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Research Article

Terlipressin versus norepinephrine to counteract intraoperative paracentesis induced refractory hypotension in cirrhotic patients

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KEYWORDS

Terlipressin;
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Abstract *Back ground:* Some of tense ascitic patients with end stage liver disease and portal hypertension were presented to our emergency department with surgical acute abdomen that required urgent abdominal surgery which might be associated with inevitable rapid and relatively complete evacuation of this ascitic fluid with possible occurrence of post-paracentesis-induced hypotension. The aims of this study were to compare between the intraoperative use of terlipressin versus norepinephrine for the management of paracentesis induced refractory hypotension not responding to colloid resuscitation or ephedrine in patients with end-stage liver disease during emergency abdominal surgery.

Patients and method: Thirty-four patients experienced refractory hypotension during or shortly after the paracentesis process were randomized to receive either bolus dose of terlipressin (1 mg over 30 min) followed immediately by a continuous infusion of 2 μg/kg/h (T group, $n = 17$) or norepinephrine infusion at starting dose of 0.1 μg/kg/min (N group, $n = 17$).

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Measurements: Hemodynamic parameters, cardiac output, systemic vascular resistance, blood gases, lactic acid, liver and kidney functions.
Results: All patients of both groups showed significant decreases in MAP during or immediately after the paracentesis process to reach mean values of 57 ± 1.4 and 58 ± 1.8 mmHg in terlipressin or norepinephrine groups respectively. This was associated with drop in the SVR that reached mean values of 445 ± 28 and 425 ± 20 dynes/sec/cm^5 in both terlipressin and norepinephrine groups respectively. At the 2nd day post operative there was significant increase in serum creatinine values in the norepinephrine group.
Conclusion: Terlipressin and norepinephrine successfully counteracted the post-paracentesis refractory hypotension and the drop of the systemic vascular resistance. It also showed the renal protective effects of terlipressin in the immediate postoperative period.

1. Introduction

Ascites is considered to be one of the major complications in patients with end stage liver disease and portal hypertension [1]. Occasionally, some of those patients were presented to our emergency department with surgical acute abdomen that required urgent abdominal surgery which might be associated with inevitable rapid and relatively complete evacuation of this ascitic fluid with possible occurrence of post-paracentesis-induced hypotension [2–4]. Some reports revealed that administration of albumin is very beneficial in the scenarios of therapeutic paracentesis [5–7]. Other studies have shown that a marked decrease in systemic vascular resistance (SVR) appears very early after the mobilization of ascites [8–10], especially in patients who develop paracentesis induced circulatory dysfunction (PICD) and hypotension. Since the plasma volume remains unchanged after paracentesis [11,12], an accentuation of arterial vasodilatation, which is already present in these patients, has been suggested to explain the rapid decrease in SVR after paracentesis [10,13]. However, none of these reports were done intraoperatively during emergency abdominal surgery.

Terlipressin acts via the vasopressin system, which may be considered an important physiologic system for the regulation of arterial blood pressure. This long-acting synthetic analog of vasopressin has a half-life of 6 h and a higher vascular selectivity for vascular receptors compared with vasopressin [14], and Significant vasoconstriction was clearly demonstrated in experimental setting [15]. In addition to its effects on microvascular blood flow, terlipressin might reduce cardiac output via reflex mechanisms mediated through glossopharyngeus baroreceptors reflex (aortic/carotid sinus baroreceptors) [16].

1.1. Aim of the study

The aims of this study were to compare between the intraoperative use of terlipressin versus norepinephrine for the management of paracentesis induced refractory hypotension not responding to colloid resuscitation or ephedrine in patients with end-stage liver disease during emergency abdominal surgery regarding the intraoperative hemodynamic parameters, metabolic parameters fluid requirements, post operative liver and renal functions.

