EFFECTS OF 2,5-HEXANEDIONE ON RAT SPINAL PYRAMIDAL TRACT

Yuji YAGI*

The present study was undertaken to investigate central axonal neuropathy caused by 2,5-hexanedione (2,5-HD) administration. The effects of 2,5-HD on rat pyramidal tract were studied electrophysiologically and histopathologically. Male rats were given 0.5% 2,5-HD in their drinking water for 4, 6 or 8 wk. Spinal motor conduction velocity was measured by direct electrical stimulation and was examined histopathologically by electron microscopy. Rats treated with 2,5-HD had reduced spinal motor conduction velocity at 6 wk and their pyramidal tract axons had decreased cross-sectional areas at 4 wk. The mean number of neurofilaments in myelinated axons of rats treated with 2,5-HD for 8 wk was 42.1% of that found in control rats, but the number of microtubules was not significantly different. The ratio of neurofilaments to microtubules in rats treated with 2,5-HD for 4 wk was less than 1.0 in 30.9% of myelinated fibers, while in control rats it was greater than 1.0 in all axons. These changes in spinal pyramidal tract were detected relatively early after 2,5-HD administration. These results in rats indicate the need for further studies of 2,5-HD neurotoxicity in the human central nervous system.

Key words: 2,5-hexanedione; central axonal neuropathy; spinal pyramidal tract; spinal motor conduction velocity; neurofilament

INTRODUCTION

N-hexane and methyl *n*-butyl ketone (MnBK) produce metabolites, 2-hexanol and 2,5-hexanedione (2,5-HD). 2,5-HD is considered the most neurotoxic metabolite of *n*-hexane and has been shown by electrophysiology and histopathology to induce central-peripheral distal axonopathy in experimental animals.

Despite the finding that 2,5-HD causes central axonal neuropathy characterized by giant axonal swellings, experimental studies on the effects of 2,5-HD on the central nervous system have not been conducted because of the existence of peripheral neuropathy.

As the long ascending and descending tracts of the spinal cord have been reported by Schaumburg and Spencer^{1,2)} to be particularly vulnerable to 2,5-HD neuropathy, the present study examined the influence of 2,5-HD on rat spinal pyramidal tract. The relationship between conduction disturbances and histopathologic changes in rat pyramidal tracts exposed to 2,5-HD was studied using spinal conduction velocity and quantitative

histopathologic analysis. 2,5-HD was found to have prominent effects on rat pyramidal tract, indicating a need for further studies on the effects of 2,5-HD on the central nervous system in humans.

METHODS

1. Materials. Thirty-six male Sprague-Dawley rats, 8 weeks of age at the beginning of the experiment, were studied. Eighteen rats were given 0.5% 2,5-HD in their drinking water for 4, 6 or 8 wk (6 rats in each group). Eighteen rats were used as controls, given regular tap water over the same time period. Spinal motor conduction velocities were measured by electrical stimulation and the spinal pyramidal tract was examined histopathologically by electron microscopy.

2. Measurement of spinal motor conduction velocity. Within 2 days after completion of 2,5-HD administration, spinal motor conduction velocity was measured. The rats were anesthetized with intraperitoneal injection of sodium pentobarbital (Nembutal, 60 mg/kg body weight). Tracheotomy was performed for artificial respiration, and body temperature was maintained with a heating pad. The animals were placed in a

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stereotaxic holder, and cervical laminectomies (C3 and C4) were carefully performed with a dental drill under a surgical microscope. Two stainless steel needle electrodes were implanted epidurally, one on C3 and the second on C4. Stimulus pulse was given for 0.1 ms, and the stimulus intensity was increased until adequate compound muscle action potentials (MAc) in the plantar muscle were recorded with surface electrodes. F and M waves were recorded by stimulating the posterior tibial nerve at the foot joint. After completion of the physiologic studies, the rats were perfused through the heart with 1.5% glutaraldehyde. The cervical spinal cord was left *in situ* and fixed for 3 to 4 h. The spine was then opened and the distance between the stimulation site in the cervical spinal cord and L5 portion was measured. The spinal conduction time was calculated using the following formula:

Spinal motor conduction time (SMCT) = MAc - [(F - M - 1)/2 + M].

