in the 26 Type A was not more increased than in the 12 Type B men for the cardiovascular variables (heart rate, pulse transit time, finger vasoconstriction and blood pressure), but for body movement, EMG, respiration frequency. Body movement and EMG were the best predictable physiological variables for the Type A behavior pattern, and this mainly in the two speech tests.

Résumé

Réactivité psychophysiologique et sa relation avec le style de comportement Type A

Cette relation était examiné pour 77 hommes sains. La comparaison entre 26 hommes de Type A et 12 hommes de Type B n'a pas montré de réactivité élevée pour les Type A envers le stress en ce qui concerne les variables cardiovasculaires (fréquence cardiaque, le temps de transmission du pouls, vasoconstriction du doigt et pression sanguine) mais elle en a montré pour la motorique corporelle, l'EMG et la respiration. La motorique corporelle et l'EMG prédisaient mieux le style de comportement Typ A, particulièrement dans les deux tests de conversation.

Literatur

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3D Display of Stillbirth in Indonesian Obstetrics Part 7: Expansion to Neonatal Death

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Introduction

In six previous reports in this journal (1), the risk of stillbirth in Indonesian university obstetrics for 36,802 singleton-birth deliveries from 1978 to 1980 was selectively controlled for 9 factors (birth weight, complications, morbidity, registration, antenatal visits, contraceptive use, parity, residence and infant sex) and displayed accessibly for medical, paramedical and health research personnel. This report broadens into the study of another endpoint, namely hospital neonatal death (ND) and its relations with stillbirth (SB) by imposing additionally two bivariate control systems: birth weight/infant sex and maternal age/selected obstetrical complications.

Material and Method

The material and method remains unchanged. However, for immediate comparison, the risks of neonatal death (ND) are given along with the risks of stillbirth (SB) and their relationships expressed as 'percent perinatal death occurring neonatally'. The structured display of statistics is given in *Fig. 20* for the control system birth weight/infant sex, and in *Fig. 21* for the control system maternal age/selected obstetrical complications (none, prolonged/obstructed labor, placenta praevia).

A methodological expansion is to give not only rates or percent and cell size but also the number of events. In other words, figures may be both checked and regrouped, examined and tested according to later needs. Panels are thus becoming versatile 'reference baselines' of risks/prevalences identified by place, time, population and control system. Possible magnification of the figures leads thus to a ready-for-use work tool. They may constitute building blocks in epidemiological history of reproductive health.

Results

The risk of hospital neonatal death (ND) shows across birth weight a pattern quite similar to that of stillbirth (SB) – a reversed J-shaped profile *(Fig. 20.2)*. However, in all three low birth weight categories, an *excess ND risk for boys over girls* is apparent (1.29, 1.34, 1.12) with an aggregate excess ND risk of 31%. In addition, for male LBW-infants an *excess ND share of perinatal death* (PD) emerges for the three groups (1.15, 1.34, 1.11) with an aggregate excess ND share of 15% *(Fig. 20.3)*. For infants weighing 3000–3499 g, there is also excess ND risk and excess ND share of PD (2.24, 1.77). This points to greater male vulnerability in the early neonatal period for contrasting birth weights with likely different causes operating already in utero, latest during the birth process. It matters thus to control for specific obstetrical complications.

As shown in *Fig. 21.2* (left roof), the neonatal death risk (ND) is lowest for no complications (13.0/1000), around threefold for prolonged/obstructed labor (41.8/1000) and around ninefold for placenta previa (111.9/1000) with relative small variation across maternal age. One may hypothesize that placenta previa links mainly with low birth weight infants and prolonged/obstructed labor with higher birth weights – an implicit connection of the two figures.

