Decreased carotid artery distensibility as a sign of early atherosclerosis in viscose rayon workers

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Carbon disulphide (CS2) is known to accelerate atherosclerosis and to increase the risk for cardiovascular diseases. To assess the effect of CS2 on the functional (distensibility and compliance) and the structural (intima media thickness) properties of the common carotid artery, and blood pressure and lipid metabolism parameters, a cross-sectional study on 85 workers from a viscose rayon factory and 37 controls was carried out. Exposure to CS2 was assessed by personal monitoring and was well below the threshold limit value-time weighted average. Carotid arterial wall properties were determined using a non-invasive ultrasound wall movement detector system. No significant effect of CS2 on blood pressure, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol or triglycerides was found. Among the vascular parameters studied, only distensibility was significantly lower and heart rate was significantly higher in exposed subjects compared with the controls. In conclusion, occupational exposure to CS2 may cause early alterations in arterial elastic properties in young individuals, and even before lipid and clinical findings have occurred, important functional changes in the vessel wall are present.

Key words: Arterial compliance; arterial distensibility; blood pressure; carbon disulphide; common carotid artery; heart rate; lipid metabolism; occupational exposure.

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Introduction

Carbon disulphide (CS2) is an organic solvent widely used in the viscose, rubber and chemical industries. Epidemiological studies carried out in the UK [1], Finland [2], the USA [3] and other countries [4–6] indicated that occupational long-term exposure to high concentrations of CS2 may increase the risk for cardiovascular diseases (CVD) and, in particular, for coronary heart disease (CHD) mortality. A significant excess of death from cerebrovascular diseases has also been reported [5,7]. Various morbidity studies have indicated that CS2 increases the prevalence of arterial hypertension, CHD and lipid metabolism disturbances [8–10]. Several studies, however, did not reveal a significant increased risk for CVD, in particular in workers exposed to low levels of CS2 [11–13]. The mechanisms by which CS2 may contribute to cardiovascular disease are not well defined. The most discussed hypothesis is that CS2 may cause disturbances of lipid metabolism and accelerate atherosclerosis. Some mechanisms by which CS2 may play a role in cardiovascular disease are: (i) an increase in the levels of blood cholesterol and low-density lipoprotein (LDL) cholesterol, and/or an induction of lipid accumulation in vessel walls; (ii) induction of a lipid peroxidation process; and (iii) promotion of a mutation in
the arterial wall. Other possible mechanisms are an increase in blood pressure, impaired neurovegetative regulation, disturbances in thyroid function (hypothyroidism) and catecholamine metabolism, depressed fibrinolytic activity, and a direct toxic effect on the heart and vessels. Experimental results revealed changes in lipid metabolism, and a number of biochemical and structural disorders in the heart muscle and vessels [14–16]. In accordance with the homocystinuria theory of arteriosclerosis, an induced vitamin B6 deficiency state, which predictably led to a homocystinuria-like state, has been reported in workers exposed to CS2 [17].

The threshold limit value-time weighted average (TLV-TWA) proposed by the American Conference of Governmental Industrial Hygienists (ACGIH) for CS2 is 31 mg/m³ [18]. The higher levels that existed many years ago are no longer found in the workplace in most industrialized countries, but they are still an important socio-economic and health problem in the older industries in the developing and East European countries. Currently, the basic questions to be addressed concern the effect of CS2 exposure below the TLV-TWA and the recommendation of a technique that might detect the early-stage changes in the cardiovascular system. The clinical results concerning the effects of exposure to low concentrations of CS2 on the cardiovascular system are controversial. The effects of CS2 on haemodynamic arterial properties are not well established. Some studies from the 1960s and 1970s reported CS2-induced alterations of rheographically determined cerebral circulation [19,20]. There are a few studies published recently of the vascular effects of CS2 evaluated by ultrasound measurements of the stiffness of the carotid artery and the aortic wall, and the results are not convincing [13,21,22]. The non-invasive, high-resolution pulsed Doppler echocardiography technique may be very useful because it can detect functional and structural arterial abnormalities at an early stage.