2. Patient and method

2.1. Patients

This study was carried out in the National Liver Institute – Menofeya University after approval from the local ethical committee and informed consents from patients. Among 93 adult tense ascitic patients with end stage liver disease (Child B–C) scheduled for emergency abdominal surgery were prospectively included in the study between March 2008 and August 2011. Of them 34 patients experienced refractory hypotension during or shortly after the paracentesis process after being stable after induction of anesthesia, hence they were randomized through closed envelops and analyzed in the study. Paracentesis-induced hypotension was defined as mean arterial pressure (MAP) <60 mmHg or 30% drop of MAP associated with the reduction of 10% or greater in SVR than pre-paracentesis values not responding to albumin and colloid replacement therapy [17,18]. The pre-paracentesis value was determined by the mean of three sets of measurements with 10 min intervals obtained 30 min before peritoneal opening and drainage or spillage of the ascitic fluid and only patients who experienced hypotension in the presence of optimized volume status were analyzed. Patients were excluded if they had one of the following conditions: cardiac failure, sepsis, preoperative use of plasma expanders or paracentesis within 1 week, preoperative ruptured or leaking hernia, evacuated ascitic fluid volume less than 5 L, preoperative beta blockers and if there was primary renal dysfunction. The clinical characteristics of the study groups are summarized in (Table 1).

2.2. Anesthetic technique

After preoxygenation, general anesthesia was induced with propofol 2 mg/kg IV, fentanyl 1 μg/kg IV and IV followed by endotracheal intubation which was facilitated by rocuronium 1.2 mg/kg. All patients were mechanically ventilated to maintain an end-tidal carbon dioxide 35 ± 5 mmHg. Anesthesia was maintained cisatracurium–isoflurane keeping entropy reading (GE healthcare – Helsinki, Finland) between 40 and

Table 1 Demographic and data and patient characteristics.

	T group (n = 17)	N group (n = 17)
Age (years)	57.3 ± 4.1	61.5 ± 4.6
Gender (M/F)	13/4	12/5
Height (cm)	165.3 ± 7.2	167.2 ± 5.8
Weight (kg)	81.4 ± 5.7	82.5 ± 4.8
Child score: B/C	10/7	11 ± 6
Liver disease		
HCV	15	15
HBV	1	1
HCV + HBV	0	1
Alcoholic	1	0
Ascites removed (L)	7.7 ± 2.6	8.4 ± 1.9

T: terlipressin group; N: norepinephrine group; Data are presented as mean ± SD or number of patients. There are no significant differences between the two groups (p-value > 0.05).

60. When State entropy increases above 60 the isoflurane was increased. In contrast, if State entropy was in the recommended range, but response entropy increases 5–10 units above it, this was interpreted as a sign of uncovered nociception, and more analgesic medication is required [18]. An arterial line was placed in the left radial artery and central lines were inserted in the right internal jugular vein with triple-lumen catheter. Normothermia was maintained using fluid warmer in addition to forced warming air blanket (Bair Hugger®; Arizant, United Kingdom). During the postoperative period of the study, patients received oxygen via Venturi mask to maintain PaO_2 at greater than 100 mmHg.

2.3. Perioperative fluid optimization

In all patients 500 mL of Ringer's acetate was infused during induction followed by a 2 mL/kg/hr continuous infusion. At the stage of paracentesis simultaneous intravenous albumin was administered (8 g/l of ascitic fluid removed) using 20% human albumin solution [19]. Transesophageal doppler (TED) was inserted after induction and to ensure euvolemic state, for patients with corrected flow time (FTc) of less than 350 ms which suggests hypovolaemia. The following protocol was applied afterward [20]:

- If Stroke volume (SV) remains the same or increased and FTc < 350 ms – repeat the fluid challenge.
- If SV increased by 10% and FTc > 350 ms – repeat the fluid challenge till no increase in SV.
- If FTc > 400 ms then no further fluids are required till FTc or SV decrease by 10%.

2.4. Protocol of the study

Patients included in study protocol were experienced refractory hypotension during or shortly after the process of acetic fluid spillage or paracentesis although optimized volume status and also not or temporarily responding to ephedrine increments. They were randomized to receive either bolus dose of terlipressin (1 mg over 30 min) followed immediately by a continuous infusion of 2 μg/kg/h (T group, n = 17) or norepinephrine infusion at a starting dose of 0.1 μg/kg/min (N group, n = 17). Both infusions were progressively titrated to increase MAP to the pre paracentesis values. The dose of terlipressin was selected taking into account previous studies on terlipressin [21]. In case of failure of both treatment and if MAP was not restored (*i.e.*, MAP was < 60 mmHg or > 30% reduction of pre paracentesis value) for 10 min, patients received an intravenous bolus dose of 0.2 mg of epinephrine that can be repeated.

