The Prevalence of Rheumatic Heart Disease in Western Kenya:

An Echocardiographic Study

by

Thomas Holland

Duke Global Health Institute
Duke University

Date:_______________________

Approved:

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Christopher Woods, Supervisor

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G. Ralph Corey

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Nathan Thielman

Thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the Duke Global Health Institute in the Graduate School of Duke University

2012
ABSTRACT
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Abstract

Background:
Despite its declining incidence in resource-rich nations, rheumatic heart disease (RHD) remains a leading cause of morbidity and mortality in the developing world. Recent studies have demonstrated its high prevalence in Aboriginal populations in northern Australia, in Fiji, Tonga, and even parts of Africa including Nigeria and Mozambique, but little is known about its prevalence in Kenya. This epidemiologic data is needed to help inform prevention efforts.

Methods:
Transthoracic echocardiograms were performed on 526 randomly selected hospitalized patients, ages 5-35, on the surgical wards. These patients did not have known rheumatic heart disease. Data were collected on socioeconomic status, living conditions, and medical comorbidities. The primary outcome was the prevalence of echocardiographically confirmed rheumatic heart disease.

Results:
A total of 526 patients were enrolled. 9 had echocardiographic evidence of rheumatic heart disease (1.7%, 95% CI 0.6-2.8). Patients with rheumatic heart disease were more likely to be female. An association between RHD and crowded living conditions or socioeconomic status was not demonstrated in this study.

Conclusions:
Screening with echocardiography in Western Kenya has the potential to identify
subclinical rheumatic heart disease, which can inform secondary prevention efforts. Given low overall rates of rheumatic heart disease in the population in this study, efforts at primary prevention, i.e. identification and treatment of streptococcal pharyngitis and acute rheumatic fever, may yield greater population-level benefits.
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1. Introduction

1.1 Background

Cardiovascular disease continues to account for a significant burden of disease worldwide. However, it has been well established that the epidemiology of cardiovascular disease varies widely between resource-rich and resource-poor areas of the world. While coronary artery disease remains the preeminent contributor to cardiovascular disease burden in most Western countries, rheumatic heart disease (RHD) is the major cause of valvular pathology and premature heart failure in the developing world. Ideally, RHD would be preventable by aggressive efforts to identify and treat cases of streptococcal sore throat, so that acute rheumatic fever (ARF) and subsequent progression to valvular disease could be averted (i.e. primary prevention). However, large-scale efforts to employ school-based screening programs for this purpose have not been able to reduce incident cases of ARF. [1] Therefore, emphasis in RHD control efforts remains on secondary prevention – identification of patients who have valvular pathology from prior episodes of ARF, who may now benefit from secondary prophylaxis with monthly intramuscular penicillin to avert future episodes of ARF and the hastening of clinical progression of valvular disease. However, these efforts are hampered by a lack of reliable data about population-based incidence and prevalence rates of RHD.

As with many other disease processes with a predilection for resource-poor countries, the presumption is that sub-Saharan Africa is likely to be most severely impacted by RHD. Initial WHO estimates of RHD prevalence in sub-Saharan Africa were published in 1992, with a prevalence of 12.6/1000 children in Zambia and 10.2/1000 in Sudan. [2] More recently, the WHO has estimated that RHD currently afflicts 15.6 million worldwide with an estimated 233,000 deaths as a result annually. [3]
However, these estimates have been called into question by findings from more recent literature. The primary shortcoming of the aforementioned estimates is that the data is based on echocardiographic confirmation of patients with clinically suspected RHD. Intuitively, “clinical suspicion” is clinician-dependent and thus highly variable. Accordingly, it can be postulated that a substantial amount of RHD cases would be missed by these criteria. Due to these concerns the 2004 World Health Organization Expert Consultation Panel declared that in resource-poor nations, clinically silent rheumatic valve involvement should be managed as rheumatic heart disease until proven otherwise. [4]