The objective of this study was to evaluate the effects of occupational exposure to CS2 concentrations below the TLV-TWA on structural (lumen diameter and arterial wall thickness) and functional (distensibility and compliance) properties of common carotid artery, and blood pressure and lipid metabolism parameters. Early changes in vessel wall morphology and function have been studied by examining the intima media thickness of the vessel wall, and arterial compliance and distensibility.

**Subjects and methods**

**Study population**

The study was performed in 85 male workers aged between 20 and 54 years in a Belgian viscose rayon factory. The controls were 37 men aged between 23 and 63 years, working in a metal, a plastic, a textile and a starch processing factory without occupational exposure to chemical factors. Participation was voluntary, and the response rate was 58% in exposed and 48% in non-exposed subjects. All subjects included in this study gave written informed consent for their participation and the protocol of the study was approved by the Ethics Committee of the Faculty of Medicine of Ghent University.

**Assessment of exposure**

Six job titles from three departments (viscose preparation, spinning and bleaching departments) of the viscose rayon factory with CS2 exposure were identified. The only chemical in the working environment that was considered to be important for the investigated vascular and lipid parameters in our study was CS2. Exposure to CS2 was assessed by personal monitoring. Measurements were made using pumps (Dupont P125A, Gilair-3 and -5, Ametek MG4) and charcoal tubes (SKC no. 226-01, 400 mg/200 mg) fixed onto the face of the employees inside the respirators, serially connected to a drier tube (SKC, 270 mg Na2SO4). Charcoal tubes were fixed onto the shoulder or collar of the employees in order to measure the CS2 concentrations outside the respirators. The CS2 concentrations were analysed using NIOSH method 1600.

The CS2 exposure was considered in two ways: (i) binary (exposed versus non-exposed) and (ii) a cumulative exposure index (CS2 index), calculated for each worker by multiplying the number of years he had held a particular job in the viscose factory by the CS2 exposure (in mg/m³) in that job.

**Medical examinations**

Self-administered questionnaires, checked for completeness by a physician on the day of the examination, were used to determine personal data (age, smoking habits, alcohol consumption, level of education, job stress, interpersonal problems at work, rotating shift work, ethnic origin, medications, etc.), and medical and job history (previous jobs, duration of employment, use of personal protective equipment, etc.) [23]. With regard to ethnic origin, the study population was divided into two groups: Belgian descendants and non-Belgian (Turkish and North Africans) descendants. Height and body weight were measured, and a body mass index (BMI; kg/m²) was calculated.

Measurements of the common carotid artery were performed with a non-invasive ultrasound wall movement detector system [24]. It consisted of a conventional ultrasound imager (Sonotron/Diasonics, model PRISMA, Les Ulis, France) with a 7.5 MHz transducer, a data acquisition system and a personal computer. The transducer was positioned on the surface of the skin directly...
above the artery being studied. The arterial diastolic diameter (with a precision of ~10 mm) and the arterial wall distension (with a precision of <10 mm) were recorded for 5 s. Simultaneously, blood pressure (BP) was measured with an automated device to calculate pulse pressure (systolic minus diastolic BP). From the arterial diameter, the change in diameter during the heart cycle (distension) and the pulse pressure, arterial distensibility and compliance were calculated according to the equations:

\[ \text{Distensibility coefficient (DC)} = \left( \frac{2\Delta D/D_d}{\Delta P} \right) \]

\[ \text{Compliance coefficient (CC)} = \left( \frac{\pi D_d \times \Delta D}{2\Delta P} \right) \]

where \( \Delta D \) is the arterial wall distension, \( D_d \) is the arterial diastolic diameter and \( \Delta P \) is the pulse pressure; thus, the compliance coefficient could be calculated by multiplying the distensibility coefficient by the cross-sectional area of the vessel lumen. The same experienced investigator with no knowledge of exposure status performed all measurements. Intra-observer intra-session variability of vascular wall properties, expressed as variation coefficients, was calculated from 10 consecutive measurements of regional relative diameter changes at the common carotid artery. Intra-observer intra-session variabilities were 8 ± 2% (\( \Delta D/D_d \)), 5 ± 2% (DC) and 9 ± 3% (CC).

