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Screening for developmental dysplasia of the hip

Magda M. Boere-Boonekamp[1] and Paul H. Verkerk[2]

[1]*Centre for Health Care Research, University Twente, PO Box 217, 7500 AE Enschede, The Netherlands*
[2]*TNO Prevention and Health, PO Box 2215, 2301 CE Leiden, The Netherlands*

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The success rates of screening programmes for Developmental Dysplasia of the Hip (DDH) vary widely. Studies on screening programmes for DDH based on a Medline search for the years 1966–1997 are reviewed. The percentage treated in most studies, especially those using ultrasound, are high and suggest substantial over-treatment. Neonatal clinical screening has the best results, but programme effectiveness increases when this is combined with secondary screening of the high-risk population. The extra costs are compensated by reduced treatment costs of late-diagnosed cases.

Introduction

Early diagnosis of developmental dysplasia of the hip (DDH) has many advantages. Treatment is shorter and less invasive and results are better [1–6]. After the first year the chances of successful treatment diminish considerably. This matters because subluxation and dislocation, as well as acetabular dysplasia without dislocation, are frequent causes of osteoarthritis in later adult life [7, 8].

Since hips may dislocate at variable times after birth, the old term 'congenital dislocation of the hip' has been replaced by 'developmental dysplasia of the hip'. The new nomenclature indicates a dynamic disorder which may get better or worse as the infant develops. The term DDH includes the following conditions diagnosable during the first months of life: dislocated hip, dislocatable hip, subluxated hip and hip dysplasia [9–12].

The scope for primary prevention of DDH is limited, although gentle handling after delivery, the avoidance of traction or suspension on the lower limbs and the maintenance of a favourable hip position by correct diapering may promote normal hip development [12]. Roser proposed secondary prevention by early detection in the late nineteenth century [12] and in 1935 Ortolani described a springing and jerking sensation that can be felt when a dislocated hip is abducted [13]. In 1962 Barlow applied direct anterior thumb pressure on the femoral head to detect so-called 'dislocatable' hips. Since the early sixties newborn screening programmes have been set up all over the world. Repeated physical examination during the first year has been advocated to detect 'missed' DDH. Radiographic screening has been abandoned because of radiation exposure, but ultrasonographic screening was introduced in the last decade [14–18]. In this paper we review the results of screening programmes for DDH since 1960.

Methods

A Medline search for the years 1966–1997 was extended by cascade retrieval of references in identified publications. The following MESH (MEdical Subject Headings) terms were used: (1) congenital hip dislocation/pc; (2) congenital hip dislocation; (3) mass screening; (4) reproducibility of results, clinical trials, programme evaluation, evaluation studies; (5) cost–benefit analysis; (6) infant, newborn. We selected all studies with the

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term 1, terms (2 and 3), and terms (2 and 4) as well as the terms (1 or (2 and 3)) and 5. The search was further restricted with term 6 (infant, newborn) and to studies written in the English, German, Dutch or French language. Studies without a detailed description of the screening programme, of less than 500 children, or which did not report the percentages of screen-positives, or of treated children were excluded.

Fifty-nine screening programmes (described in 54 publications) were identified and included in the review. They were categorized on the basis of timing (neonatal versus infant screening), method (physical examination versus ultrasound), and on the number of screening occasions (solitary versus repeated screening) into seven categories:

1 Neonatal screening by physical examination,
2 Neonatal screening by ultrasound,
3 Neonatal screening by physical examination and ultrasound,
4 Infant screening by physical examination,
5 Neonatal and infant screening by physical examination,
6 Neonatal screening by physical examination plus secondary screening of the high-risk population,
7 Selective screening by physical examination and ultrasound only.

Results

The only randomized controlled trial identified compared neonatal clinical screening, neonatal ultrasound, and a combination of clinical screening with selective ultrasound of high-risk cases [41]. All other studies evaluated only one screening procedure.

