

Effects of ethanol on human fractionated response times

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The effects of ethanol (EtOH) on response components varying along a central vs. peripheral dimension were studied in five subjects. Reaction times (RTs) were fractionated by electromyographical recordings into premotor (central) and motor (peripheral, contractile) components. Highly practiced subjects performed a simple and discrimination RT task and related movement without significant impairment at the moderate blood ethanol concentration (BEC) (0.10%). At the higher BEC (0.17%), all components involving central processing (response time, RT and premotor time) were impaired in both simple and discrimination RT. More peripheral components (contractile time and movement time) were little affected. Contractile time was slowed slightly but significantly, but only in the combination of EtOH and the discrimination task which suggests that the stimulus discrimination stage of information processing can influence the activation of motor units involved in carrying out the movement.

Key words: reaction time; fractionated reaction time; ethanol; alcohol; movement time; contractile time; premotor time; motor time

Introduction

The observation that EtOH slows simple and complex RT in humans has been amply recorded, beginning with Jellinick and McFarland's classic paper in 1940 [1] and exemplified by Moskowitz and Burns [2]. EtOH levels as low as 0.05–0.06% have been reported to slow simple RT significantly [3]. Some investigators have reported that EtOH has no effect on RT [4–8], but others have suggested that the failure to observe changes in RT under EtOH conditions could have been confounded by factors such as low blood ethanol concentrations (BECs), sex differences, inadequate practice, stimulus-response compatibility variations and motivational differences [9,10]. Although the evidence is not unanimous, it is generally

accepted that most individuals with BECs from 0.08 to 0.10% or higher are substantially impaired in manual tasks [3]. The prevailing belief is that the major effect of EtOH is on the information processing (premotor) occurring prior to the activation of skeletal muscle. Based on findings from studies involving animal models, we suspected that the doses of EtOH administered to human subjects are too low to produce changes in peripheral response events such as the muscle contractile properties.

Response time, the time to perceive an environmental stimulus such as a light or sound and make a goal-directed response to it, may be differentiated into RT and movement time. RT, by the use of electromyographical (EMG) recording, may be further fractionated into premotor time and contractile time [11]. Premotor time, from the onset of the stimulus until the recording of the first electrical activity in

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the muscle prior to observable movement, is thought to be a measure of central nervous system information processing, uncontaminated by peripheral mechanics. Contractile time, from the first EMG activity of the muscle to when the limb is lifted off the starting switch, is proposed to be the time for the peripheral biochemical mechanisms necessary to produce shortening and generate enough force to initiate a movement in the limb. Movement time, beginning at the end of RT and contractile time, is the duration of time taken for the limb to move from the starting switch to a target switch. Our purpose was to study the locus (central vs. peripheral) of response slowing with EtOH.

While the effects of EtOH on simple and complex RTs are well known, its effects on the components of a fractionated response (premotor time and contractile time) and movement time are not. Sutton and Kimm [12] reported a non-significant slowing in premotor time of 12.3 ms after low doses of EtOH; however no analysis was made of contractile time. The studies of Tharp et al. [10] and Moskowitz and Burns [2] strongly suggest that the major effects of EtOH are to slow decision making and response selection (premotor time), not to lengthen contractile time. There have been no previous reports on the effects of EtOH on the response components, premotor and contractile time, of the same well-practiced subjects. The reported effects of EtOH on movement time are mixed. King [13] found that by producing very high BECs (0.22%) both subject-initiated and experimenter-initiated movement times were 55% and 45% slower, respectively. Landauer and Howat [14] reported no EtOH effect on movement time, but their BECs were somewhat low (0.07%) and the movement in their experimental paradigm followed the presentation of an extremely complex stimulus display.

In the present study we have recorded EMG to study the effects of EtOH on movement time, RT and its fractionated components: premotor time and contractile time. A simple RT and a discrimination RT task which required identical movements following the two different stimulus processing requirements were selected

in order to examine EtOH effects on response components varying along a central vs. peripheral mechanism dimension. We have argued that the use of a choice RT is not appropriate for central vs. peripheral questions because the muscle activation are compared in responses involving different muscle actions and in most cases different muscles. The simple RT/discrimination RT paradigm allows these comparisons to be made in the same muscles producing the identical movement.