3. *Electron microscopy of the spinal pyramidal tract.* The pyramidal tract at C7 was examined by electron microscopy. The blocks were rinsed in phosphate buffer and post-fixed in 2% OsO₄, dehydrated, and embedded in epoxy resin. Ultra-thin sections were examined under an electron microscope (HU-12A, Hitachi Co., Ltd. Japan). To analyze the axonal cross-sectional area, electron micrographs were taken at a magnification of $\times 5,000$ and printed at a final magnification of $\times 20,000$. The cross-sectional area and myelin sheath thickness of individual myelinated axons were measured using a computer-assisted digitizing system (XL-500, Olympus Co., Ltd. Japan). An average of 500 myelinated fibers was measured in each animal and the percent distribution of axons in the pyramidal tract was determined. To quantify neurofilaments and microtubules, pictures were printed at a final magnification of $\times 60,000$. An average of 50 myelinated fibers was examined in each animal. The area occupied by mitochondria and the smooth endoplasmic reticulum was subtracted from the total area of the axon in both control and experimental rats. Determination of the number of microtubules and neurofilaments was obtained by direct count in a total of 300 control axons and 300 experimental axons. The ratio of neurofilaments to microtubules was calculated in individual myelinated axons.

4. *Statistical analysis.* The data were tested for significance using Student's *t*-test with $p < 0.01$ chosen as the level of significance. All values are given as means $\pm$ SD.

RESULTS

1. Spinal motor conduction velocity

The compound muscle action potential from the plantar flexor muscle was obtained by direct spinal stimulation, and F and M waves were obtained by posterior tibial nerve stimulation in control and 2,5-HD treated rats (see Fig. 1 for examples).

Spinal motor conduction velocity decreased with the length of 2,5-HD administration, as shown in Fig. 2. The mean spinal motor conduction velocity in the control group after 8 wk of tap water administration was 13.9 ± 0.44 m/s, which was not significantly different from the spinal motor conduction velocity in control rats at 4 wk. The spinal motor conduction velocity was significantly lower in rats receiving 2,5-HD for 6 to 8 wk ($p < 0.01$), reaching a minimum of 10.5 ± 0.73 m/s in 2,5-HD treated rats at 8 wk.

2. Histopathology of the spinal pyramidal tract

(1) Cross-sectional area

The cross-sectional area of myelinated pyramidal tract axons was smaller in 2,5-HD treated rats than in control rats, as shown in Fig. 3. All myelinated axons had decreased cross-sectional areas, which became smaller with increase in duration of 2,5-HD administration. The mean cross-sectional area of myelinated axons in rats treated with 2,5-HD for 8 wk ($0.33 \pm 0.03 \mu\text{m}^2$, $n = 6$) was $28.2 \pm 2.2\%$ smaller than in control rats ($0.46 \pm 0.04 \mu\text{m}^2$, $n = 6$). This difference was statistically significant ($p < 0.01$).

(2) Myelin sheath thickness

Histograms of myelin sheath thickness plotted against axon size revealed that the myelin sheath was thinner in small myelinated axons and thicker in large myelinated axons in 2,5-HD treated rats as compared to control rats (Fig. 3). The mean myelin sheath thickness in rats treated with 2,5-HD for 8 wk ($0.158 \pm 0.017 \mu\text{m}$, $n = 6$) was $11.73 \pm 1.1\%$ thicker than in control rats ($0.179 \pm 0.012 \mu\text{m}$, $n = 6$) ($p < 0.01$).

(3) Neurofilament and microtubule concentrations

Visual inspection of the electron micrographs

Fig. 1. Example of compound muscle action potentials obtained by spinal stimulation and example of F and M waves obtained by posterior tibial nerve stimulation. (a) Continual record of compound muscle action potentials obtained by electric extradural stimulation of the cervical spinal cord. (b) Continual record of M and F waves obtained by electric stimulation of the posterior tibial nerve at the foot joint. The latency of compound muscle action potentials obtained by spinal stimulation and the latency of F and M waves obtained by posterior tibial nerve stimulation were prolonged in 2,5-HD treated rats.

suggested that control rat axons had more neurofilaments than axons of comparable size from 2,5-HD treated rats (Fig. 4). To examine this more quantitatively, neurofilaments and microtubules were counted in randomly sampled spinal pyramidal tract axons in 2,5-HD treated and control rats. The mean number of neurofilaments in axons from rats treated with 2,5-HD for 8 wk was 33.4 ± 3.3 ($n=6$), compared to 57.7 ± 4.5 ($n=6$) in axons of similar size in control rats.