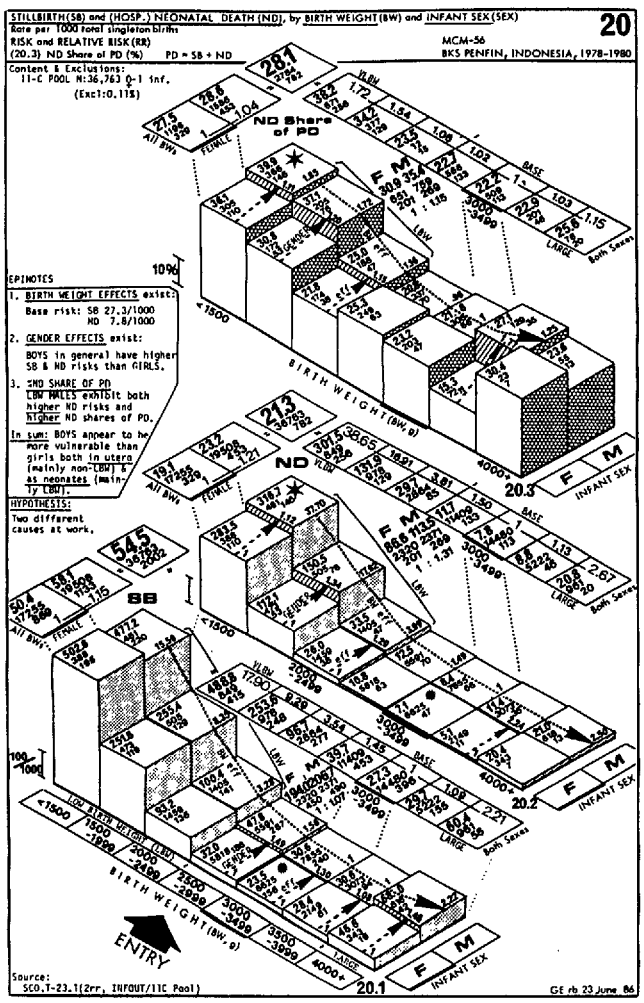

Fig. 20. Stillbirth, neonatal death and relationship, by birth weight and infant sex.

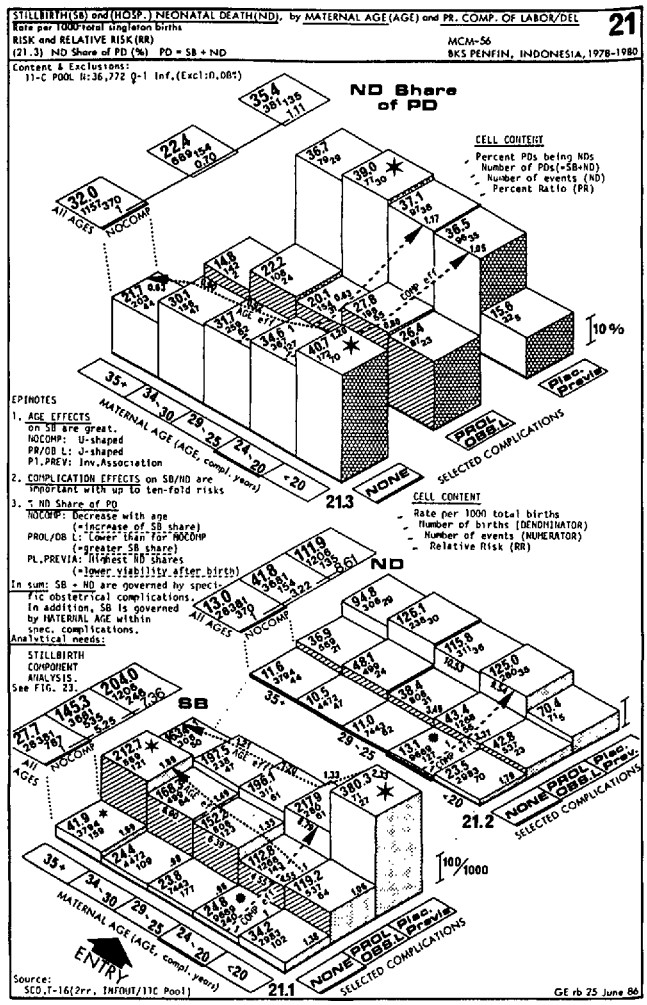

Fig. 21. Stillbirth, neonatal death and relationship, by maternal age and selected complications.

The postpartum 1:3:9 ND risk ratio, quite independent of age *(Fig. 21.2)*, stands in sharp contrast to the risk profiles of stillbirth (SB) with opposite patterns across age (Fig. 21.1). This observation orients the inquiry towards a component analysis of stillbirth to be carried out in part 8 of this series.

Discussion

The risk patterns of hospital neonatal death in the two control systems generate consistent information with the hightest ND risks being linked to low birth weight of male infants and placenta previa. The hypothesis is formulated that the three factors are linked. This could constitute one future focus in surveillance research with maternity care monitoring data from developing countries.

Summary and Outlook

Part 7 of the 'SB risk series' is a transition paper from stillbirth to neonatal death. The findings are internally consistent in two control systems which opens the way to systematic analysis of the neonatal component of 'hospital perinatal death'. Both at low and adequate birth weight, greater male vulnerability was noted in the neonatal hospital period. The risk of hospital neonatal death was 111.9/1000 for placenta previa, 41.8/1000 for prolonged/obstructed labor against a base risk of 13.0/1000 for women with no obstetrical complications. The stillbirth risk patterns in the two selected control systems are differentiated and merit thus disagregation by time of occurrence: the next analytical step (part 8).