The results are presented in Table 1. The screen-positive percentage includes, when applicable, identified high-risk children, and the treated rate applies to each study's treatment criteria. Follow-up for cases 'missed at screening' was usually by review of hospital records. Only a few studies included follow-up physical examination, X-ray or ultrasound. The length of follow-up and the percentages of 'missed dislocations' are given when applicable. Finally, the percentages of treated cases in whom surgery was performed and in whom treatment was complicated by avascular necrosis are shown.

Summary results for each screening category are shown in Table 2: the median and range of percentages of screen-positive children; of treated cases; and of dislocations 'missed at screening'. The percentages of affected hips in ultrasound studies are assumed to represent only bilaterally affected children so that the figures in screening categories 2 and 3 are a minimum estimate. The percentage of screen-positive children is lowest among neonates screened by physical examination (category 1). The percentage of treated children is also low in this category, but equally low in neonatal clinical examination programmes with secondary universal clinical screening in infancy (category 5) or with a secondary screening by ultrasound or X-ray of the high-risk population (category 6). The percentage of dislocations 'missed at screening' varies from 0–0.73%. The highest percentage of 0.73% was found in a study with individual follow-up of all children with a negative screening result [32].

The percentage of treated children needing surgery (usually adductor tenotomy, at times open reduction) despite detection by screening varied from 0–7.2% in 25 studies. The frequency of avascular necrosis of the femoral head varied from 0–1% in 17 studies, although three studies reported values of 1.9%, 3.3% and 3.6%.

Discussion

Success of screening programmes for DDH

The different screening programmes show striking differences in the percentages of treated and missed cases. Neonatal clinical examination programmes (category 1) and programmes that combine neonatal clinical examination with a secondary screen of the high-risk population (category 6) appear the best.

Without screening the incidence of established hip dislocation is 0.07–0.22% [1, 70], although the incidence of dysplasia is less certain. It has been claimed that 30–70% of patients undergoing hip replacement in later life, show evidence of acetabular dysplasia. Assuming that 1% of adults eventually need joint replacement, this translates to 0.3–0.7% rate of hip dysplasia. If so, acetabular dysplasia (without dislocation) is at least as common as hip dislocation [71, 72] and 0.4–0.9% of children with DDH would require treatment. Since treatment percentages in DDH screening programmes are often much higher (0.2–12.6%), either