Methods

Subjects

Five healthy subjects (three males and two females), without any neurological disorders volunteered to participate. All subjects were classified as 'moderate drinkers' with three being subdivided as 'frequent moderate' and two as 'infrequent moderate' [15]. All subjects drank primarily beer and wine with infrequent use of distilled spirits. The age ranged from 26 to 48 years with a mean age of 34 years. All subjects had fasted at least 12 h prior to being tested.

Apparatus

Subjects sat in an adjustable dental chair facing a wall-mounted stimulus light panel set at eye level approximately 1 m away. The red and green stimulus lights were 120 V AC neon lamps, measuring 2 cm in diameter and set in a flat-black background 5 cm apart. The response apparatus was designed to simulate both an accelerator (the RT microswitch) and a brake pedal (the movement time microswitch) of an automobile. These two foot-switches were mounted adjacently at 40° angles to the subject with a wooden block containing the brake microswitch 18 cm to the left and slightly above the accelerator. The foot-switch apparatus was set on the floor in front of the subject directly below the stimulus panel.

General procedures

Subjects were tested on 7 separate days. Days 1, 2 and 3 were practice days to minimize the well-known effects of learning on these tasks. On days 4 and 5 subjects were tested in

the high EtOH dose session. Day 6 was a practice day. On day 7, subjects were tested in the moderate EtOH dose session. Control values were compared to the pre-EtOH values to insure that baseline values were obtained.

In the simple version of the task (simple RT), upon hearing a warning buzzer, subjects depressed the accelerator (RT) switch with the heel of the foot. After a randomly assigned warning interval, the red stimulus light was activated. Subjects responded as quickly as possible to the onset of a red light by lifting the heel from the accelerator (simple RT) and moving to depress the brake switch (movement time). Simple RT stimuli were administered in 2 blocks of 28 trials each. In the discrimination version of the task, the procedure was similar except that either the green or red light ($P = 50\%$) was activated in a random order. When the red light was activated, the subjects' response was the same as in the simple version of the task, but subjects made no response to the green light except to maintain the depression of the accelerator switch and await the next trial. Discrimination RT trials were administered in 2 blocks of 56 trials, (28 red per block). A 2-min rest was given after every 28 trials. Although the intertrial interval remained constant at 6 s, the warning interval for each trial was a randomly assigned 3, 4 or 5 s.

Administration of EtOH

Subjects ingested a 1.0 g/kg dose of 95% UPS EtOH to provide a BEC approximating 0.10%, or the legal intoxication level for the moderate dose test session and a 1.5 g/kg dose to provide BECs of approximately 0.16% for the higher dose test session. These values were targeted because more than 90% of the population is impaired in driving ability with BECs of 0.10%, and higher BECs were desired as a comparison [3]. The EtOH was diluted in orange juice (1 part EtOH : 5 parts juice) and subjects were encouraged to consume the mixture throughout a 30-min period. Triplicate blood samples (50 μ l; finger prick) were taken at the beginning and the conclusion of the RT testing, providing an interval between samples of approximately 40 min.

Procedures for high dose testing session (target BEC = 0.16)

After 3 days of practice which consisted of 2 simple RT blocks (56 trials) and 2 blocks of discrimination RT trials (112 trials, 56 of which were 'go' trials), the protocol was repeated except that warm-up trials (15 go trials each of simple RT and discrimination RT) were administered prior to test trials and the responses were fractionated by EMG recordings. On day 5 following warm-up trials, subjects were measured by the same EMG protocol following the consumption of EtOH. Each day contained 2 simple RT blocks (28 go trials per block) and 2 discrimination RT blocks (56 trials per block, 28 go). Block order was rotated on practice days, but each subject's order was constant for the test days; three subjects began with discrimination RT and two with simple RT. Following warm-up trials subjects were prepared for EMG recording and consumed, within a 30-min period, the specified amount of EtOH. RT testing began at the end of the 30-min period and the RT analysis trials lasted for about 20 min.

Procedures for moderate dose testing session (target BEC = 0.10%)

Five months following the first session, a second but lower EtOH dose session was repeated with the same subjects who were given one RT practice session on day 6 (2 simple RT and 2 discrimination RT blocks) to insure that responses were well retained. Since an ANOVA from the first session's data showed no trial and block differences, the number of EMG test trials (day 7) was halved (28 simple RT and 28 discrimination RT go trials) to enable pre-EtOH EMG and EtOH EMG data to be taken on the same day with the same electrode placement (1 simple RT and 1 discrimination RT block per condition). (Although EMG latency comparisons are acceptable with repeated electrode placements, other EMG analyses are valid only for a single placement; however, since no behavioral differences were evident, no additional EMG analyses were added.) Following warm-up trials, all trials were administered in the same manner as in the first session.