These concerns were validated by a landmark 2007 study investigating the prevalence of RHD in school-age children in Cambodia and Mozambique. In this study, conventional approaches to diagnosing RHD (clinical examination findings plus confirmation by echocardiography) yielded an RHD prevalence of 2.2 cases per 1,000 schoolchildren and 2.3 cases per 1,000 schoolchildren in Cambodia and Mozambique respectively. When broad-based screening of schoolchildren was employed regardless of clinical suspicion, the prevalence of rheumatic heart disease rose to 21.5 cases per 1,000 in Cambodia and 30.4 per 1000 in Mozambique, a ten-fold increase. [5] These results appear to be more in accordance with a smaller study conducted in Kenya in 2006 that found the prevalence of mitral regurgitation detectable by TTE was approximately 62 per 1000. [6] This study probably over-estimated the true prevalence of RHD, because it reported trivial mitral regurgitation of unknown significance as well. However, it does bring attention to our inability to estimate accurately the number of people in the general population who have clinically significant RHD. To date, no formal population-
based studies have been performed to elucidate the prevalence of RHD-specific valvular disease in East Africa.

The caveat to all the above studies is the study population. All of the aforementioned studies focused on echocardiographic screening of school-age children (ages 6-17), at least partially because this is a relatively easy group to capture via school-based screening. There are several limitations of this approach. First, many of this subset of patients are still at risk to develop acute rheumatic fever, so older patients, if studied, might have even higher prevalence of RHD (though if mortality rates are sufficiently high in RHD patients, it is also possible that by older adulthood, prevalence is actually lower as patients with RHD have died). Second, ARF-related valvular pathology in this group may not have become detectable by echocardiography until well after the subjects obtained their TTE in the study. Seminal cohort studies by Bland and Jones in the 1950s defined the chronicity of RHD even in the pre-echocardiography era, with most of the patients developing heart failure after the age of eighteen. [7] In a Brazilian study 8% of patients with no evidence of carditis at the time of diagnosis of ARF had TTE-significant mitral disease at 2-year followup. [8] Third, school-based studies may bias in favor of lower RHD rates, since risk factors for rheumatic fever include overcrowding and poverty, which are also associated with lower school attendance.

An additional controversy in the efforts to quantify RHD prevalence has been the echocardiographic criteria used to make the diagnosis. WHO criteria were developed in 2001, [9] however these have been challenged more recently by Marijon et al., [10] who have argued for a more inclusive echocardiographic definition which incorporates morphologic criteria as well as Doppler-based assessment of regurgitant flow. Using
these expanded criteria may result in up to a 3-fold increase in case detection rates, [10] though importantly it is not known whether increased detection of subclinical disease would lead to more successful secondary prevention efforts. That is, it is not known whether these cases identified by morphologic criteria alone have a similar risk of progression to clinical disease, and therefore whether these patients would benefit from IM penicillin. Nevertheless, these expanded criteria offer the possibility of correctly identifying more subclinical disease in time to prevent progression to clinical RHD.

With the caveats noted above, we set out to obtain population level data about rheumatic heart disease prevalence in western Kenya, enrolling a group that was more inclusive in age than school-based screening.

1.2 Problem Statement

Despite the presumption that the majority of the burden of rheumatic heart disease falls on resource-limited countries, there is limited epidemiologic data demonstrating the scope of the problem. This is especially true of adult populations as most epidemiologic data is derived from studies of schoolchildren. In areas in which valve surgery is not commonly available, it is especially important to understand the burden of disease, so that appropriate secondary prevention practices can be instituted to prevent progression to clinical heart failure.
2. Study Design

2.1 Overall Design

This was a cross-sectional study conducted among inpatients on the orthopedic and general surgical wards of Moi Teaching and Referral Hospital (MTRH).

2.2 Study population:

Male or female patients ages 5-35 were eligible for participation, provided they were able to give informed consent (or assent for older children ages 12-17, consent of a parent or guardian for children ages 5-11) and did not have any contraindication, such as significant chest trauma, to performing an echocardiographic exam.

2.3 Sample size:

Planned sample size was 500 subjects. Based on estimates derived from prior large-scale RHD prevalence studies in Mozambique and Cambodia, [5] expected prevalence was approximately 2-3%. The selected sample size of N=500 will guarantee that the expected width of the 95% confidence interval is between 2.543% and 3.010% provided that the true prevalence is between 2.15% and 3.04% (see table 1).