Systolic (SBP) and diastolic (DBP) blood pressures (Korotkoff phases I and V, respectively) were measured in the supine position after 5 min rest with a mercury manometer, and their values were used for BP analysis.

Venous blood was collected from sitting, fasting subjects and analysed for total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG). TC and TG were measured with an enzymatic assay [25,26]. HDL-C was assayed automatically without sample pre-treatment [27]. The coefficients of variation ranged between 1.3 and 6.7%. LDL cholesterol (LDL-C) was calculated using the formula of Friedewald et al. [28].

The level of significance for statistical testing was set at \( \alpha = 0.05 \). All calculations were carried out using SPSS statistical software.

Results

The results on the exposure values for the different job titles (given as the geometric means of the individual sample data per job title) showed that the CS\(_2\) concentrations outside the respirators ranged from 3.9 to 32.4 mg/m\(^3\) in the spinning department and from 1.3 to 20.8 mg/m\(^3\) in the viscose preparation department. Although the exposure data showed some variability, the personal exposure was below the TLV-TWA. The geometric means of CS\(_2\) exposure inside the respirators ranged from 2.2 to 13.0 mg/m\(^3\) and from 6.00 to 10.09 mg/m\(^3\) in the spinning and viscose preparation departments, respectively. The CS\(_2\) exposure in the bleaching department was very low (up to 3.2 mg/m\(^3\)). There were no significant differences between exposed and non-exposed subjects concerning their age, BMI, alcohol consumption (g/day), smoking habits (pack-years), educational level, rotating shift work, interpersonal tension, stress at work and ethnic descent (Table 1).

Exposed workers were younger, and were less likely to drink alcoholic beverages or to work in shifts than non-exposed workers. The proportion of Turkish and North African descendants was higher in the exposed workers. The average BMI and proportions of subjects with considerable interpersonal tensions, low educational level and stress at work were similar in both groups.

The comparison of the average values of the investigated vascular parameters between exposed and non-exposed workers is shown in Table 2. The exposed workers had a significantly lower DC than the controls. The difference was significant after adjustment for age, smoking (pack-years), alcohol consumption (g/day), BMI, ethnic descent, heart rate (HR) and BP.

### Table 1. Characteristics of the population exposed to CS\(_2\) and those who were not exposed

<table>
<thead>
<tr>
<th></th>
<th>Exposed (n = 85)</th>
<th>Non-exposed (n = 37)</th>
<th>( P^* )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [mean (SD), years]</td>
<td>37.1 (8.0)</td>
<td>40.5 (8.73)</td>
<td>0.13</td>
</tr>
<tr>
<td>BMI [mean (SD), kg/m(^2)]</td>
<td>24.9 (3.4)</td>
<td>25.3 (3.2)</td>
<td>0.77</td>
</tr>
<tr>
<td>Alcohol [mean (SD), g/day]</td>
<td>7.7 (12.3)</td>
<td>11.4 (14.8)</td>
<td>0.15</td>
</tr>
<tr>
<td>Pack-years [mean (SD)]</td>
<td>9.1 (10.3)</td>
<td>8.0 (11.9)</td>
<td>0.20</td>
</tr>
<tr>
<td>Low educational level (%)</td>
<td>26.2</td>
<td>27.0</td>
<td>0.99</td>
</tr>
<tr>
<td>Rotating shift work (%)</td>
<td>75.0</td>
<td>87.9</td>
<td>0.14</td>
</tr>
<tr>
<td>Stress at work (%)</td>
<td>33.3</td>
<td>33.3</td>
<td>0.99</td>
</tr>
<tr>
<td>Interpersonal tension (%)</td>
<td>20.2</td>
<td>21.2</td>
<td>0.99</td>
</tr>
<tr>
<td>Turkish and North African descent (%)</td>
<td>39.3</td>
<td>35.1</td>
<td>0.69</td>
</tr>
</tbody>
</table>

*According to the Mann–Whitney test for comparing distributions of continuous variables and Fisher’s exact test for comparing proportions.
ethnic descent, BMI, HR and SBP. There were no significant differences between exposed and non-exposed workers with regard to cross-sectional compliance coefficient and intima media thickness (IMT). The HR in exposed workers was significantly higher when compared with the controls (Table 3). No significant differences were found concerning TC, HDL-C, LDL-C, TG, SBP and DBP.