Electromyography

From pilot testing the following muscles were selected for monitoring: agonists — iliopsoas, rectus femoris, adductor magnus and antagonists — gluteus maximus and semitendinosus. Standard Beckman surface electrodes were applied, and acceptable skin impedance was 10 k Ω . EMG was amplified by Bak Differential Amplifiers with a mean amplification of 1100. The low frequency cutoff was 5 kHz; high was 50 Hz. Signals were monitored (Tektronix oscilloscope) and recorded on a Hewlett Packard Instrumentation tape recorder with EMG channels calibrated at 2.5 V. Analog EMG signals and DC signals indicating stimulus occurrence, release of the RT microswitch, and depression of the movement time switch were sampled at 2 kHz for computer analyses. The software (R.C. Electronics) enabled the computer display to mimic an oscilloscope displaying all channels of data and by using manual cursors to read the first change above baseline activity with the accuracy of 1 ms. Premotor time was defined according to Weiss [11] as the onset of EMG in the first responding agonist muscle following the stimulus. (Responding muscles tended to be silent prior to the rapid response).

Blood EtOH analysis

The blood samples were transferred to 20 ml vials and frozen until analysis. The vials were heated to 60°C in a heated rotary turntable and analyzed by headspace gas chromatography with a Perkin Elmer Sigma 2000 chromatograph. Analytical parameters were aluminum column, 1.8 mm \times 3 mm with Porapak OS, 80–100 mesh, column temperature 190°C; carrier (nitrogen flow), 30 ml/min; injection and detector temperatures, 200°C. External standards were used for calibration.

Data analyses

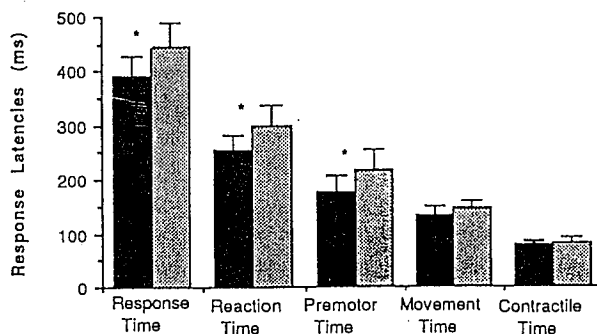
Initially the RTs and movement times of practice day blocks were compared to insure that subjects' responses were stable. Error responses and response latencies that were beyond ± 2 S.D. from each subject's block mean

for each condition were eliminated from the analyses, and the means and S.D. were re-computed. Following the high dose session, the test data from the pre- and EtOH days were analyzed (ANOVA), and no main effects for blocks or trials were found. For the primary analyses, the corresponding first 2 blocks of the pre- and EtOH conditions for the high dose session were compared to those for the moderate dose session. A within-group repeated measures ANOVA was completed on the means and within-subject S.D. for each dependent variable (response time, RT, premotor time, contractile time, movement time). This procedure resulted in a subjects (5) by dose (high dose session, moderate dose session) by EtOH condition (pre, EtOH) by response type (simple RT, discrimination RT) by warning interval (3, 4 and 5 s). Each subject's cell score represented the mean of at least 7 trials.

Results

BECs

Mean BECs were 0.10% \pm 0.02 prior to RT testing and 0.13% \pm 0.02 at the conclusion of the testing session (approximately 40 min) for the moderate dose session. In the high dose session, the mean of the initial samples was



*Denotes EtOH score is significantly slower than pre-EtOH score at $p < .05$.

Fig. 1. Pre-EtOH and EtOH means and S.D. were collapsed across high and moderate EtOH sessions for each dependent variable. All variables containing the 'central processing component' were significantly slowed (*) by EtOH. Variables representing the peripheral components were little affected by EtOH. ■, Pre-EtOH; ▨, EtOH.

