Table 1. Detection of abnormality of fetal growth or amniotic fluid

	USE Every 2 weeks (N=112)	USE Every 4 weeks (N=116)	RR (95% CI)	P
Primary outcome	46 (41)	44 (38)	1.11 (0.78-1.60)	0.68
FGR	21 (19)	16 (14)	1.40 (0.74-2.67)	0.43
LGA--Estimate	17 (15)	15 (13)	1.19 (0.59-2.4)	0.68
Oligohydramnios	4 (4)	6 (5)	0.83 (0.22-3.0)	0.68
Polyhydramnios	4 (4)	7 (6)	0.69 (0.2-2.4)	0.68
CMM	29 (26)	26 (22)	1.22 (0.73-2.02)	0.37
CNM	16 (14)	14 (12)	1.2 (0.58-2.5)	0.68

Data presented as N (%)
USE, ultrasound examination; RR, relative risk; CI, confidence interval
FGR, fetal growth restriction (estimated fetal weight < 10th percentile for gestational age)
LGA, large for gestational age (estimated fetal weight > 90th percentile for gestational age)
Oligohydramnios (AFI ≤ 5.0 cm or MVP ≤ 2.0 cm); Polyhydramnios (AFI ≥ 24.0 or MVP ≥ 8.0 cm)
CMM, composite maternal morbidity: Chorioamnionitis, diabetic ketoacidosis, transfusion, wound infection, venous thromboembolism, admission to intensive care unit or death
CNM, composite neonatal morbidity: Apgar score < 5 at 5 min, umbilical arterial pH < 7.00, hyperbilirubinemia, intraventricular hemorrhage grade III or IV, periventricular leukomalacia, intubation for over 24 hours, necrotizing enterocolitis grade 2 or 3, stillbirth or death within 28 days of birth

Table 2. Detection of abnormal fetal growth

	Sensitivity	Specificity	PPV	NPV	LR (+)	LR (-)
Small for gestational age						
USE Every 2 weeks	86% (72-94%)	88% (78-94%)	83% (68-92%)	91% (81-96%)	7.3 (3.8-14.2)	0.2 (0.1-0.3)
USE Every 4 weeks	82% (67-91%)	90% (80-96%)	84% (69-93%)	89 % (79-95%)	8.3 (4.1-17.1)	0.2 (0.1-0.4)
Large for gestational age						
USE Every 2 weeks	32% (19-48%)	94% (85-98%)	76% (50-92%)	71% (60-79%)	5.6 (1.9-16.1)	0.7 (0.6-0.9)
USE Every 4 weeks	36% (22-53%)	96% (88-99%)	82% (56-95%)	75% (65-83%)	9.2 (2.8-30.2)	0.7 (0.5-0.8)

PPV, positive predictive value; NPV, negative predictive value; LR, likelihood ratio; USE, ultrasound examination

4 Reducing time to treatment for severe maternal hypertension through statewide quality improvement

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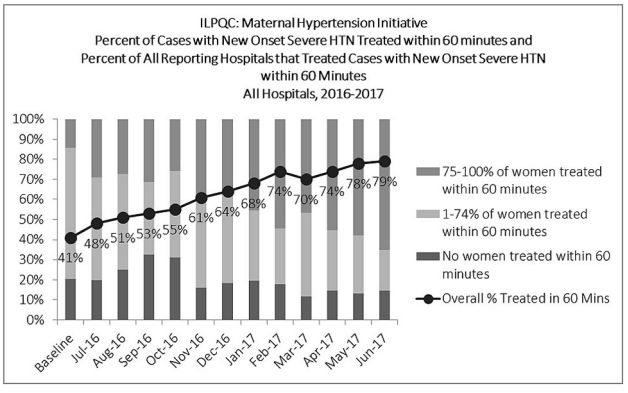
OBJECTIVE: The Illinois Perinatal Quality Collaborative (ILPQC) launched a Severe Maternal Hypertension (HTN) quality improvement (QI) initiative in May 2016 aiming to reduce maternal morbidity associated with HTN in 110 participating hospitals by reducing time to treatment of HTN and standardizing patient education and follow up at discharge. The objective of this analysis is to assess the improvement in these key process measures associated with HTN identification and treatment in the first full year of the initiative through June 2017.