2.5. Measurements

1. Hemodynamic parameters including: Heart rate, electrocardiography (for any type of dysrhythmias), invasive arterial blood pressure (radial artery catheter), central venous pressure (CVP) and urine output.
2. TED data which include: stroke volume (SV), cardiac output (COP), and systemic vascular resistance (SVR).
3. Metabolic parameters: Arterial blood gases and lactic acid level in blood.
4. Liver and renal function parameters: Total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), international normalized ratio (INR) and serum creatinine. Samples taken preoperatively and 2nd postoperative day.

2.6. Statistical analysis

Data are shown as mean and standard deviation or frequency. Statistical analysis was performed using the unpaired Student's t-test, the Mann–Whitney U test and chi square test with Fisher correction. Data were statistically analyzed using SPSS (statistical package for social science) program version 17 for windows. A p-value < 0.05 was considered to be significant.

3. Results

During the inclusion period, 93 adult tense ascitic patients with end stage liver disease (Child B-C) scheduled for emergency abdominal surgery. Fifty nine patients were not included in the study protocol for the following reasons: 21 patients did not develop significant hemodynamic changes during or after the paracentesis process, 12 patients developed temporary hypotension that responded to ephedrine increments, 15 patients were receiving beta blocker to control portal hypertension blockers, six had organic renal disease, and five did not give consent to participate. Therefore, 34 patients whom developed refractory hypotension that did not respond to colloid replacements or ephedrine increments were included in the present study; 17 patients received terlipressin (T group), and the other 17 patients received norepinephrine (N group). (Table 1) shows characteristics of the enclosed patients in this study. No significant differences were detected between both groups regarding clinical data, volume of ascites removed, and mean arterial pressure at pre-paracentesis period. There were no complications associated with either the administration of terlipressin, norepinephrine or the use of the TED. During recovery and postoperative period, no patient experi-

Table 2 Hemodynamic data.

	Before paracentesis	After paracentesis	End of surgery
HR (bpm)			
T	85 ± 6.8	88 ± 5.7	88 ± 5.8
N	87 ± 4.9	90 ± 3.5	88 ± 4.4
MAP (mmHg)			
T	90 ± 6.9	57 ± 1.4[#]	94 ± 6.3
N	95 ± 5.8	58 ± 1.8[#]	93 ± 4.9
CVP (mmHg)			
T	9 ± 1.3	9 ± 1.5	9 ± 1.1
N	8 ± 1.3	9 ± 1.1	8 ± 1.2
SV (cc/beat)			
T	101 ± 2.6	99 ± 9.6	99 ± 7.1
N	100 ± 6.8	101 ± 3.3	99 ± 4.9
COP (L/min)			
T	8.5 ± 0.7	8.7 ± 0.9	8.8 ± 0.7
N	8.8 ± 0.6	9.1 ± 0.5	8.7 ± 0.6
SVR (dynes/sec/cm^5)			
T	795 ± 30	445 ± 28[#]	773 ± 20
N	784 ± 18	425 ± 20[#]	770 ± 36

T: terlipressin group; N: norepinephrine group; HR: heart rate; MAP: mean arterial blood pressure; CVP: central venous pressure; SV: stroke volume; COP: cardiac output; SVR: systemic vascular resistance. All data presented as mean ± standard deviation.

[#] Significance when compared with before paracentesis ($p < 0.05$).