<table>
<thead>
<tr>
<th>Cases per 1000</th>
<th>Prevalence</th>
<th>Width of 95% CI (sample size 500)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21.5</td>
<td>0.0215</td>
<td>0.02543</td>
</tr>
<tr>
<td>30.4</td>
<td>0.0304</td>
<td>0.03010</td>
</tr>
</tbody>
</table>
2.4 **Data Collection**

The research assistant screened admissions to the orthopedic and surgical wards on a rolling basis, Monday through Friday. After informed consent was obtained, the intake questionnaire was administered and the echocardiogram subsequently performed by one of the Moi Teaching and Referral Hospital echocardiography technicians.

The intake questionnaire (see Appendix A) was designed to assess clinical and personal elements related to the risk of rheumatic heart disease. The baseline data elements collected on the questionnaire were:

- **General data elements:** age, gender, place of residence (rural vs. urban), reason (diagnosis) for current admission, comorbid medical conditions including prior history of cardiovascular disease, cerebrovascular disease, and HIV status (if known). Using an established method, wealth was characterized by a summary variable accounting for household ownership of five items (automobile, flushing toilet, television, electricity and refrigerator) by allotting one point for each affirmative answer and assigning a score from 0-5. [11]

- **Rheumatic heart disease-related elements:** history of acute rheumatic fever, known history of rheumatic heart disease, number of family members with known heart disease.

Echocardiographic assessment consisted of resting 2-dimensional spectral and color flow Doppler imaging (see appendix B). Standard views obtained include parasternal (beside the breast bone) long axis and apical (on the left side of the chest)
four chamber views noting valve morphology on cross-sectional two-dimensional imaging and the degree and extent of mitral and aortic regurgitation using color flow Doppler. The apical imaging included anterior angulation to allow evaluation of the left ventricular outflow tract and the aortic valve. Transvalvular flow was assessed by measuring the peak velocity with continuous wave Doppler.

If the echocardiographic findings were of concern and warranted further investigation (whether rheumatic heart disease or other incidentally-discovered cardiac abnormalities), an appropriate referral to the cardiology clinic was made.

2.4.1 Case definition of rheumatic heart disease

Rheumatic heart disease was defined according to the criteria recommended by Marijon et al, [10] which combine WHO Doppler criteria and morphologic criteria as follows:

Doppler characteristics of the valvular regurgitation are defined by the association of a regurgitant jet 1 cm in length, seen in at least 2 planes, a mosaic color jet with a peak velocity 2.5 m/s, persisting throughout systole or diastole. Cases meeting these Doppler criteria will be classified as rheumatic heart disease.

Additionally, morphological criteria are based on the study of (1) leaflet morphology (typical marked thickening of the margins); (2) leaflet mobility (abnormal motion due to the posterior leaflet tip restriction); and (3) subvalvular apparatus morphology (prominent thickening, most often just below the valve, and shortening of chordal structures). In the presence of any Doppler-detected valvular regurgitation, presence of at least two morphologic rheumatic valvular criteria will also be classified as rheumatic heart disease.
Standard practice at Moi Teaching and Referral Hospital is for the echocardiography technicians to interpret the images at the time of acquisition. That is, echocardiograms are not routinely reviewed or “over-read” by cardiologists. For the purposes of the data presented in this paper, the presence or absence of rheumatic heart disease is based on the bedside interpretation of the echocardiography technician.
3. Results

Data entry was in Microsoft Excel and all descriptive and statistical analysis was conducted in Stata v11.0.

3.1 Patient Demographics

See Table 2 for a summary of patient demographics. Compared to those subjects without RHD, those with RHD were similar in age, crowding as measured by the number of residents in the patient’s home, a wealth score (summary score of ownership of five household items), and the proportion that live in urban versus rural areas. None of the patients had a previously known personal or family history of rheumatic fever or heart disease; two patients had a history of congenital heart disease (data not shown).