Table 1. Details and results of 59 screening programmes for DDH

Cat.	Author (Year)	[Ref]	Country (Period)	Population (n)	Screening		Screen-positive (%)	Treatment criterion	Cases treated (%)	Operated (%)	Follow up	Missed (%) all (disl)	Avasc necr. (%)
					Method	Age							
1	Barlow (1962)	[19]	UK (1957–1961)	9289	Phys. ex.	<1 wk	1.7	Pos. Barlow	1.7	—	Clin. 1 yr	None	—
1	Fredensborg (1976)	[20]	Sweden (1956–1972)	58,759	Phys. ex.	<4 days	0.9	Abn. phys. ex.	0.9	—	—	0.01 (0.01)	None
1	Cyvin (1977)	[21]	Norway (1969–1974)	19,864	Phys. ex.	Day 2– and day 4–6	1.9	Instability	1.9	—	h.r.	0.22 (0.09)	—
1	Jones (1977)	[22]	UK (1968–1972)	29,266	Phys. ex., recheck abn. cases	Neonate	0.3	Abn. phys. ex.	0.3	5.3	h.r.	0.06 (0.06)	—
1	Galasko (1980)	[23]	UK (1975–1980)	11,980	Phys. ex., recheck abn. cases	—	1.5	—	0.2	None	h.r.	0.08 (0.08)	—
1	Mendes (1980)	[24]	Israel 1976–1979	8439	Phys. ex.	<3 days	1.4	Pos. Ort/Barlow	1.4	None	h.r.	0.08 (0.08)	None
1	Lehmann (i) (1981)	[25]	Canada (1967–1971)	7189	Phys. ex. by non-exp. screeners	Neonate	0.5	Abn. phys. ex.	0.5	—	h.r.	0.08 (0.08)	—
1	Lehmann (ii) (1981)	[25]	Canada (1967–1971)	16,045	Phys. ex. by exp. screeners	Neonate	0.6	Abn. phys. ex.	0.6	—	h.r.	0.03 (0.03)	—
1	Tredwell (1981)	[26]	Canada (1967–1976)	32,480	Phys. ex.	<5 days	1.0	Pos. Barlow	1.0	1.6	h.r.	0.02 (0.003)	—
1	Heikkilä (1984)	[27]	Finland (1966–1975)	151,924	Phys. ex.	Neonate	0.6	—	0.6	—	h.r.	0.08 (0.08)	3.3
1	Palmén (1984)	[1]	Sweden (1950–1974)	28,000	Phys. ex.	<4 days	0.6	Instability	0.6	—	—	0–0.2 (0–0.2)	—
1	Dunn (1985)	[5]	UK (1970–1979)	23,002	Phys. ex., recheck abn. cases	Day 1 day 2–10	1.9	Persistent abn. phys. ex.	1.9	—	h.r.	0.04 (0.04)	None
1	Rao (1986)	[28]	New Zealand (1973–1982)	13.841	Phys. ex., recheck abn. cases	<5 days day 7–10	1.8	Persistent abn. phys. ex.	0.4	—	h.r.	0.01 (0.01)	—
1	Hadlow (1988)	[29]	New Zealand (1964–1985)	20,657	Phys. ex., recheck abn. cases	<7 days	3.2	Persistent abn. phys. ex.	1.6	0.9	h.r.	0.01 (0.01)	0.3
1	Miranda (1988)	[30]	Spain (1980–1984)	49,937	Phys. ex.	Day 1	0.6	Instability	0.6	0.03	h.r.	0.12 (0.12)	0.06
1	Macnicol (1990)	[31]	UK (1962–1986)	117,256	Phys. ex.	Neonate	0.6	Instability, confirmed (X-ray, orth)	0.6	—	clin. 10 mo	0.05 (0.05)	1.0
1	Myles (i) (1990)	[32]	UK —	3205	Phys. ex., recheck abn. cases	Neonate	4.2	Orth. diagnosis	4.2	—	Clin. 1 yr	0.22 (0.22)	—
1	Tönnis (i) (1990)	[33]	Germany (1982–1983)	1301	Phys. ex.	<3 days	2.2	Instability	2.2	—	—	—	—
1	Yngve (1990)	[34]	USA (1976–1988)	26,455	Phys. ex.	<4 days	0.4	Pos. Ort/Barlow	0.4	—	h.r.	0.02 (0.02)	—
1	Sanfridson (1991)	[35]	Sweden (1980–1987)	19,398	Phys. ex.	<4 days	1.9	—	1.9	—	—	0.06 (0.06)	—
1	Krikler (1992)	[36]	UK (1980–1990)	37,511	Phys. ex.	Neonate	1.2	Abn. phys. ex.	1.2	3.63	h.r.	0.003 (0.003)	None