STUDY DESIGN: Participating hospitals recorded data on key process measures for all cases of new onset severe HTN (>160 systolic or >110 diastolic) in pregnancy to 6 weeks postpartum in the ILPQC Data System monthly. ILPQC facilitated collaborative learning opportunities, rapid-response data, and quality improvement support. Baseline data on key process measures from prior to the initiation of the QI initiative (Oct.-Dec. 2015) were compared to data one year into the initiative (June 2017).

RESULTS: 102 hospitals entered any data and an average of 79 hospitals entered data each month with a total of 9818 cases of severe maternal HTN reported. Hospital characteristics are available for 98 hospitals (Tab.). The percentage of new onset severe HTN cases treated within 60 minutes increased from 41.5% (baseline) to 78.9% (June 2017) (Fig.). The percentage of hospitals with 75-100% of women treated within 60 minutes increased from 14% to 65% (Fig.). The percentage of cases: receiving preeclampsia education at discharge increased from 37% to 81%; scheduling follow up appointments within 10 days of discharge increased from 53% to 75%; with debrief after event increased from 2% to 44%.

CONCLUSION: Women with severe HTN are often not treated within the recommended 60 minutes. Results suggest that a statewide QI effort, including collaborative learning, rapid response data and QI support, can reduce time to treatment of severe HTN with antihypertensives, increase provider-nurse debriefs and patient education and follow-up appointments at discharge across IL hospitals serving diverse populations in diverse settings.

Hospital Characteristic	Category	% of Hospitals Participating in ILPQC HTN QI (n=98)
Urbanization	Urban	77%
	Rural	23%
Birth Volume	Low (<500)	25%
	Low to Moderate (500 to <1000)	20%
	Moderate to High (≥1000 to <2000)	33%
	High (≥2000)	22%
Non-Hispanic White	Low to Moderate (<60%)	43%
	High (≥60%)	57%
Medicaid Payment	Low to Moderate (<60%)	69%
	High (≥60%)	31%
WIC Recipients	Low to Moderate (<60%)	77%
	High (≥60%)	23%



5 Valnoctamide rescues CMV-induced deafness in a murine model

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OBJECTIVE: Congenital cytomegalovirus (CMV) is the leading infectious cause of non-hereditary sensorineural hearing loss in newborns and children. No treatments are currently recommended for

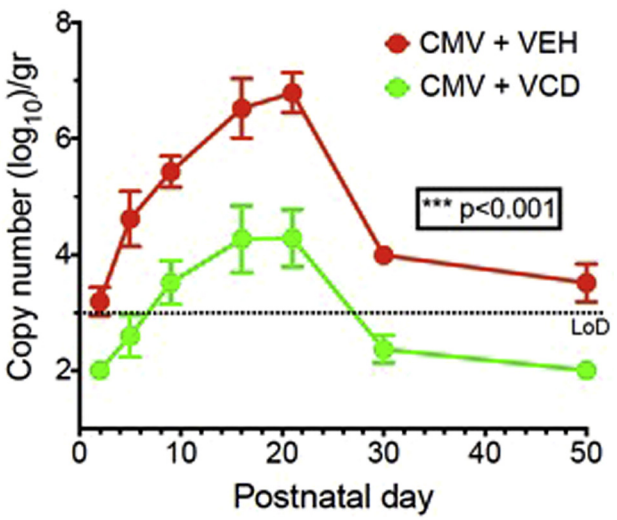
infected fetuses and postnatal therapy is only available for the most severe cases of infection due to substantial safety concerns. We showed that valnoctamide (VCD), a mood stabilizer, effectively blocks CMV. Here we investigate CMV infection in the developing auditory system of newborn mice and the potential benefits of VCD on long-term hearing outcomes.