Table I.
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Table I. Summary of significant ANOVA findings for mean comparisons. Only significant findings ($P > 0.05$) are listed. Values are means \pm one S.D. SRT, simple reaction time; DRT, discrimination reaction time.

| | | <i>F</i> | <i>(d.f.)</i> |
|--|-----------------|----------------|---------------|
| Response time | | | |
| <i>EtOH condition</i> | | | |
| Pre-EtOH | | EtOH | 11.95 (1, 4) |
| 392 \pm 35 | | 445 \pm 44 | |
| <i>Response type</i> | | | |
| SRT | | DRT | 23.84 (1, 4) |
| 392 \pm 31 | | 444 \pm 48 | |
| <i>Warning interval</i> | | | |
| 3 s | 4 s | 5 s | 4.31 (2, 8) |
| 433 \pm 39 | 412 \pm 41 | 409 \pm 39 | |
| <i>Significant interactions (none)</i> | | | |
| Reaction time | | | |
| <i>EtOH condition</i> | | | |
| Pre-EtOH | | EtOH | 29.56 (1, 4) |
| 256 \pm 27 | | 299 \pm 39 | |
| <i>Response type</i> | | | |
| SRT | | DRT | 25.23 (1, 4) |
| 253 \pm 22 | | 302 \pm 44 | |
| <i>Warning interval</i> | | | |
| 3 s | 4 s | 5 s | 4.80 (2, 8) |
| 293 \pm 34 | 275 \pm 35 | 266 \pm 30 | |
| <i>Significant interactions (none)</i> | | | |
| Premotor time | | | |
| <i>EtOH condition</i> | | | |
| Pre-EtOH | | EtOH | 23.15 (1, 4) |
| 178 \pm 27 | | 217 \pm 38 | |
| <i>Response type</i> | | | |
| SRT | | DRT | 20.23 (1, 4) |
| 174 \pm 21 | | 221 \pm 44 | |
| <i>Warning interval</i> | | | |
| 3 s | 4 s | 5 s | 4.49 (2, 8) |
| 212 \pm 34 | 195 \pm 35 | 186 \pm 28 | |
| <i>Interaction (EtOH dose \times EtOH condition)</i> | | | |
| | Pre-Etoh | EtOH | 7.76 (1, 4) |
| High dose: | 174 \pm 27 | 244 \pm 49 | |
| Moderate dose: | 182 \pm 28 | 190 \pm 26 | |
| Movement time | | | |
| <i>Significant main effects and interactions (none)</i> | | | |
| Contractile time | | | |
| <i>Significant main effects (none)</i> | | | |
| <i>Interaction (EtOH) condition \times response type)</i> | | | |
| | SRT | DRT | 7.38 (1, 4) |
| Pre-EtOH: | 77.4 \pm 7.3 | 76.7 \pm 8.2 | |
| EtOH: | 80.8 \pm 10.4 | 84.2 \pm 9.0 | |

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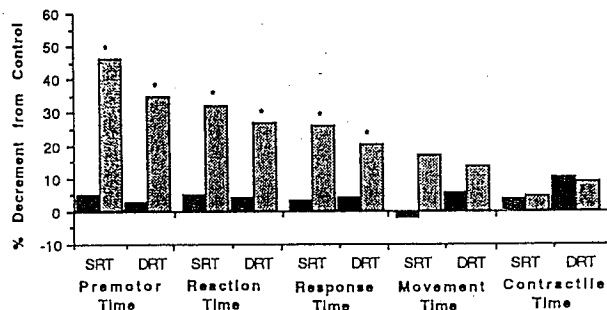
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0.17% \pm 0.03 and the final samples was 0.17% \pm 0.03. Since only the first 20 min of RT trials were included in the analyses, it is likely that all subjects were on the ascending limb of the blood EtOH curve.

Performance latencies

As seen in Fig. 1 in which all variables are collapsed across EtOH dose level, three of the five dependent variables were very similar. (See Table I for significant ANOVAs). For these three variables (response time, RT and premotor time), the pre-EtOH trials were faster than EtOH trials, the simple RT condition was faster than the discrimination RT condition, and responses following a 3-s warning interval were slower than those following a 4- or 5-s interval. In Fig. 2 the percent decrement with EtOH from pre-EtOH means is plotted for all variables in the simple and the discrimination RT condition. Clearly EtOH levels must be quite high to produce a substantial slowing of either RT response in highly practiced subjects. Moderate BECs (0.10%) had almost no influence on either simple or discrimination RT, but high



*Denotes EtOH score is significantly slower than pre-EtOH score at $p < .05$.