Table 1. *Continued*

Cat.	Author (Year)	[Ref]	Country (Period)	Population (*n*)	Screening		Screen-positive (%)	Treatment criterion	Cases treated (%)	Operated (%)	Follow up	Missed (%) all (disl)	Avasc necr. (%)
					Method	Age							
1	Poul (1992)	[37]	Czech/Slov (1984–1989)	35,550	Phys. ex.	<7 days	1.8	Instability	1.8	0.3	X-ray 3 mo	0.98 (0.06)	0.3
1	Tredwell (1992)	[38]	Canada (1981–1986)	5900	Phys. ex.	<3 days	1.1	Pos. Barlow	1.1	1.6	h.r.	None	None
1	Bjerkreim (1993)	[39]	Norway (1980–1989)	141,893	Phys. ex.	Neonate	1.1	—	1.1	—	Clin.	0.23 (0.23)	—
1	Fiddian (1994)	[40]	UK (1982–1992)	42,421	Phys. ex.	<2 days	0.6	Abn. phys. ex.	0.6	2.4	h.r.	0.03 (0.03)	None
1	Rosendahl (i) (1994)	[41]	Norway (1988–1990)	3924	Phys. ex.	<2 days	1.8	Persistent abn. phys. ex.	1.8	—	h.r.	0.26 (0.13)	—
2	Ganger (1991)	[42]	Austria (1986–1987)	1291	US+ recheck type II–IV	Day 3 6 wk, 12 wk	9.2	US IIc–IV	9.2	None	h.r.	None	None
2	Leonhardi (1993)	[43]	Germany (1985–1987)	3396	US+ selective US	Day 4 6 wk	9.7	US IIg–IV	4.2*	—	—	—	—
2	Stöver (1993)	[44]	Germany —	726	US	Day 10	7.6	—	3.9	—	US—10 wk	0.4 (—)	—
2	Deimel (1994)	[45]	Germany (1985–1990)	2317	US	Neonate	28.7*	US IID–IV	12.6*	None	—	—	—
2	Rosendahl (ii) (1994)	[41]	Norway (1988–1990)	3613	US+FU of IIa hips	<2 days	16.4	US IIc–d+ unstable/US IIIa–IV	3.4	—	—	0.14 (0.03)	—
3	Berman (1986)	[46]	Canada (1985–1986)	1001	Phys. ex.+US+ID of high risk pop.	<1–2 days	6.9	US IIIa–IV	0.5	—	h.r.	None	—
3	Dorn (i) (1990)	[47]	Austria (1984–1988)	8221	Phys. ex.+US+FU of IIa hips	<5 days	27.5*	US IIc–IV/IIa– at 6 wk	4.8*	—	US sample	—	—
3	Hauck (1990)	[48]	Germany (1985–1988)	1500	Phys. ex.+US	<5 days	4.1	Unstable/US IIg–IV	4.1	—	—	—	—
3	Tönnis (ii) (1990)	[33]	Germany (1985–1987)	2578	Phys. ex.+US	<3 days	32.6*	US IIc–IV	4.4	—	h.r.	0.08 (0.08)	—
3	De Pellegrin (1991)	[49]	Germany (1988–1989)	1000	Phs. ex.+US+FU of IIa hips	<5 days 4–6 wk	25.4*	US IIc–IV/IIa– at 4–6 wk	3.3*	None	—	—	—
3	Marks (1994)	[50]	UK (1989–1992)	14,050	Phys. ex.+US+FU of abn. US hips	<7 days	6.0	Persistent abn. US ex.	0.2	8.8	h.r.	None	None
4	Hees-v. d. Laan (1981)	[51]	Netherlands (1976)	1059	5 × phs. ex.+ID of high-risk pop.	3 wk–1 yr	23.7	Orth. diagnosis	3.9	—	h.r.	—	—
4	Hees-v. d. Laan (1985)	[52]	Netherlands (1984)	2467	5 × phs. ex.+ID of high-risk pop.	3 wk–1 yr	19.1	Orth. diagnosis	2.7	—	h.r.	—	—
4	Nijhuis (1987)	[53]	Netherlands (1985–1986)	600	5 × phys. ex.	2 wk–1 yr	7.3	Orth. diagnosis	3.0	—	—	—	—
4	Bower (1989)	[54]	Australia (1981–1983)	66,640	Phys. ex. (247 neon. cases excl.)	2 wk	1.4#	Orth. diagnosis	0.2#	7.2	h.r.	0.05 (0.05)	—
4	Pauw-Plomp (1994)	[55]	Netherlands (1989–1990)	929	4 × phys. ex.+ID of high-risk pop.	1–12 mo	6.0	Orth. diagnosis	0.9	—	—	—	—