This difference was significant ($p < 0.01$) and corresponded to a $42.1 \pm 10.2\%$ decrease in the mean number of neurofilaments in control rats. In contrast, the mean number of microtubules in axons from 2,5-HD treated rats (34.5 ± 2.5 , $n=6$) was not significantly different from that observed in control rats (34.0 ± 1.6 , $n=6$). The histogram of the number of neurofilaments plotted against axon size showed that the number of neurofilaments increased with axon size in both control and 2,5-HD treated rats, although the number of

neurofilaments was decreased in axons of all sizes in 2,5-HD treated as compared to control rats. The number of microtubules also increased with axonal size in both control and 2,5-HD treated rats (Fig. 5).

(4) Ratio of neurofilaments to microtubules

The ratio of neurofilaments to microtubules was calculated in axons of different sizes. The mean ratio of neurofilaments to microtubules in axons from rats treated with 2,5-HD for 8 wk (0.95 ± 0.12 , $n=6$) was $40.9 \pm 6.7\%$ smaller than that of control rats (1.61 ± 0.14 , $n=6$). This difference was statistically significant ($p < 0.01$). Although the ratio of axonal neurofilaments to microtubules in control rats was greater than 1.0 in axons of all different sizes, it was less than 1.0 in $30.9 \pm 9.8\%$ of axons from rats treated with 2,5-HD for 4 wk, and less than 1.0 in $66.7 \pm 12.2\%$ of axons in rats treated with 2,5-HD for 8 wk (Fig. 6).

Fig. 2. Changes in spinal motor conduction velocity according to the duration of 2,5-HD administration. Spinal motor conduction velocity in rats receiving 2,5-HD for 6 or 8 wk was significantly lower than in control rats at 6 or 8 wk ($p < 0.01$).

DISCUSSION

2,5-HD has been shown to induce distal axonal neuropathy by disturbing axonal transport in peripheral nerves of 2,5-HD treated rats. This neuropathy is characterized by giant axonal swellings containing neurofilamentous accumulations and by reduced nerve conduction velocity. In the central nervous system, the long ascending and descending tracts of the spinal cord have been reported to be especially vulnerable to 2,5-HD neuropathy.¹⁻³⁾ However, the central distal axonopathy seen in 2,5-HD treated rats has not been well characterized pathologically or electrophysiologically. The aim of the present study was to perform a quantitative histopathologic analysis of the spinal pyramidal tract in 2,5-HD treated rats and to clarify the association between histopathologic changes in the pyramidal tract and spinal motor conduction velocity. Spinal motor conduction velocity was measured to assess the effects of 2,5-HD neurotoxicity on the pyramidal tract.

Various protocols using electric and magnetic

Fig. 3. Cross-sectional area and myelin sheath thickness of myelinated axons in control and 2,5-HD treated rats. The mean cross-sectional area of myelinated axons in rats treated with 2,5-HD for 8 wk was $28.2 \pm 2.2\%$ smaller than in control rats ($p < 0.01$). The mean myelin sheath thickness of the myelinated axons in the rats treated with 2,5-HD for 8 wk was $11.73 \pm 1.1\%$ smaller than that in control rats ($p < 0.01$).

Fig. 4. Electron micrographs of spinal pyramidal tract in rats. (a) control group, 6 wk ($\times 20,000$). (b) control group, 6 wk ($\times 60,000$). (c) 2,5-HD group, 6 wk ($\times 20,000$). (d) 2,5-HD group, 6 wk ($\times 60,000$). Scale bar: (a), (c) 1 μm , (b), (d) 0.5 μm .

stimulation of the motor system have been used to estimate motor conduction velocity in the pyramidal tract.^{4,5)} Machida et al.⁶⁾ have reported that recording motor action potentials from surface electrodes following epidural stimulation of the spinal cord provides a valid estimation of motor conduction velocity in the spinal descending tract, since amputation of the pyramidal tract and administration of muscle relaxants abolish motor action potentials. Although it is unlikely that action potentials recorded from the surface of the spinal cord or from peripheral nerves after direct or indirect stimulation of the spinal cord are derived solely from the descending tract of the spinal cord, motor action potentials recorded from the musculature are considered to be derived solely from the descending tract of the spinal cord through peripheral nerves. To estimate spinal motor conduction velocity, spinal motor conduction time was calculated from the latency of compound muscle action potential elicited by epidural stimulation of the spinal cord, and the

latency of F and M waves was measured by stimulating the posterior tibial nerve at the foot joint. The F wave study measures the latency between the afferent and efferent arcs of motor fibers from the stimulating site and anterior horn cells, and the M wave study measures the latency between the stimulating site and limb musculature. Spinal conduction time was calculated by subtracting the effects of peripheral nerve axonopathy in order to reveal the conduction time of the pure descending tract in the spinal cord.

