Introduction

Social impairment is a defining clinical feature of Autism Spectrum Disorder (ASD). However, little is known about how it concretely unfolds during social exchanges, how interlocutors pick up and react to social cues, and how patterns of interaction are affected (Dale et al. 2013). Previous research highlighted distinctive qualitative aspects of speech and gesture and their relation to social communication deficit (Fusaroli et al. 2016; Lambrechts et al. 2015).

Here we want to develop automated, quantitative methods to assess social impairment in conversation involving adults with ASD.

1. How does autistic social impairment manifest itself in conversations?
2. How does the interlocutor react to dysfluency?
3. Are these dynamics related to specific clinical features?

Methods

Procedure: The framework of a previous naturalistic experiment (Maras et al., 2013) provided us with provided audio recordings of 17 ASD and 17 matched Typically Developing (TD) adults. Participants took part in a live event scenario in which they performed first aid manipulations on a mannequin following a script. Later on, participants were interviewed on what they recalled of the event. The first part of the interview was a monologue, followed by a dialogical Q&A session. The current analysis focuses on the dialogue data only (same interviewer across recordings).

Materials: We automatically separated vocal productions from the interviewer and the interviewee using a custom-made speaker diarization system. The initial monologue was excluded.

This yielded 2866 total utterances:
- 800 produced by interviewees with ASD and 853 by their interlocutors
- 600 produced by control interviewees and 613 by their interviewer
- 600 produced by interviewees with ASD and 853 by their interviewer

Time-coded transcripts were used to calculate:
- Turn duration
- Inter-turn latency
- Number of turns for each interlocutor
- Percentage of spoken time in the conversation (for the interviewee)
- Backchanneling (for the interviewer)

1. Results: Participants’ distinctive behaviours

### Duration

- Shorter utterances in ASD (10.31±15s) compared to TD (12.81±14.4s) participants; (B=1.29, CIs:1.58-0.96)
- All participants increased utterance length over time (B=0.27, CIs: 0.18-0.35), with steeper increase in ASD interviewers (B=-0.13, CIs: -0.23 -0.02)
- Clinical features: longer utterances associated with higher AQ scores (B=0.33, CIs: 0.07 0.59) and lower ADOS communication scores (B=-0.48, CIs: -0.87 -0.07).

### Turn duration

- No main effect of diagnosis (B=0.00, CIs: -0.14 0.14)
- Latency increased with time (B=0.16, CIs: 0.07 0.25) and this increase was steeper for neurotypical than for ASD (B=0.14, CIs: -0.24 -0.03)
- AQ and ADOS RSI improved the model, but had negligible effect sizes (B=0, CIs: -0.01 0.01).

### Inter-turn latency

- No main effect of diagnosis (B=0.00, CIs: -0.14 0.14)
- Latency increased with time (B=0.16, CIs: 0.07 0.25) and this increase was steeper for neurotypical than for ASD (B=0.14, CIs: -0.24 -0.03)
- AQ and ADOS RSI improved the model, but had negligible effect sizes (B=0, CIs: -0.01 0.01).

2. Results: Interviewer’s distinctive behaviours

- No main effect of diagnosis (duration: B= -0.01, CIs: -0.33 0.16; inter-turn latency: B=0.01, CIs: -0.24 0.26)
- Turns (B= -0.06, CIs: -0.1 -0.01) and inter-turn latency (B=0.09, CIs: -0.18 -0.01) decreased in length over time
- Neither interaction with diagnosis nor clinical features improved the model

Note: All relevant measures were centred and scaled, so estimates are reported as standard deviations. We employed conservative Laplace priors (Mean=0 and SD=0.1) for all effects. WAIC-based model selection was used to determine which predictors to include.

3. Results: Coordinative Dynamics

Participants adjusted to their interlocutor’s duration (B=0.34, CIs: 0.19 0.48), although those with ASD adjusted less (B= -0.2, CIs: -0.4 -0.01). ADOS RSI scores likely affected the adaptation process (B=-0.11, CIs: -0.2 0).

The interviewer only adjusted to the interviewee’s inter-turn latency and backchanneling. The longer the participant’s latency, the longer the interviewer’s latency (B=0.29, CIs: 0.2 0.36), an effect that was not modulated by diagnosis or clinical features. Interestingly, the amount of backchanneling and general scaffolding provided by the interviewer was related to the participants’ inter-turn latency (B=1.4, CIs: 0.3 2) in interaction with duration (B=-2.4, CIs: -3.2 -1). The longer the latency, the more backchanneling, especially when the participant had just produced short utterances. These effects were affected by AQ scores and ADOS Communication (in both cases the higher the score, the stronger the effects).

These coordinative dynamics (adaptation, lack thereof and compensation) might also explain why the amount of information provided by the participants was not likely affected by diagnosis (B=0.06, CIs: -0.2 0.12) or severity of clinical features.

Conclusions

We observe conversational markers of ASD and social impairment (as measured by AQ and ADOS): shorter utterances, decreased adaptation to one’s interlocutor.

The interviewer seems to provide adaptive scaffolding to the conversation on a turn-by-turn basis, increasing latency and providing more backchanneling for slower participants, the more so the more severe the clinical features.

Future work will explore the role of context and familiarity in these dynamics and computational models of the adaption to better understand the underlying mechanisms.

References


This work was supported by an Interacting Minds Center seed funding.