Table 1. *Continued*

Cat.	Author (Year)	[Ref]	Country (Period)	Population (*n*)	Screening: Method	Screening: Age	Screen-positive (%)	Treatment criterion	Cases treated (%)	Operated (%)	Follow up	Missed (%) all (disl)	Avasc necr. (%)
4	Boere-Boonekamp (1996)	[56]	Netherlands (1992–1993)	1968	2–4 × phys. ex.+ID of high-risk pop.	1, 3, 4, 6 mo	20.1	Orth. diagnosis+ abn. X-ray/US	3.2	None	US 6 mo	0.5 (0.1)	None
5	Myles (ii) (1990)	[32]	UK —	5456	Ort/Barlow+ phys. ex. CHC	Neonate 3 mo	6.4	Orth. diagnosis	1.4	—	Clin 1 yr	0.73 (0.73)	—
5	Darmonov (1996)	[57]	Bulgary (1985–1990)	20,417	Ort/Barlow+ phys. ex. CHC	<3 days 2–3 mo	0.6	Abn. phys. ex.+ abn. X-ray	0.6	0.8	h.r.	None	0.8
6	Monk (1980)	[58]	UK (1973–1977)	25,263	Phys. ex.+clin. FU of high-risk pop.	<1 day monthly	6.1	—	0.8*	—	—	0.01* (0.008)	None
6	Bernard (1987)	[59]	UK (1977–1983)	21,004	Phys. ex.+X-ray of high-risk pop.	<3 days 3–6 mo	18.1	Abn. phys. ex./ abn. X-ray	1.0	0.12	h.r.	0.005 (0.005)	—
6	Clarke (1989)	[60]	UK (1986)	4617	Phys. ex.+clin. FU/US high-risk pop.	Neonate <2 wk, 6 wk	9.7	Abn. phys. ex.+abn. US exam	0.4	None	h.r.	0.06 (0.06)	—
6	Jones (1989)	[61]	UK (1985–1986)	3289	Phys. ex.+clin. FU of high-risk pop.	<1 day 6 wk–1.5 yr	13.0	Persistent abn. phys. ex.+X-ray	1.1	2.9	—	None	—
6	Burger (1990)	[62]	Netherlands (1971–1979)	14,264	Phys. ex.+X-ray of high-risk pop.	Neonate 5 mo	6.7	Pos. Barlow/abn. X-ray	1.8	1.9	X-ray 24 mo	2.0 (0.02)	1.9
6	Dorn (ii) (1990)	[47]	Austria (1978–1984)	14,695	Phys. ex.+clin. FU/X-ray of abn. cases	<4 days 4 mo	21.9	—	2.7	—	—	—	—
6	Jones (1990)	[63]	UK (1987)	3879	Phys. ex.+US of high-risk pop.	Neonate	10.5	US IIIa–IV	1.1	—	Clin.	None	None
6	Garvey (1992)	[64]	Ireland (1986–1988)	13,662	Phys. ex.+X-ray of high-risk pop.	Neonate 4 mo	3.7	Instability/abn. X-ray	1.1	1.3	h.r.	None	—
6	Walter (1992)	[65]	USA (1987–1988)	1772	Phys. ex.+US of high-risk pop.	Neonate <3 mo	5.7	Abn. phys. ex./ mild or severe US dyspl	0.5	None	h.r.	None	—
6	Boeree (1994)	[66]	UK (1988–1992)	26,952	2 × phys. ex.+US of high-risk pop.	<1 day, 6 wk 2–6 wk	7.0	US (sub)lux./ pers. minor dysplasia	0.4	4.2	h.r.	0.22 (0.22)	None
6	Holen (1994)	[67]	Norway (1988–1990)	4450	Phys. ex.+clin. FU/US high-risk pop.	<4 days 2–5 mo	12.3	Abn. phys.+US ex./US +X-ray abn.	1.5	—	h.r.	0.02 (—)	—
6	Larchet (1994)	[68]	France (1987–1991)	5621	Phys. ex.+US of high-risk pop.	day 1+5 3–4 wk	12.0	—	1.9	1.9	h.r.	0.04 (0.04)	—
6	Rosendahl (iii) (1994)	[41]	Norway (1988–1990)	4388	Phys. ex.+US high-risk pop., FU IIa	<2 days	3.8	Abn. phys. ex./ IIc–d unstable/IIIa–IV	2.0	—	h.r.	0.21 (0.07)	—
7	Rosendahl (1992)	[69]	Norway (1987)	3457	Phys.+US exam. girls and high-risk boys	2 days	9.4	Abn. phys. ex./ US dysplasia	2.7	—	h.r.	0.09 (0.03)	—

The programmes are grouped into seven screening categories: (1) neonatal screening by physical examination; (2) neonatal screening by ultrasound; (3) neonatal screening by physical examination and ultrasound; (4) infant screening by physical examination; (5) neonatal and infant screening by physical examination; (6) neonatal screening by physical examination plus secondary screening of the high-risk population; (7) screening of only a selected part of the population by physical examination and ultrasound.