Since 2,5-HD neuropathy is a distal axonal neuropathy and motor neurons usually remain intact in 2,5-HD treated rats,⁷⁾ it is not necessary to account for the synaptic delay between the pyramidal tract and motor neurons including the spinal motor conduction time, or the delay of the F wave in the motor neuron. The mean spinal motor conduction velocity in control rats was 14.0 ± 0.35 m/s, proximal to the value expected from the axonal diameter of rat pyramidal tract.⁹⁾ Spinal motor conduction velocity in 2,5-HD

Fig. 5. Histograms of the number of neurofilaments and microtubules plotted in relation to axon size in control and 2,5-HD treated rats. The mean number of neurofilaments in axons from rats treated with 2,5-HD for 8 wk was $32.6 \pm 10.2\%$ less than in control rats ($p < 0.01$).

Fig. 6. The number of myelinated axons with neurofilament to microtubule ratios of less than 1.0 increased in proportion to the duration of 2,5-HD administration. The mean neurofilament to microtubule ratio in axons from rats treated with 2,5-HD for 8 wk was $40.9 \pm 6.7\%$ less than that measured in control rats ($p < 0.01$).

treated rats was significantly reduced after 6 wk of administration. In a previous report,⁹⁾ a reduction in peripheral nerve conduction velocity was seen after 3 wk of administration.

A quantitative histopathologic analysis was also performed on both 2,5-HD treated and control rats. The cross-sectional area of the entire population of myelinated axons in rats treated with 2,5-HD for 4 wk was smaller than in control rats and this decrease was proportional to the duration of 2,5-HD administration.

The mean cross-sectional area of myelinated axons in rats treated with 2,5-HD for 8 wk was $28.2 \pm 2.2\%$ smaller than that observed in control rats. The number of neurofilaments was decreased in myelinated axons of all different sizes in rats treated with 2,5-HD. The mean number of neurofilaments in myelinated axons from rats treated with 2,5-HD for 8 wk was $42.1 \pm 10.2\%$ smaller than that found in control rats.

Monaco et al.^{10,11)} have reported that axonal size is reduced in the optic nerve of 2,5-HD treated rats and have postulated that axonal atrophy in 2,5-HD treated rats reflects a decrease in the number of neurofilaments, resulting from an increase in the rate of axonal transport. This hypothesis is supported by our findings of reduced

size and decreased number of neurofilaments in the pyramidal tract of 2,5-HD treated rats.

Although giant axonal swellings containing neurofilamentous accumulations have previously been reported to be part of central axonal neuropathy in 2,5-HD treated rats,¹⁻³⁾ the present study showed a reduction in axonal size. The number of giant axonal swellings with neurofilamentous accumulations in the pyramidal tract was only 2.0% after 4 wk of 2,5-HD administration and 2.7% after 8 wk, and thus did not change with the duration of 2,5-HD administration. Decreased numbers of neurofilaments reflect increased axonal transport rate, and the histogram of neurofilament number plotted against axonal size correlated better with 2,5-HD neurotoxicity than did the number of giant axon swellings.

While the ratio of neurofilaments to microtubules in control rats ranged from 1.2 to 2.5, with an average of 1.61 ± 0.14 , it ranged from 0.5 to 1.5, with an average of 0.95 ± 0.12 , in rats treated with 2,5-HD for 8 wk. The ratio of neurofilaments to microtubules in rats treated with 2,5-HD for 8 wk was less than 1.0 in $66.7 \pm 12.2\%$ of myelinated fibers in the pyramidal tract. This reduced ratio of neurofilaments to microtubules reflects a decrease in the number of neurofilaments in all sizes of myelinated axons. Because none of the myelinated axons in control rats had a ratio of neurofilaments to microtubules of less than 1.0, such a ratio presumably reflects abnormal myelinated axons. The histogram of the number of neurofilaments after 4 wk of 2,5-HD administration shows a slight decrease in axonal size; 30.9% of the myelinated axons in the pyramidal tract had a neurofilament to microtubule ratio of less than 1.0.