Résumé et Perspective

Mortinatalité dans les hôpitaux indonésiens

7e partie: Extension à la mortalité néonatale

La 7e partie de l'analyse de la mortinatalité en Indonésie marque une transition vers l'étude de la mortalité néonatale. Les résultats révèlent une structure différenciée pour deux systèmes de contrôle, incitant ainsi une analyse plus systématique de la composante néonatale de la mortalité périnatale des hôpitaux. Pour des groupes de poids à la naissance différents une plus grande vulnérabilité des nouveau-nés vivants mâles fut observée pour la période néonatale. Le risque de mortalité néonatale dans les hôpitaux fut de 13,0/1000 pour les parturientes sans complications, de 41,8/1000 pour les accouchements au travail prolongé/arrêté, voire de 111,9/1000 pour les placentae praeviae. Pour deux systèmes de contrôle la constella-

tion des risques de mortinatalité s'avère très différenciée, d'où l'attrait de subdiviser ces risques selon le moment de leur incidence (8e partie).

Zusammenfassung und Ausblick
Totgeburten in Indonesischen Geburtskliniken
Teil 7: Ausweitung auf Neugeborenensterblichkeit
Teil 7 dieser Totgeburtenstudie in Indonesien ist ein Übergang zur Studie der Neugeborenensterblichkeit. Für zwei Kontrollsysteme weisen die Resultate stukturelle Konsistenz auf, was die systematische Analyse der neonatalen Komponente der perinatalen Sterblichkeit an den Geburtskliniken ermöglichen sollte. Für verschiedene Geburtsgewichte wurde eine grössere männliche Betroffenheit in der frühen neonatalen Zeitspanne festgestellt. Die Spitalrisiken des Neugeborenentodes waren im Vergleich zu 13,0/1000 für Frauen ohne Komplikationen 41,8/1000 für verzögerte/obstruktive Geburten und gar 111,9/1000 für Placenta Praevia. Die Risikostrukturen der Totgeburten für zwei Kontrollsysteme sind stark differenziert, was in der nächsten Analyse zur Unterteilung nach Zeit des Geschehens Anlass gibt (Teil 8).

Reference
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3D Display of Stillbirth in Indonesian Obstetrics Part 8: Component Analysis (intra-/antepartum)

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Introduction

This analysis is to keep the same two control systems used in the previous report [1] but to study the two components of stillbirth (SB) instead of the dichotomy of perinatal death (PD). Risks of intrapartum (IPD) and antepartum death (APD) and their relationships are thus controlled for birth weight/infant sex and maternal age/selected obstetrical complications.

Material and Method

The same 36,802 singleton-birth deliveries and method are used. The two panels give in the two control systems the risks of antepartum and intrapartum death and their relationships as 'percent stillbirth occurring during delivery' (% IPD share of SB). The structured display of statistics is given in *Fig. 22* for the control system birth weight/infant sex, and in *Fig. 23* for the control system maternal age/selected complications (none, prolonged/obstructed labor, placenta previa).

Results

As developed in *Fig. 22.2*, intrapartum death (IPD) shows a risk pattern across birth weight quite similar to the pattern of antepartum death (APD) – a reversed J-shaped profile, though much less accentuated at the lower BW side. Indeed, among LBW-infants, the risk of antepartum death is 136.4/1000 *(Fig. 22.1)* against only 64.0/1000 for intrapartum death *(Fig. 22.2)*. By contrast, for infants weighing 3000 + grams, the risk structure is reversed with a greater IPD risk (18.0/1000) than APD risk (11.5/1000). Likely, the two BW ranges are linked with different causes of death: at low birth weight the insult may start quite early in utero with an increased risk of APD, whereas for higher birth weights the insult may start predominantly during the birth process with an increased risk of IPD over APD.

Fig. 22.3 gives the IPD share of SB. As shown on the right roof, the indicator increases steeply with birth weight from 24.1% for VLBW-infants to 65.5% for large infants. The death among stillbirths occurring intrapartum is a gradual process directly related with birth weight, that is infant size. Problems of mechanical passage appear to be one main cause of intrapartum death.

As shown in *Fig. 23.2*, intrapartum death risk (IPD) is governed by both maternal age and complication. For women with no obstetrical complication, the IPD risk is 6.0/1000. Note the 54% excess risk for teenagers. For women with prolonged/obstructed labor, the IPD risk soars to 102.7/1000, with an important positive age effect that doubles the risk from 78.1/1000 in the early twenties to 147.6/1000 for ages 35 + years. For women with placenta previa, the IPD risk is 107.0/1000, but the risk relates inversely with maternal age, lowest for 35 + years with 75.2/1000 and threefold for teenagers with 225.4/1000. This *opposite age relationship of IPD for two contrasting pathologies* merits inquisitive analysis. We suggest IPD for placenta previa to be studied for a possible relationship with 'BW for date'. In the low twenties, the IPD ratio chain for NOCOMP – PROL/OBST LABOR – PL PREVIA is 1:12.8:21.1. By contrast, the corresponding chain for antepartum death is 'only' 1:1.86:4.78 as shown in *Fig. 23.1*.