The percentages of screen-positive children, treated children and DDH cases (all, or only dislocations) refer to the screened population; the percentages of operated children and of those complicated by avascular necrosis refer to the total number of treated children.

Cat.=screening category (see text); Avasc necr.=avascular necrosis of the femoral head; —=unknown; Phys. ex.=physical examination; exp.=experienced; abn. cases=abnormal cases; US=ultrasound; FU=follow-up; ID=identification; neon. cases excl.=neonatal cases excluded; Ort=Ortolani; pop.=population; wk=week(s); mo=month(s); yr=year(s); pos.=positive; orth=orthopaedic; clin.=clinical; h.r.=hospital records; disl=dislocations; *=hips; #=the 247 neonatal cases are excluded.

Table 2. Summary of the results of all screening programmes grouped per screening category

Category	Studies n	Screen-positives median (range) %	Treated median (range) %	Missed dislocations median (range) %
1	26	1.2 (0.3–4.2)	1.1 (0.2–4.2)	0.06 (0–0.23)
2	5	9.7 (7.6–28.7)	4.2 (3.4–12.6)	0.02 (0–0.03)
3	6	16.2 (4.1–32.6*)	3.7 (0.2–4.8*)	0.00 (0–0.08)
4	6	13.2 (1.4–23.7)	2.9 (0.2–3.9)	0.08 (0.05–0.1)
5	2	3.5 (0.6–6.4)	1.0 (0.6–1.4)	0.37 (0–0.73)
6	13	9.7 (3.7–21.9)	1.1 (0.4–2.7)	0.01 (0–0.22)
7	1	9.4	2.7	0.03

Categories were (1) neonatal screening by physical examination; (2) neonatal screening by ultrasound; (3) neonatal screening by physical examination and ultrasound; (4) infant screening by physical examination; (5) neonatal and infant screening by physical examination; (6) neonatal screening by physical examination plus secondary screening of the high-risk population; (7) screening of only a selected part of the population by physical examination and ultrasound.
The median value of the percentages of test-positive children, treated children and dislocations missed at screening, is calculated for each screening category. In calculating the median percentage of missed dislocations only those studies are included which give this specific information in their results section.
*hips.
Note: In calculating the median values, the percentages of affected hips (as reported in ultrasound studies) are taken into account as if they represent only bilaterally affected children.

the incidence of DDH has increased or there is substantial over-treatment. Variation in treatment percentages cannot all be explained by geographical differences. Varying diagnostic criteria, timing of screening, and screening methods, probably explain more.

The rate of missed dysplasia in neonatal clinical screening programmes (category 1) varies between zero and 0.98%, and of missed dysplasia with dislocation from zero to 0.23%. Either the examination techniques are inadequate, the examiners lack experience, or normal infants develop DDH after the neonatal period. Ultrasound screening aims to detect not only serious dysplasia, accompanied by subluxation or dislocation, but also those babies whose hips show an immature aspect in the ultrasound image. Such babies have an increased risk of deterioration to DDH, so monitoring them till they show normal development or deteriorate, permits early treatment and explains why rates of screen-positives in ultrasound programmes are high. Despite this, zero to 0.08% of DDH cases are still missed on ultrasound screening (category 2 and 3). Different methods of ultrasonography have been developed: Graf's morphological approach (Europe), Harcke's dynamic assessment (USA, UK) and the assessment based on measurements of the femoral head coverage or FHC (Norway). Although the additional use of ultrasound has reduced neonatal treatment for DDH in some studies, other studies report the opposite effect [46, 50]. High treatment rates probably reflect lack of knowledge about the significance and natural course of immaturity of the newborn hip, which remains to be clarified through large prospective studies [69].