The number of myelinated axons with a neurofilament to microtubule ratio of less than 1.0 was the first sign of 2,5-HD neurotoxicity in the pyramidal tract, and seemed to demonstrate histopathologic changes in a quantitative analysis. Since reduced spinal motor conduction velocity paralleled the decreased axonal size and myelin sheath thickness in 2,5-HD treated rats, it is likely that decreased axonal size and myelin sheath thickness reduce spinal motor conduction velocity. As the change of myelin sheath thickness is relatively small, decreased axonal size is more likely to be responsible for the reduced spinal motor conduction time.

The number of neurofilaments decreased more in large than in small myelinated axons and, as a result, the number of large myelinated axons decreased in 2,5-HD treated rats. A decrease of large myelinated axons may be associated with reduced spinal motor conduction velocity. Myelinated axons with neurofilament to microtubule ratios of less than 1.0 may progressively reduce their axonal size with the duration of 2,5-HD administration, and decreased spinal motor conduction velocity may result from the functional damage that Bouldin and Cavanagh¹²⁾ have termed "chemical transection of axon."

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英文論文の和文要旨

EFFECTS OF 2,5-HEXANEDIONE ON RAT SPINAL PYRAMIDAL TRACT

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2,5-hexanedione のラット脊髄錐体路に対する影響

八木祐吏

本研究は、2,5-hexanedione (2,5-HD) の中枢神経障害である central axonal neuropathy を明らかにするために、2,5-HD の脊髄錐体路に対する影響は、電気生理学的あるいは病理組織学的に検討された。雄性ラットに 0.5% 2,5-HD を飲水内に加えて 4, 6, 8 週間投与した。脊髄刺激により脊髄下行性伝導速度を計測し、電顕にて脊髄錐体路を組織形態学的に検索した。2,5-HD 投与ラットでは脊髄下行性伝導速度は 6 週間投与群で低下し、脊髄錐体路の軸索面積は 4 週間投与群から縮小化した。2,5-HD 投与ラットの脊髄錐体路有髓

神経ではマイクロチュブル数は変化しないが、ニューロフィラメント数は 8 週間投与群において対照群と比較して平均 42.1% 減少していた。対照群ラットの脊髄錐体路有髓神経ではニューロフィラメントとマイクロチュブル数の比は 1.0 以上であるが、2,5-HD 投与ラットでは 1.0 未満の有髓神経が 4 週間投与群において既に 30.9% 出現していた。2,5-HD 投与ラットの脊髄錐体路に対する影響は比較的早期から出現していた。ラットにおけるこの結果は、2,5-HD のヒト中枢神経系に及ぼす神経毒性が検討されることを要求している。

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A NEW DYNAMIC VISUAL ACUITY DEVICE—AGING AND SEX DIFFERENCE IN DYNAMIC VISUAL ACUITY—

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新しい動体視力計の開発—動体視力の加齢影響と性差—

石垣尚男, 宮尾 克

装置：左右に動く対象を識別する能力である Dynamic Visual Acuity (DVA・動体視力) の測定装置を新たに開発し、この装置で 5 才から 92 才までの 826 名の動体視力の加齢影響と性差を調べた。

新たに開発した装置は、90° の白色円形スクリーン上をランドルト環が左から右に水平に動くものである。被検者とスクリーンの距離は 1.2 m である。ランドルト環の切れ目の幅は視角 40' であり、視力値で 0.025 に相当する。ランドルト環は初速 210°/s で動き、4.8°/s で自動的に減速する。被検者は顎台に顎をのせ、眼球運動のみでランドルト環を追跡し方向を識別する。被検者は切れ目の方向（上下左右）が識別できたら、直ちに方向を応答する。正しい応答の場合、検者はストップボタンを押す。識別できたときの速度がデジタルで表示され

る。動体視力のパラメータは、識別できた速度である。測定はこれを 5 回繰り返し、平均値を用いた。

結果：動体視力は男女とも 5 才から 15 才の間に急速に発達していた。とくに、5 才から 10 才の間の発達が顕著であった。この測定では動体視力のピークは男女とも 15 才であった。20 才以降、動体視力は加齢とともにほぼ一定比で低下していた。調査したほとんどの年齢で男性の動体視力が優れていたが、有意差があったのは 5 才児のみであった。生得的に男性は女性より動体視力が優れているのではないかと推測した。この調査により、動体視力は身体の発育期に発達し、成熟後は加齢とともに低下することが明らかとなった。この結果は動体視力 (DVA) の加齢影響は静止視力 (SVA) のそれとは全く異なるものであることを示した。

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