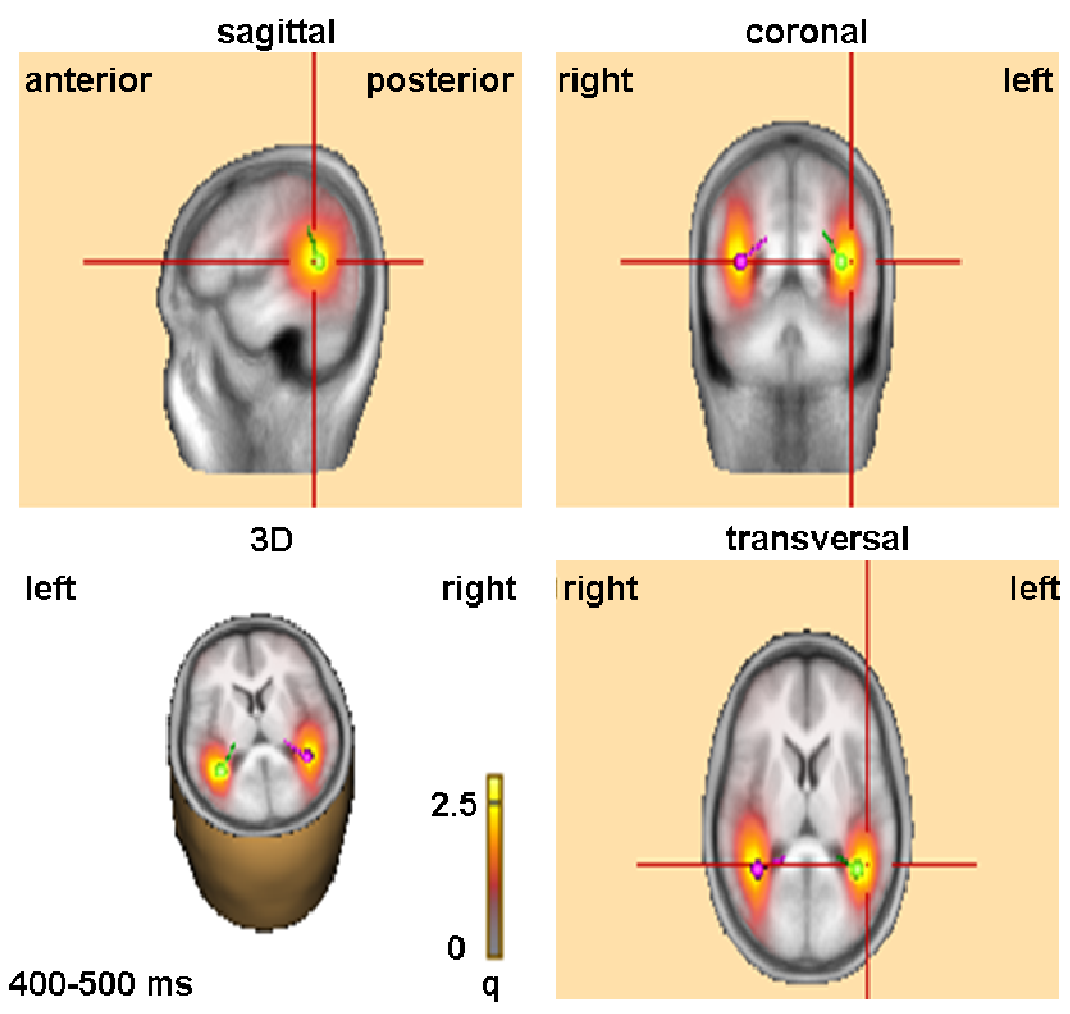


Supplementary table 1 (part 1): mean amplitudes (μV) and latencies (ms) for all ERPs and mean dipole activation (nAm) and explained variances separate for participants with autism spectrum disorder (ASD) and typically developing controls (TYP).

	ASD	TYP	<i>statistics</i>
P100 Amplitude (μV)			
<i>Walker</i>			
O1 ($\pm\text{SD}$)	7.79 (± 3.37)	10.32 (± 5.47)	
O2 ($\pm\text{SD}$)	8.49 (± 5.11)	12.67 (± 5.23)	
<i>Scramble</i>			
O1 ($\pm\text{SD}$)	8.79 (± 3.60)	10.76 (± 6.21)	GROUP
O2 ($\pm\text{SD}$)	8.92 (± 5.13)	13.13 (± 5.76)	$F_{1,35}=4.9$; $p=0.03$
P100 Latency (ms)			
<i>Walker</i>			
O1 ($\pm\text{SD}$)	125.9 (± 10.1)	137.4 (± 12.7)	
O2 ($\pm\text{SD}$)	126.4 (± 10.8)	135.5 (± 15.6)	
<i>Scramble</i>			
O1 ($\pm\text{SD}$)	126.5 (± 12.2)	133.7 (± 12.9)	GROUP
O2 ($\pm\text{SD}$)	127.7 (± 12.9)	136.3 (± 18.1)	$F_{1,35}=6.7$; $p=0.014$
N200 Amplitude (μV)			
<i>Walker</i>			
P9 ($\pm\text{SD}$)	-10.08 (± 4.66)	-9.13 (± 5.44)	
P10 ($\pm\text{SD}$)	-9.96 (± 5.71)	-13.05 (± 6.96)	
<i>Scramble</i>			
P9 ($\pm\text{SD}$)	-8.83 (± 4.65)	-8.50 (± 4.39)	GROUP*HEMISPHERE
P10 ($\pm\text{SD}$)	-8.23 (± 5.34)	-11.16 (± 5.47)	$F_{1,35}=6.1$; $p=0.018$