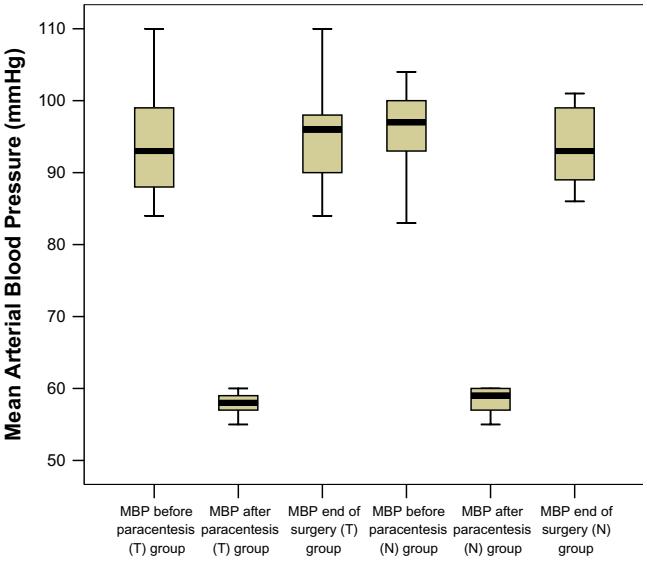

Figure 1 Changes in the (MAP) mean arterial blood pressure in both groups: before, after paracentesis and at the end of the surgical procedure (T) terlipressin group; (N) norepinephrine group.

enced bradycardia, tachycardia, hypertension or hypotension that required medication. Terlipressin and norepinephrine administrations did not alter the position of the ST segment on the electrocardiogram and did not result in dysrrhythmias.

Table 3 Metabolic parameters: blood gases, lactate, oxygen consumption and CO_2 production.

	Before paracentesis	After paracentesis	End of surgery
pH			
T	7.33 ± 0.4	7.32 ± 0.3	7.33 ± 0.2
N	7.34 ± 0.4	7.33 ± 0.3	7.34 ± 0.4
$PaCO_2$			
T	35.1 ± 1.8	34.8 ± 1.7	33.6 ± 1.6
N	36.6 ± 1.2	36.8 ± 1.6	35.8 ± 1.5
PaO_2			
T	146 ± 12.7	164 ± 14.6	162 ± 16.3
N	141 ± 13.8	167 ± 17.2	166 ± 18.1
HCO_3 (meq/L)			
T	22.8 ± 1.6	23.1 ± 1.9	23.1 ± 1.7
N	23.8 ± 1.7	22.7 ± 1.9	23.2 ± 1.7
Lactate (mg/dl)			
T	11.1 ± 2.6	12.7 ± 2.8	12.1 ± 2.4
N	11.6 ± 2.3	12.2 ± 2.7	11.9 ± 2.5

T: terlipressin group; N: norepinephrine group. All data presented as mean ± standard deviation. Normal range of lactic acid (4.5–9.8 g/dl), $p > 0.05$ in all data.

Successful weaning of both drugs was done within 24 h postoperatively.

However, all patients of both groups showed significant decreases in MAP during or immediately after the paracentesis process to reach mean values of 57 ± 1.4 and 58 ± 1.8 mmHg in terlipressin or norepinephrine groups respectively (Table 2), (Fig. 1). This was associated with drop in the SVR that reached mean values of 445 ± 28 and 425 ± 20 dynes/sec/cm^5 in both terlipressin and norepinephrine groups respectively (Table 3) (Fig. 2). Both blood pressure and systemic vascular resistance were restored after either terlipressin or norepinephrine infusions (Table 2) and (Figs. 1 and 2). There were no significant differences between the two groups regarding the COP (Table 2) and (Fig. 3), metabolic parameters which included: arterial blood gases, lactate levels (Table 3), intraoperative fluid management or the urine output (p-value > 0.05) (Table 4). Baseline liver function tests and serum creatinine did not differ between both groups (Table 5). However, at the 2nd day post operative there was significant increase in serum creatinine values in the norepinephrine group (p-value < 0.05) (Table 5). Renal impairment, in the form of rise in serum creatinine to more than 50% of baseline occurred in five patients in this group, and eight patients in the norepinephrine group showed serum creatinine level >1.5 mg/dL.

4. Discussion

Since ascites raises the intra-abdominal pressure by displacing the diaphragm and increasing intrathoracic pressure, Venous return may be impaired because of inferior vena cava and right chambers compression [22]. Moreover, compression of the descending aorta increased the after load imposed on the left ventricle [22]. Ascites evacuation has an immediate effect, because it improves cardiac performance through the Frank-