Fig. 2. In the high dose session (mean BEC of 0.17%), the percent decrements with EtOH from pre-EtOH values reflect significant slowing (*) for simple reaction time (SRT) and discrimination reaction time (DRT) tasks in all variables containing the 'central processing component'. The variable most representative of central processing, premotor time, showed the largest percent decrement. In the variables representing the peripheral components and in all variables in the moderate EtOH-dose-session (mean BEC of 0.10%), the small decrements reflect non-significant mean differences. ■, Moderate dose; ▨, high dose.

BECs (0.17%) produced a dramatic decrement in performance on both tasks. EtOH, therefore, did not differentially affect discrimination RT. In fact, the time taken to make the discrimination in the pre-EtOH (discrimination RT—simple RT) was 48.1 ms, and with EtOH it was 49.1 ms. The pre to EtOH decrement for response time, RT, and premotor time ranged between 3–5% with moderate BECs, and between 20 and 46% with high BECs. Note also in Fig. 2, that the EtOH decrement is greatest for premotor time (46 and 35%), the variable most representing the central component. For premotor time the interaction between BEC and the pre-EtOH condition was significant. Newman-Keuls Test for Ordered Means indicated that the high EtOH condition was slower than the other three means indicating that the slowest premotor times were produced in the high EtOH session. Movement time was slightly but not significantly slowed even by the high BECs.

Among the most interesting findings were those of small, but significant differences in contractile time, a response component that traditionally has been considered an invariant peripheral factor of the response. For contractile time, the interaction between the factors of RT condition and EtOH was significant. Analysis of the interaction revealed that with EtOH in the discrimination RT response contractile time was slower than in all of the other response conditions and that with EtOH contractile time in the simple RT condition was slower than in either of the pre-EtOH conditions.

Subject consistency

Analyses of the subject's consistency (the S.D. of subjects' responses around their own trial block means (Table II) revealed similar results. For response time, RT and premotor time, subjects were more consistent with moderate BECs than with high BECs, in Pre-EtOH than in EtOH test sessions, and in simple RT trials than in discrimination RT trials. Additionally, BEC interacted with the pre/EtOH condition and response type to reveal that

Table II. Summary of significant ANOVA findings for comparisons of within-subject S.D. Only significant findings ($P > 0.05$) are listed. Values are means. SRT, simple reaction time; DRT, discrimination reaction time.

| | | | F | (d.f.) |
|---|---------------|---------------|---------------|--------|
| Response time | | | | |
| <i>EtOH</i> dose | | | | |
| High dose | Moderate dose | | 7.82 | (1, 4) |
| 44.4 | 34.6 | | | |
| <i>EtOH</i> condition | | | | |
| Pre- <i>EtOH</i> | <i>EtOH</i> | | 16.27 | (1, 4) |
| 35.4 | 43.6 | | | |
| <i>Response type</i> | | | | |
| SRT | DRT | | 13.73 | (1, 4) |
| 30.9 | 48.1 | | | |
| <i>Significant interactions (none)</i> | | | | |
| Reaction time | | | | |
| <i>EtOH</i> dose | | | | |
| High dose | Moderate dose | | 8.44 | (1, 4) |
| 38.5 | 27.5 | | | |
| <i>EtOH</i> condition | | | | |
| Pre- <i>EtOH</i> | <i>EtOH</i> | | 8.18 | (1, 4) |
| 27.0 | 39.1 | | | |
| <i>Response type</i> | | | | |
| SRT | DRT | | 12.98 | (1, 4) |
| 22.4 | 43.7 | | | |
| <i>Significant interactions (none)</i> | | | | |
| Premotor time | | | | |
| <i>EtOH</i> dose | | | | |
| High dose | Moderate dose | | 8.69 | (1, 4) |
| 37.8 | 27.1 | | | |
| <i>EtOH</i> condition | | | | |
| Pre- <i>EtOH</i> | <i>EtOH</i> | | 7.98 | (1, 4) |
| 27.4 | 37.5 | | | |
| <i>Response type</i> | | | | |
| SRT | DRT | | 11.93 | (1, 4) |
| 21.3 | 43.6 | | | |
| <i>Interaction (EtOH dose × EtOH condition × response type)</i> | | | | |
| | High dose | | Moderate dose | |
| | SRT | DRT | SRT | DRT |
| Pre: | 15.6 | 38.2 | 16.7 | 38.9 |
| Post: | 34.3 | 63.2 | 18.6 | 34.0 |
| | | Pre: | | |
| | | <i>EtOH</i> : | | |
| Movement time | | | | |
| <i>Warning intervals</i> | | | | |
| | 3 s | 4 s | 5 s | 4.88 |
| | 13.5 | 15.2 | 15.9 | |
| <i>Interaction (EtOH condition × warning interval)</i> | | | | |
| | 3 s | 4 s | 5 s | 11.55 |
| Pre: | 13.3 | 15.2 | 13.4 | |
| <i>EtOH</i> : | 13.7 | 15.2 | 18.4 | |
| Contractile time | | | | |
| <i>Main effects and interactions (none)</i> | | | | |