STUDY DESIGN: Pups inoculated i.p. with CMV (750 PFU) on the day of birth received VCD (n=8) or vehicle (VEH, n=8) daily (1.4mg/mL) from p1 to p21. Brain development of newborn mice parallels that of an early 2nd trimester human fetus. Uninfected animals served as controls (n=8). CMV load and distribution in the cochlea and central auditory regions were assessed by qPCR and histochemistry at multiple time-points post-infection. Hearing was investigated blindly with respect to the experimental group using Auditory Brainstem Responses in 7 week-old mice. Statistical significance was determined by mixed-model ANOVA with repeated measures.

RESULTS: CMV was detected in the cochlea as early as 2 dpi, with viral load peaking at p16-21 and viral particles still measurable at p50. CMV-infected cells were identified in several areas of the inner ear, including the stria vascularis, the temporal bone, and the cochlea, where selective loss of outer hair cells was evident by p12. CMV+ cells were also recognized in central components of the auditory system, such as cochlear nuclei, inferior colliculus, and auditory cortex. Infected mice showed increased hearing thresholds at multiple frequency tone stimuli. VCD substantially reduced CMV load in the cochlea (Fig. 1) and the brain, and ameliorated hearing development with restoration of normal auditory responses (Fig. 2).

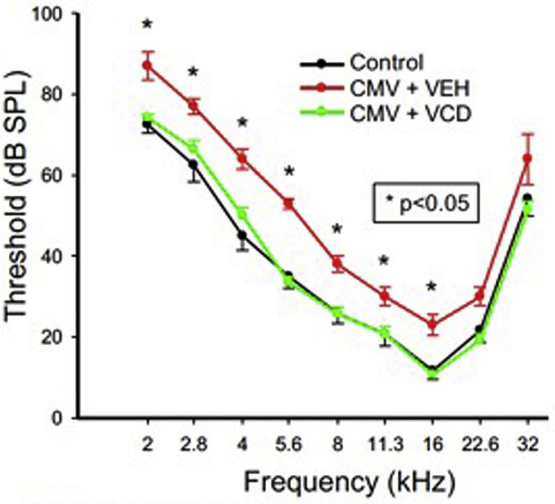
CONCLUSION: VCD effectively blocks CMV infection in the developing auditory system and rescues virally induced hearing impairment. VCD is approved for treatment of neuropsychiatric disorders and lacks teratogenic activity. Thus, it may merit consideration as a novel approach in treating CMV-mediated deafness during development.

Fig. 1. VCD suppresses CMV load in the cochlea.



LoD, limit of detection.

Fig. 2. Auditory responses in 7 week-old mice.



dB SPL, decibel sound pressure level.

6 The impact of the HYPITAT I trial on obstetric management and outcome for gestational hypertension and preeclampsia in the Netherlands

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OBJECTIVE: In 2009, the HYPITAT I study showed that in women with pregnancy induced hypertension or preeclampsia at term, induction of labor reduces maternal morbidity compared with expectant management, without compromising neonatal outcome or cesarean section rate. We aimed to evaluate the impact of the HYPITAT I trial results on obstetric management and subsequent maternal and neonatal outcomes in the Netherlands, five years after the trial, as compared to before the trial.

STUDY DESIGN: We studied aggregated data from the Dutch National Perinatal Registry from 2000 to 2014. We studied women with hypertension in pregnancy or preeclampsia and a singleton fetus in cephalic position between 36 to 41 weeks' gestation. Outcome measures were induction of labor, mode of delivery and the occurrence of maternal and neonatal complications. We compared the proportions of the period before the trial (2000-2005) to the period after the trial (2008-2014). We also compared outcomes to a reference group of women without hypertensive disorders in pregnancy.

RESULTS: We evaluated data of 55 780 women before to 70 890 after the trial (Table 1). Induction of labor increased from 51.1% to 64.2% (RR 1.26 (95 %CI 1.24-1.27)). This increased rate was more pronounced in HYPITAT I participating hospitals compared to non-participating hospitals (participating: 48.5% to 64.5% (RR 1.33; 95% CI 1.31-1.35); non-participating: 53.4% to 63.9% (RR 1.20; 95% CI 1.18-1.21)). A reduction was observed in the instrumental delivery rate after the trial compared to the period before the trial (16.5% to 13.1%; RR 0.79; 95% (CI 0.77-0.81)). Spontaneous delivery rates