Table 2. Average values (SD) of vascular parameters of the common carotid artery in relation to CS₂ exposure

<table>
<thead>
<tr>
<th></th>
<th>Exposed (n = 85)</th>
<th>Non-exposed (n = 37)</th>
<th>Significanceᵃ</th>
<th>Significanceᵇ</th>
<th>Significanceᶜ</th>
</tr>
</thead>
<tbody>
<tr>
<td>CC [mean (SD)]</td>
<td>0.92 (0.22)</td>
<td>0.91 (0.26)</td>
<td>0.94</td>
<td>0.34</td>
<td>0.39</td>
</tr>
<tr>
<td>DCᵈ [mean (SD)]</td>
<td>18.52 (4.95)</td>
<td>21.61 (10.67)</td>
<td>0.01</td>
<td>0.03</td>
<td>0.02</td>
</tr>
<tr>
<td>IMT [mean (SD)]</td>
<td>0.53 (0.13)</td>
<td>0.55 (0.15)</td>
<td>0.94</td>
<td>0.88</td>
<td>0.86</td>
</tr>
</tbody>
</table>

For abbreviations, see the text.
ᵃAdjusted for age.
ᵇAdjusted for age, pack-years, alcohol (g/day), ethnic descent, BMI and heart rate.
ᶜAdjusted for age, pack-years, alcohol (g/day), ethnic descent, BMI, heart rate and systolic blood pressure.
dNatural logarithmic transformation used for statistical analysis.

Table 3. Average values of blood pressure (BP), heart rate (HR) and lipid parameters in relation to CS₂ exposure

<table>
<thead>
<tr>
<th></th>
<th>Exposed (n = 85)</th>
<th>Non-exposed (n = 37)</th>
<th>Significanceᵃ</th>
<th>Significanceᵇ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP [mean (SD)]</td>
<td>127.01 (13.48)</td>
<td>127.22 (14.81)</td>
<td>0.85</td>
<td>0.97</td>
</tr>
<tr>
<td>Diastolic BP [mean (SD)]</td>
<td>84.17 (10.02)</td>
<td>82.13 (9.58)</td>
<td>0.53</td>
<td>0.23</td>
</tr>
<tr>
<td>HR [mean (SD)]</td>
<td>65.48 (10.23)</td>
<td>60.87 (10.16)</td>
<td>0.04</td>
<td>0.04</td>
</tr>
<tr>
<td>Cholesterol [mean(SD)]</td>
<td>203.16 (47.82)</td>
<td>214.89 (39.70)</td>
<td>0.09</td>
<td>0.21</td>
</tr>
<tr>
<td>HDL-C [mean (SD)]</td>
<td>58.25 (11.54)</td>
<td>61.77 (10.66)</td>
<td>0.25</td>
<td>0.27</td>
</tr>
<tr>
<td>LDL-C [mean (SD)]</td>
<td>117.89 (43.44)</td>
<td>129.10 (37.88)</td>
<td>0.10</td>
<td>0.14</td>
</tr>
<tr>
<td>Triglycerides [mean (SD)]</td>
<td>138.18 (100.23)</td>
<td>125.19 (70.29)</td>
<td>0.68</td>
<td>0.53</td>
</tr>
</tbody>
</table>

For abbreviations, see the text.
aSignificance of difference with reference group according to the Mann–Whitney test.
bAdjusted for age, pack-years, alcohol (g/day) and ethnic descent.
cNatural logarithmic transformation used for statistical analysis.

dTable 4. Multiple linear regression analysis (regression coefficients and significance)