Table III. Summary of error responses.

| | High BEC | | Moderate BEC | |
|--------------------------------|----------|------|--------------|------|
| | Pre | EtOH | Pre | EtOH |
| Missed brake | 4 | 15 | 5 | 8 |
| Anticipated light | 1 | 0 | 2 | 0 |
| Responded incorrectly to green | 9 | 22 | 9 | 16 |
| Failed to respond to red | 0 | 4 | 0 | 1 |
| Failed to respond to movement | 0 | 1 | 0 | 0 |
| Total errors | 14 | 42 | 16 | 25 |

although subjects' consistency was unaffected at moderate BECs for both simple and discrimination RT, high BECs produced increases in performance variability, especially on the discrimination task. Analysis of warning intervals indicated that subjects became less consistent under EtOH at the 5-s, or longest warning interval. None of the experimental factors influenced subjects' consistency in contractile time.

Errors

A frequency tally of the error responses revealed three types of errors committed in the pre-EtOH condition: (1) the subject missed the brake pedal; (2) the subject anticipated the stimulus and responded too soon; (3) the subject incorrectly responded when the stimulus was green instead of red (Table III). During the EtOH responses, subjects no longer made the anticipation errors, but they continued to miss the brake on some trials and respond incorrectly to the green light. With EtOH, subjects also made two other types of errors: (1) they failed to respond to the red stimulus light and (2) once in the high BEC session, a subject released the RT switch but failed to complete the response by making the movement. As expected the frequency count for all types of errors except anticipation was greater with higher BECs. A chi square analysis of the total number of errors in the high and moderate sessions using the number of errors of the pre-sessions as the expected frequency revealed that with EtOH the number of errors was greater in both the moderate and high dose sessions. Using the number of errors in the moderate

dose session as the expected frequency showed that the number of errors in the high dose session was greater than in the moderate dose session.

Discussion

We have concluded that individuals who are highly practiced on non-verbal information processing movement tasks requiring simple identification and discrimination of stimuli are not greatly impaired (2–5%) with moderate BECs of 0.10% which is the level that constitutes the legal definition of driving while intoxicated and is the value that Baker et al. [3] have suggested impairs most individuals. A few other experimenters have reported no EtOH effects on RT [4–8]; but generally at this BEC, we expect impairment in most manual tasks. Our mean differences at the moderate BEC are really quite small, and although we cannot determine the cause from our data, we suggest three probable contributing factors. (1) The subjects were highly practiced with very stable baseline data and EtOH was not introduced until the responses were well learned. We have not found reports of this level of practice on EtOH impairment in a simple movement task. (2) The two tasks are very simple with no movement choices to be made other than go or no go. The stimulus is simply a trigger, containing no additional information, to initiate a well-practiced movement. In fact the EtOH and response type interaction was not significant indicating that both tasks were equally affected by EtOH. The choice RT paradigm which has been com-

monly used in studies of EtOH and RT was rejected for this study because we wanted the same movement for our comparisons of contractile time. EtOH impairment at very low BECs (0.02–0.06%) has been reported in skills performance of more complex tasks involving tests with divided attention and more complex information processing requirements [16]. (3) All reactions followed a random but optimum foreperiod which allowed the subject to concentrate on the stimulus for a short period of time just prior to the response. A probable effect of EtOH is on response preparation mechanisms. Gottsdanker [17] has reported that a high level of preparation is an 'aversive state', avoided when possible, but controlled by and maintained when the expectancy of the stimulus is high. As subjects prepare a response to a stimulus, they subjectively calculate the probability or expectancy of the stimulus presentation [18]. RT closely follows the objective probability of the stimulus occurrence when the subject has had the opportunity to become familiar with the temporal and spatial structure of the experimental paradigm. Certainly our subjects were well-practiced and familiar with the temporal structure and EtOH did not appear to impair an individual's ability to calculate the probability of stimulus occurrence because the presence or level of EtOH did not alter the traditional warning interval findings. It is plausible, however, that high BECs may impair an individual's ability to attain or maintain a high level of preparation and/or attend to the stimulus which is about to occur. This suggestion is somewhat supported by the fact that no anticipation errors occurred in the EtOH state, i.e., with EtOH the subject was so unprepared that he/she no longer 'jumped the gun'.