Late presenting DDH cases have prompted neonatal programmes with secondary screening of infants at increased risk (category 6). Risk factors include a family history of DDH or early osteoarthritis of the hip, breech position in late pregnancy, and the presence of other deformities such as foot abnormalities, torticollis or plagiocephaly. The rate of screen-positives in these two-stage programmes (including the identified high-risk population, in need of follow-up) is rather high although treatment percentage rates of DDH cases 'missed at screening' are encouragingly low.

Screening in infancy by physical examination and identification of a high-risk population (category 4) is associated with a high rate of children submitted to additional imaging procedures (1.4–23.7%) and moderate frequencies of treated children (0.2–3.9%). Such programmes do not lead to early detection, with most cases diagnosed after three months. The two programmes that combined clinical neonatal screening with a second clinical screen in infancy (category 5) report contradictory results.

The widely varying rates of DDH cases 'missed at screening' despite similar screening procedures, can mostly be attributed to differing follow-up. If follow-up was based on review of only hospital records, many dysplasias without dislocation were

Table 3. Criteria for a successful screening programme (Wilson and Jungner 1968) [73]

1. The condition sought should be an important health problem.
2. There should be an accepted treatment for patients with recognized disease.
3. Facilities for diagnosis and treatment should be available.
4. There should be a recognizable latent or early symptomatic stage.
5. There should be a suitable test or examination to detect the disease.
6. The test should be acceptable to the population being screened.
7. The natural history of the condition, including development from latent to declared disease, should be adequately understood.
8. There should be an agreed policy on whom to treat as patients.
9. The cost of case-finding (including diagnosis and treatment of patients diagnosed) should be economically balanced in relation to possible expenditure on medical care as a whole.
10. Case-finding should be a continuing process and not a 'once and for all' project.

probably never diagnosed, lowering the apparent false negative rate. The few programmes with individual follow-up by physical examination, ultrasound or X-ray report higher rates of 'missed' cases.

The wide variation in rates of children detected early but still needing surgery is more likely related to differences in orthopaedic opinion than to the screening [9]. Rates of avascular necrosis of the femoral head among treated children in these studies were remarkably low (median value 0%) and follow-up data on this may be incomplete. Other reports suggest incidence rates between 0.18% and 30%, related to the grade of dislocation, age of the patient, and method of treatment [2, 13].

Criteria for screening

DDH screening remains controversial and meets most, but not all the criteria of a successful programme (Table 3) [73].

The condition should be an important health problem with high prevalence and serious consequences (criterion 1). The principles of treatment are agreed (criterion 2): DDH should be diagnosed as early as possible and treated. Dislocatable, immature and high-risk hips should be followed until normal maturity or deterioration to facilitate early treatment. Facilities for diagnosis and treatment are available in most Western countries (criterion 3). DDH has a recognizable latent phase. Although not all cases can be detected through physical examination, most can with diagnostic imaging procedures (criterion 4).

Several suitable screening tests are available for early detection (Table 1) (criterion 5), although education, skill and experience are important and can reduce the number of missed cases [25, 31, 36, 74]. However, it is difficult to determine the validity of these screening tests. False-negatives are usually underestimated and the number of true-positives is not known, because a gold standard is not available. The number of treated patients in most programmes is much higher than the number of patients with DDH. Even radiographic or ultrasonographically diagnosed minor dysplasia may return to normal without treatment. Neonatal clinical examination is good at identifying dislocation but not dysplasia without dislocation [5, 13, 62].

With one caveat, the tests are acceptable (criterion 6). They can be performed rapidly with minimal disturbance to the child. However some authors have suggested that the test manoeuvre itself may render some hip joints less stable [5, 21, 35, 74–76]. Others argue that those hips that can be provoked to deteriorate by the neonatal examination, were already at risk for full dislocation in later life [5, 77]. Nevertheless, the hypothesis that the test may provoke dislocation, may explain at least in part, the higher prevalence of late-diagnosed DDH in screened populations compared to the pre-screening era [6]. Finally, the obvious disadvantage of radiography, is the (small) amount of radiation exposure.