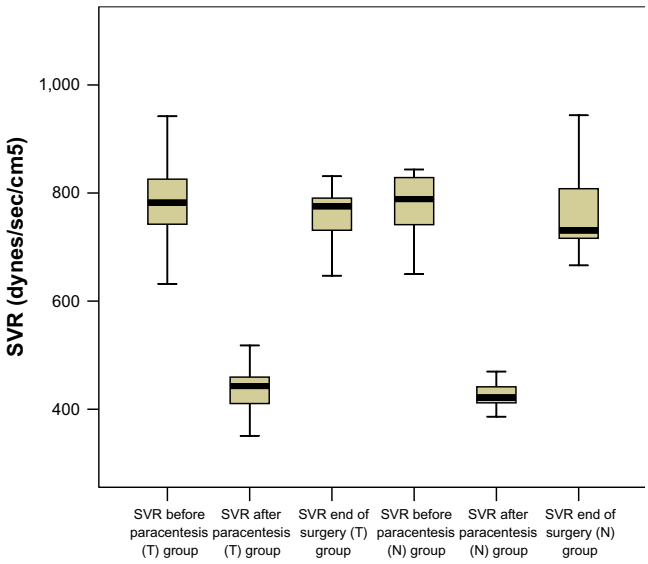

Figure 2 Changes in the (SVR) systemic vascular resistance in both groups: before, after paracentesis and at the end of the surgical procedure (T) terlipressin group; (N) norepinephrine group.

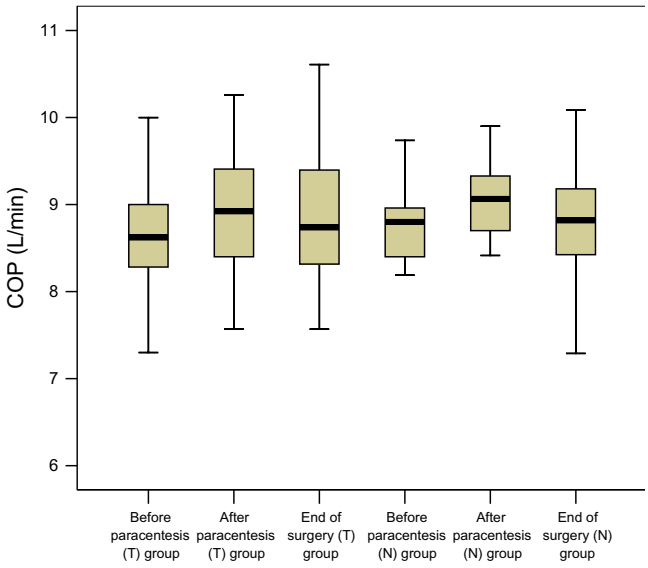

Figure 3 Changes in (COP) cardiac output in both groups: before, after paracentesis and at the end of the surgical procedure (T) terlipressin group; (N) norepinephrine group.

Table 4 Infused volumes and urine output.

	T group (n = 17)	N group (n = 17)
Crystalloids	870 ± 330	890 ± 280
Colloids (ml)	740 ± 270	690 ± 240
FFPs (unit)	4.64 ± 1.36	4.88 ± 0.99
RBCs (unit)	1.47 ± 0.51	1.58 ± 0.71
Urine output (ml/hr)	86 ± 31	89 ± 34

T: terlipressin group; N: norepinephrine group; Data are presented as mean ± SD. There are no significant differences between the two groups (p-value > 0.05).

Starling mechanism, thereby increasing cardiac output. The decrease in cardiac pressures is more evident for the right ventricle, probably because of greater compliance of this chamber [23]. This can explain that not all ascitic patients showed significant hypotension even with relatively rapid paracentesis process [24]. The decrease in SVR was relatively less than those patients who did not develop significant hypotension and were excluded from the present study.