The high BEC (0.17%), as expected, significantly slowed both simple and discrimination RT, but not the following movement. For pre-motor time, RT and response time, the type of response did not interact with either the presence or the level of EtOH. The observation that the size of decrement (Fig. 2) appears related to the degree to which the response variable represents central information processing is con-

sistent with reports that the major effects of EtOH are central rather than peripheral. Movement time, the only response variable to the uninfluenced by any level of EtOH, is almost purely a peripheral component and in this paradigm accounts for 36% of response time in simple RT and 31% in discrimination RT. Landauer and Howat [14], although they achieved relatively low BECs in their subjects reported no EtOH effects for movement time. King [13], who achieved very high BECs of 0.22% in subjects reported a 55% slowing in movement time. Since 0.23% is considered to be an anesthetic BEC, it is not surprising that movement time was slowed under these conditions.

The interaction of the EtOH condition with the discrimination type of response in lengthening contractile time suggests that rather than being solely a peripheral event, it is at least in part influenced by central information processing. Recall that the movement in this paradigm (lifting the foot from an 'accelerator' to a 'brake') was designed to be identical in both RT tasks except that in one task the subject was required to discriminate which one of two potential stimuli was activated prior to the initiation of movement. If contractile time was purely a peripheral event, comprised of delays attributable to synaptic transmission, muscle twitch velocity, one-half relaxation time, and other muscle contractile mechanisms, it should be the same duration in identical movements, independent of the complexity of central information processing stages that precede the contraction mechanics. One would not expect to see a longer time in a discrimination RT paradigm than in a simple RT paradigm unless the central processing of the discrimination was in some way reflected in the activation of motor units involved in the movement.

Animal models of EtOH indicate that it is unlikely that the EtOH doses in this study were producing these effects by acting directly on muscular properties or on spinal motor neuronal pools. Contractile capabilities of muscle are largely unaffected by moderate doses of EtOH [19]. Twitch tension is actually increased with acute applications of EtOH, but it requires

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4 g/kg to see this change [20]. Peiris et al. [21] reported nerve conduction velocity to be unaffected if intramuscular temperature is controlled, and Reed [22] did not find nerve conduction velocity slowing until EtOH doses exceeded 2.5 g/kg. One-half relaxation time of muscle was also unaffected even by a BEC as large as 4 g/kg [20]. If EtOH were affecting only these muscle contractile properties or the spinal motoneuronal pools from which the active motor units are recruited, the effects should be similar for both simple and discrimination conditions. The fact that CT was disproportionately longer in the discrimination task under EtOH intoxication suggests that central factors involved with stimulus discrimination and EtOH are influencing motor unit recruitment and thus contractile time.

Because subjects were so highly practiced prior to test sessions, their consistency of performance was very high; within-subject standard deviations were only 9% of the mean in simple RT and 21% in discrimination RT. Many investigators [23,24] have observed that EtOH increased within-subject variability of performance; however, moderate BECs (0.10%) in our highly-practiced subjects performing simple tasks did not decrease subject consistency. The high BEC, however, produced greatly increased variability of performance in simple and especially in discrimination RT.

In summary, if highly motivated individuals fully attend to a well-practiced simple and discrimination reaction task, they can perform it without impairment at a BEC described as legal intoxication. However, the failure to show EtOH impairment at the moderate dose on this simple laboratory task cannot be readily generalized to statements about performance in general where vigilance, task complexity and motivation may interact to produce impairment. However, if the BEC is as high as 0.17%, all components involving central processing (response time, premotor time, RT) are impaired in both simple and discrimination RT. EtOH effects on contractile time, evident only in the combination of EtOH and the discrimination RT condition, suggest the stimulus dis-

crimination stage of information processing least in part affects the activation of motor units involved in carrying out the motor program. Thus contractile time should not be viewed as purely a peripheral component of the motor response. Movement time was slightly, but not significantly slowed by high BECs.

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