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Exposure</th>
<th>Age</th>
<th>BMI</th>
<th>Pack-years</th>
<th>Alcohol</th>
<th>Descent</th>
<th>HR</th>
<th>SBP</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>81.348</td>
<td>−2.215</td>
<td>0.102</td>
<td>0.654</td>
<td>−0.029</td>
<td>0.114</td>
<td>−2.966</td>
<td>0.462***</td>
<td>0.230</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>42.635</td>
<td>0.109</td>
<td>0.147</td>
<td>0.393</td>
<td>0.014</td>
<td>0.151</td>
<td>0.852</td>
<td>0.039</td>
<td>0.231</td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>14.054</td>
<td>4.367</td>
<td>0.106</td>
<td>0.041</td>
<td>−0.089</td>
<td>2.366</td>
<td>0.295***</td>
<td>0.242</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC</td>
<td>150.758</td>
<td>−9.977</td>
<td>1.424</td>
<td>1.474</td>
<td>0.433</td>
<td>−0.616</td>
<td>−20.204</td>
<td>0.190</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-C</td>
<td>74.431</td>
<td>−2.067</td>
<td>0.172</td>
<td>−0.567</td>
<td>−0.197</td>
<td>0.142</td>
<td>−3.906</td>
<td>0.147</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-C</td>
<td>100.232</td>
<td>−11.956</td>
<td>0.910</td>
<td>0.968</td>
<td>0.481</td>
<td>−0.737</td>
<td>−20.721*</td>
<td>0.162</td>
<td></td>
<td></td>
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<tr>
<td>TGᵃ</td>
<td>2.850</td>
<td>0.081</td>
<td>0.016</td>
<td>0.038</td>
<td>0.007</td>
<td>3.8E−04</td>
<td>0.163</td>
<td>0.165</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td>1.903</td>
<td>0.039</td>
<td>−0.004</td>
<td>−0.002</td>
<td>−0.001</td>
<td>4.3E−04</td>
<td>−0.035</td>
<td>−0.009***</td>
<td>−0.020</td>
<td></td>
</tr>
<tr>
<td>DCᵃ</td>
<td>5.009</td>
<td>−0.130</td>
<td>−0.013***</td>
<td>−0.027***</td>
<td>−0.001</td>
<td>6.5E−04</td>
<td>0.120</td>
<td>−0.007</td>
<td>−0.005</td>
<td>0.465</td>
</tr>
<tr>
<td>IMTᵃ</td>
<td>−1.138</td>
<td>0.008</td>
<td>0.009</td>
<td>0.007</td>
<td>0.001</td>
<td>0.002</td>
<td>−0.034</td>
<td>−0.002</td>
<td>6.1E−04</td>
<td>0.210</td>
</tr>
</tbody>
</table>

For abbreviations, see the text.
ᵃP < 0.05;  ᵇP < 0.01;  ᶜP < 0.001.
ᵃNatural logarithmic transformation used for statistical analysis.

DC and SBP and HR and DC. The multiple regression analysis has been used also in order to assess the effect of the overall CS\textsubscript{2} exposure of the total employment time of the workers. No significant associations between the CS\textsubscript{2} index and investigated vascular and lipid parameters were found.