Unfortunately, prediction of the natural history of DDH is still difficult. About 80% of cases with neonatal clinical unstable hips resolve completely without treatment within a few months, leaving only 20% to deteriorate into subluxation, dislocation or dysplasia [5, 19]. These findings have been confirmed by ultrasound studies [77, 78]. Instability is thus a sign of potential dysplastic

development, while subluxation and dislocation inevitably lead to a compromised development of the acetabulum and femoral head, with difficulty in walking, unstable gait, and pain. Untreated cases are believed to cause 20–50% of cases of osteoarthritis of the hip in early adult life, either as a result of acetabular dysplasia itself or as a result of dislocatability, subluxation or dislocation in addition. The natural course of acetabular dysplasia may be benign, but 30–70% of untreated patients develop osteoarthritis of the hip joint [7, 8, 71, 72]. Unfortunately, it is not possible to predict which dysplastic hips will follow a benign course (criterion 7).

Early presymptomatic treatment of subluxation and dislocation is generally favoured, although there is debate over whether neonatally dislocatable hips should be treated, and about the cut-off for treatment in ultrasound screening programmes. Similar debates about radiographic diagnosis were resolved by choosing arbitrary but practicable cut-off criteria [2, 79]. The discussion is influenced by the fact that the invasiveness of treatment increases with the age of the child, but treatment also carries risks of avascular necrosis of the femoral head (criterion 8) [13].

The full costs of screening (criterion 9) include the costs of the implementation, diagnosis and treatment and the benefits include savings from prevention of more invasive treatment, of disabling disease and of loss of economic productivity. The cost-effectiveness of screening for DDH is largely dependent on the sensitivity of the screening test and on the ease of implementing screening in the existing health care delivery system. None of the studies on cost-effectiveness of screening for DDH takes all these factors into account. Comparison of the conclusions of the studies is largely prohibited by the fact that varying starting points have been used in the analyses (e.g. different criteria for diagnosis, for calculation of incidence or for treatment strategy). In a Canadian study, neonatal clinical screening was concluded to be cost-beneficial [80]. This conclusion was supported by a decision analysis from the USA, in which it was additionally concluded that ultrasound screening (either general or selective) would not be advantageous [81]. In Austria, however, a cost–benefit analysis comparing primary inpatient treatment in the pre-sonographic era with screening and inpatient treatment after introduction of ultrasound screening, showed that screening reduced total costs by about 40% [82]. A similar reduction of costs was observed after the introduction of ultrasound screening in Switzerland [4, 17]. In a randomized trial in Norway, the total costs of neonatal clinical screening programmes, whether or not combined with general or selective ultrasound screening, were equal and merely showed a shift of the costs for treatment of late diagnosed cases towards the cost for the screening programme itself. Data on the cost-effectiveness of the Norwegian programmes were not presented [83].

Screening for DDH should be a continuing process with programmes embedded in the regular medical services, so that population coverage increases and the organization can become more efficient and economical. The health care delivery system largely determines the chances of success of specific programmes in specific countries (criterion 10).

Conclusions

Screening for DDH is still controversial, because treatment rates are high, suggesting substantial over-treatment. However, early treatment consisting of immobilization with an abduction pillow or device is so simple and brief and has only a small impact on the child's development or on the parents, that even over-treatment may be preferable to treatment of an older infant or toddler. Nevertheless, although recent treatment advances have reduced the incidence of avascular necrosis, treatment is not absolutely risk free and over-treatment should be avoided.

The results of neonatal clinical screening programmes for DDH are encouraging when compared with other screening programmes. Neonatal ultrasound screening leads to undesirably high intervention rates, although in the near future its role after the first month of life may be better defined. Clinical neonatal screening programmes have the best results when combined with secondary screening of the high-risk population (by ultrasound or X-ray). Although more expensive, this is compensated by reduced treatment costs of children with late-diagnosed DDH.

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