Previous studies suggested that paracentesis could accentuate the vasodilatation already present in cirrhotic patients with ascites [25], and the present study confirmed this hypothesis. Immediately after paracentesis, a simultaneous decrease in both MAP and SVR was observed in patients of both groups. This drop in the two previously mentioned parameters might be attributed to an increase in production of nitric oxide, a powerful arterial vasodilator [26], which might be associated with mechanical decompression of the IVC. In the present work, since central venous pressure [27] and right atrial pressure in cirrhotic patients with ascites are directly correlated with intra-abdominal pressure [28,29], CVP was maintained in all patients by using albumin infusion in a dose of 8 g/L of ascitic fluid removed [30]. However, there are no studies in the literatures about intraoperative management of post-paracentesis circulatory dysfunction and hypotension in patients with cirrhosis and tense ascites who undergo emergency abdominal surgery. Under anesthesia, the sympathetic, renin-angiotensin, and vasopressin systems are involved in the control of MAP. The agonists of the sympathetic system are used as standard therapy to treat intraoperative hypotension. An α1 adrenergic agonist like norepinephrine is considered as more effective than ephedrine in treating arterial hypotension. It has a powerful vasoconstrictor activity, it constricts mesenteric vessels and reduces splanchnic vasodilatation [31]. An alternative vasopressor like terlipressin may be effective in treating hypotension in such patients because it acts on the vasopressin- humoral vasopressor system, although the sympathetic system is blunted by general anesthesia [32]. Eyraud et al, have demonstrated that a bolus of terlipressin can restore MAP with no concomitant impairment in the left ventricular function [32].

In our study, after establishment of norepinephrine infusion or after the bolus dose of terlipressin, in association with the restoration of MAP, we observed a significant restoration of the SVR and cardiac output. However, the vasoconstriction after administration of those two vasoconstricting drugs could cause, or at least worsen, hypoperfusion-related alterations in organ function. Accordingly, the main finding of the study was to investigate if there was significant decrease in liver and kidney functions, mainly linked to the potent vasoconstriction. Duvoux et al. reported improvement in renal function and a marked reduction in plasma active renin and aldosterone concentrations during norepinephrine treatment in patients with hepatorenal syndrome [31]. They suggested that the haemodynamic benefit derived from norepinephrine-induced peripheral vasoconstriction probably predominates over the action of norepinephrine on the renal artery and results in improved renal perfusion [31].

Another aim of the present study was to compare changes in liver and kidney functions. The mean value for serum creatinine at 2nd postoperative day showed that (N) group displayed a significantly higher serum creatinine than the (T) group. Terlipressin enhances renal perfusion through increas-

Table 5 Liver and kidney function tests.

	Preoperative		2 days postoperative	
	T group (n = 17)	N group (n = 17)	T group (n = 17)	N group (n = 17)
Liver functions tests				
Total bilirubin (mg/dl)	2.1 ± 1.1	2.2 ± 1.1	2.2 ± 1.2	2.3 ± 1.1
INR (ratio)	1.5 ± 0.11	1.4 ± 0.09	1.5 ± 0.1	1.5 ± 0.1
SGOT (U/ml)	93 ± 42.3	96 ± 38.3	95 ± 44.1	99 ± 41.9
SGPT (U/ml)	101 ± 47.8	99 ± 39.1	104 ± 37.1	106 ± 40.5
Renal function tests				
Creatinine (mg/dl)	1.38 ± 0.1	1.38 ± 0.09	1.41 ± 0.09	1.65 ± 0.21[*,#]

T: terlipressin group; N: norepinephrine group; Values are mean ± SD; ALT: alanine aminotransferase; (AST): aspartate aminotransferase; (INR) international normalized ratio.

[*] Significance compared with the other group ($p < 0.05$).

[#] Significance when compared with the preoperative value ($p < 0.05$).

ing both mean arterial pressure and effective arterial blood volume [33]. Another mechanism is the increased diuresis mediated by the stimulation of V1a receptors [34]. However, there were no significant differences regarding the liver function tests between the two studied groups. Finally, arterial pressure at the end of surgery did not differ significantly from baseline values in the norepinephrine or the terlipressin groups. These results, indicated that the overall response to treatment was similar in the terlipressin and norepinephrine groups. However, because of the small sample size of the study groups, a type II error cannot be excluded.

The present study had several limitations. We did not study the influence of both drugs either terlipressin or norepinephrine on the integrity of renal tubular function by using newly described sensitive markers such as urinary neutrophil gelatinase-associated lipocalin and glutathione S-transferase for hepatocellular integrity.

5. Conclusion

This research demonstrates that both drugs (terlipressin and norepinephrine) successfully counteracted the post-paracentesis refractory hypotension and the drop of the systemic vascular resistance. It also showed the renal protective effects of terlipressin in the immediate postoperative period for this category of patients with normal kidney function who went into emergency abdominal surgery However, we cannot judge with confidence whether our findings can be extended to patients with moderate to severe renal impairment.

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