**Discussion**

Arterial compliance and distensibility, measured by non-invasive and specific techniques, are well accepted study parameters and give information on the early vascular alterations occurring in pathophysiological conditions. Among the studied vascular parameters, only distensibility of the common carotid artery was significantly lower in exposed workers, compared with the controls. To our knowledge, our study is the first to investigate the effect of occupational exposure to CS\textsubscript{2} on the arterial distensibility of the common carotid artery. Another study concerning carotid duplex sonography in a very limited group of 10 people who had polyneuropathy after long-term high exposure to CS\textsubscript{2} revealed mild atherosclerosis with plaques (<20% stenosis) of extracranial vessels in six of them [21]. No significant effects of CS\textsubscript{2} on stiffness and blood flow of the carotid artery in Japanese rayon workers were found. The pulse wave velocity of the aorta, adjusted for BP, was significantly lower in exposed subjects compared with controls [22]. Distensibility reflects the elasticity of the artery and is a determinant of the vessel wall, while compliance reflects the buffering capacity of large arteries and is an important determinant of the afterload of the heart. Hypertension, age, atherosclerosis, hypercholesterolaemia and higher HR are known to decrease arterial distensibility and systematic compliance [29–35]. In our study, the effect of exposure on the distensibility coefficient was significant after adjustment for age, BMI, smoking, alcohol consumption, ethnic descent, HR and SBP. In linear regression analysis, the distensibility coefficient was negatively associated with exposure, age, BMI, HR and SBP. No effect of CS\textsubscript{2} exposure on arterial compliance and intima media thickness was found. While arterial distensibility is associated with early atherosclerosis, arterial compliance is altered significantly only in extensive atherosclerosis [32]. Before the evidence of morphological lesions or plaques, endothelial dysfunction, such as impairment in endothelium-dependent vascular tone regulation, may occur. The results of our study show that occupational exposure to CS\textsubscript{2} may cause alterations in arterial elastic properties in young individuals, and even before anatomic lesions have occurred, important functional changes in the vessel wall can be present. This may indicate that CS\textsubscript{2} can act directly on the arterial smooth muscle cells, leading to decreased distensibility. It may be hypothesized that CS\textsubscript{2} may induce endothelial dysfunction, leading to increased vascular tone and, as a consequence, to decreased arterial distensibility. Substantial changes in metabolic and energetic processes, in the normal state of fibrillar proteins and in the components of the intracellular substance of connective tissue of the vessel wall in rats exposed to CS\textsubscript{2}, have been reported [14].

The results of our study are in accordance with those reported by other authors concerning increased HR in workers exposed to CS\textsubscript{2}. A significantly increased HR in exposed workers, compared with the reference group, has also been reported by Kuo et al. [10] and Bortkiewicz et al. [36]. It may be speculated that the main factor in the higher HR among exposed workers was due to CS\textsubscript{2} stimulation of the sympathetic nervous system. It is an unfavourable finding because, as demonstrated by the Framingham study [37], a high HR increases risk of sudden death 1.3-fold and is recognized as an independent risk factor for CHD.

There are several limitations that should be considered in this investigation. First, in the cross-sectional design, negative selection bias may have been introduced because of the so-called ‘healthy worker’ effect. If a considerable number of subjects had indeed left the factory, this could lead to an underestimation of disease. We contacted the people who had left the plant during the last 10 years: 31\% of the viscose workers versus 13\% of the controls mentioned health complaints as a reason for leaving the industry, but vascular and heart complaints were rarely mentioned. Secondly, the incomplete response is another possible source of selection bias. Since diseased persons may be more motivated to participate, this might lead to an overestimation of disease occurrence. However, it is doubtful that these two kinds of bias would be different for exposed and non-exposed workers. Thirdly, it is well known that the investigated vascular and lipid parameters can be influenced by many major cardiovascular (smoking, age, obesity) and additional risk factors (stress at work, alcohol consumption, educational level, shift work, interpersonal tension and ethnic descent). No significant differences have been found between exposed workers and controls in relation to these possible confounders. Age, smoking, obesity, alcohol consumption and ethnic descent were considered as important potential confounders and/or modifiers of the outcomes, and were taken into consideration in the data analysis. Increased BP and higher HR are known cardiovascular risk factors, but they may represent mechanisms through which CS\textsubscript{2} may play a role in cardiovascular disease. On the other hand, it is known that the increase in HR and BP are accompanied by a reduction in arterial compliance and distensibility. To avoid confounding by these factors, all the analyses of the investigated vascular parameters
Conclusions

The results of the present study indicate that occupational exposure to CS₂ at concentrations below the TLV-TWA may increase the HR and cause early functional changes in the vessel wall (decrease in arterial distensibility). The effects could be explained through hypothetical mechanisms involving an impaired neuro-vegetative regulation of cardiovascular function and a direct toxic effect of CS₂ on the vessel wall, leading to a decrease in arterial elasticity. Up to the current TLV-TWA level of 31 mg/m³, we did not find a significant difference in BP and lipid parameters between exposed workers and controls. Measurement of arterial distensibility may be recommended as a method for the detection of early vascular alterations in workers exposed to CS₂.

Acknowledgements

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