Supplementary table 1 (part 2)

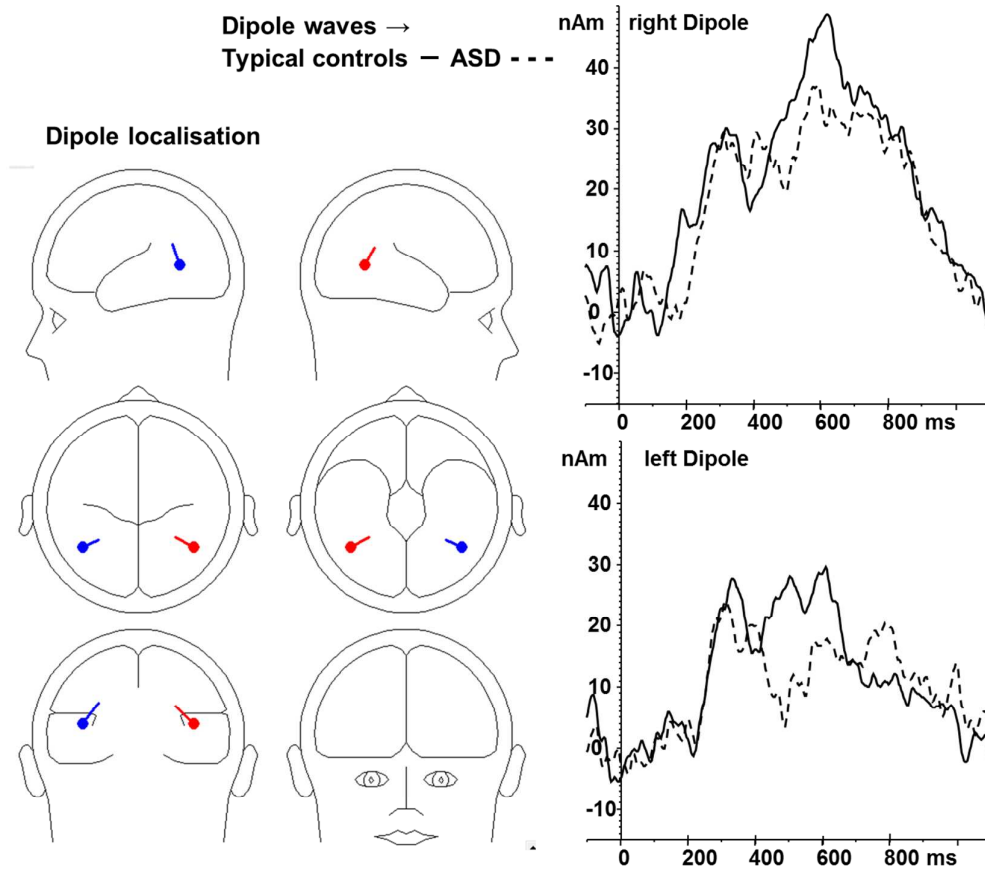
	ASD	TYP	<i>statistics</i>
N200 Latency (ms)			
<i>Walker</i>			
P9 (±SD)	225.2 (28.4)	225.3 (20.7)	
P10 (±SD)	225.8 (29.5)	228.0 (21.0)	
<i>Scramble</i>			
P9 (±SD)	215.3 (±20.6)	221.2 (±15.6)	
P10 (±SD)	210.6 (±27.7)	225.9 (±14.6)	no effects
P400+ Amplitude (µV)			
<i>Walker</i> (±SD)	8.99 (±4.07)	12.33 (±6.05)	GROUP
<i>Scramble</i> (±SD)	8.38 (±3.50)	11.14 (±5.45)	F _{1,35} =3.5; p=0.071
Dipole activation (nAm)			
<i>left</i> (±SD)	15.52 (±19.43)	31.99 (±28.24)	GROUP
<i>right</i> (±SD)	23.56 (±32.29)	33.89 (±25.41)	F _{1,35} =3.16; p=0.08



Supplementary figure 1: Automated multiple source probe scan (MSPS) implemented in BESA (Brain Electrical Source Analysis) version 5.3.

Yellow areas indicate where additional sources would explain significant additional variance of the signal. These areas are only located around the equivalent dipoles employed in our model. This indicates that there was no systematic unexplained variance left which would have required the introduction of another dipole into the model.

Results: dipole model 400-600ms



The dipole model fitted on the broader time window (400-600ms) is located lower in the temporal lobe. Area 400-600ms was exported for further analysis. No significant differences between ASD and typically developing controls were observable ($F_{1,35}=1.99$; $p=0.17$). Explained variance was somewhat lower for the ASD group compared to typically developing controls ($F_{1,35}=3.77$; $p=0.06$).

Table: Means and standard deviations for the dipole activation and explained variance (dipole orientations were refit on single subject averages)

	ASD	TYP
left dipole nAm (\pm SD)	11.89 (\pm 16.41)	24.06 (\pm 23.58)
right dipole nAm (\pm SD)	26.99 (\pm 28.90)	32.11 (\pm 24.00)
explained variance (\pm SD)	56.18% (\pm 16.60)	64.55% (\pm 9.41)