal DA increase would be not only the consequence of an increased synthesis per se.

At the behavioral level, administration of AMPT 1 or 3 h before pressure exposure was found to delay but not to decrease HLA, then tremor decreased and myoclonia slightly enhanced as compared to PBS experiments. All of these symptoms progressively decreased during the stay and decompression phases.

These results concerning myoclonia are in agreement with previous studies (6,7). They also confirm, as suggested previously (1), that the occurrence of myoclonia is not linked to the pressure-induced striatal DA increase.

Administration of AMPT significantly decreased tremor, despite having demonstrated no correlative data between the occurrence of this symptom and the pressure-induced striatal DA increase (1). This decrease of tremor could be related to the delay in the occurrence of HLA, since hyperbaric tremor essentially consists in intention tremor, i.e., associated with voluntary movement (15).

The delay in the occurrence of HLA was found to be greater in rats which were injected with AMPT 3 h before compression than in others which received AMPT 1 h before pressure exposure. These different delays in the occurrence of HLA could be related to the respective DA levels recorded during pressure exposure in rats injected with AMPT 3 h or 1 h before compression. This result supports, as described previously (1), that the pressure-induced striatal DA increase would be involved in the occurrence of HLA. Nevertheless, during experiments performed with injection of AMPT 3 h before pressure exposure, HLA suddenly occurred at 60–70 bars, despite the fact that DA levels were lower than the DA 100% control value. One plausible explanation for these data could be an activation of DA receptors by vesicular DA release, as suggested above, since the development of HLA corresponded to the higher DA levels recorded during these experiments.

In conclusion, our results confirm that high pressure induces an increase in extracellular striatal DA level. Furthermore, the present experiments also confirm that pressure-induced disturbances in DA metabolism would be involved in the occurrence and the development of HLA. However, at the present time,

further experiments are needed to better determine the mechanisms of the pressure-induced disturbances in the motor and the DAergic striatal functions.

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