<table>
<thead>
<tr>
<th></th>
<th>Patients without RHD, ± SD (N=517)</th>
<th>Patients with RHD, ± SD (N=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean, in years)</td>
<td>17.5 ± 0.4</td>
<td>17.6 ± 2.7</td>
</tr>
<tr>
<td>% female</td>
<td>26.4 ± 2.0</td>
<td>77.8 ± 15.7</td>
</tr>
<tr>
<td># residents in the home</td>
<td>6.4 ± 0.1</td>
<td>7.2 ± 0.7</td>
</tr>
<tr>
<td>Wealth score</td>
<td>0.56 ± 0.05</td>
<td>0.89 ± 0.51</td>
</tr>
<tr>
<td>% urban dwelling (self-reported)</td>
<td>22 ± 2</td>
<td>22 ± 15</td>
</tr>
</tbody>
</table>

3.2 RHD prevalence

Overall prevalence of RHD was 17.1 per 1000 (95% CI, 6.0-28.2). As has been found in other populations studied, females were more likely to be affected (4.9% vs
0.5%, OR 9.71, 95% CI 1.99-47.30). There was no significant difference in RHD prevalence by self-identified rural vs. urban residence (OR 0.99, 95% CI 0.20-4.84), wealth score (OR 1.22, 95% CI 0.77-1.92) or with increased crowding as measured by the number of residents in the home (OR 1.13, 95% CI 0.88-1.44).

A description of the patients diagnosed with RHD is provided in Table 3.

### Table 3: Description of RHD Cases

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Rural vs Urban</th>
<th># of residents in the home</th>
<th>Reason for admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>8</td>
<td>Rural</td>
<td>8</td>
<td>Congenital megacolon</td>
</tr>
<tr>
<td>Female</td>
<td>9</td>
<td>Rural</td>
<td>10</td>
<td>Septic wound</td>
</tr>
<tr>
<td>Female</td>
<td>10</td>
<td>Rural</td>
<td>4</td>
<td>Fracture of tibia/fibula</td>
</tr>
<tr>
<td>Female</td>
<td>12</td>
<td>Rural</td>
<td>6</td>
<td>Hydatidosis</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
<td>Rural</td>
<td>8</td>
<td>Mastoiditis/brain abscess</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
<td>Rural</td>
<td>10</td>
<td>Peritonitis</td>
</tr>
<tr>
<td>Female</td>
<td>21</td>
<td>Rural</td>
<td>7</td>
<td>Fracture of radius/ulna</td>
</tr>
<tr>
<td>Male</td>
<td>23</td>
<td>Urban</td>
<td>9</td>
<td>Osteogenic sarcoma</td>
</tr>
<tr>
<td>Male</td>
<td>26</td>
<td>Urban</td>
<td>6</td>
<td>Ulnar fracture</td>
</tr>
</tbody>
</table>
4. Discussion

In this group of patients, rheumatic heart disease, as detected by bedside transthoracic echocardiography, was found in just under 2% of study participants (17.1 per 1000). This point estimate is similar to those provided in the seminal study by Marijon et al. in Cambodian and Mozambican schoolchildren, in which RHD was diagnosed in 21.5 per 1000 subjects in Cambodia and 30.4 per 1000 in Mozambique. [5] Females were disproportionately affected, as was suggested (though not statistically significant) in the Marijon study. Perhaps unsurprisingly, none of the participants had a known history of acute rheumatic fever or rheumatic heart disease. The socioeconomic status and household crowding of those with RHD was similar to those without.

This study has several limitations. First, it is possible that the population described here is not representative of the broader western Kenyan population. The surgical wards were chosen as a proxy for the general population as these patients are most commonly admitted for trauma – i.e., a condition that is both unlikely to be related to the likelihood that they have underlying RHD, and one that would impel patients to present to the hospital regardless of financial or logistic constraints. This is in contrast to the general medicine wards, where patients with rheumatic heart disease are disproportionately represented, as many are admitted for complications of the disease. However, not all patients on the surgical wards are admitted for trauma - for example, the RHD patient with hydatidosis, a disease that, like RHD, is more common among poorer patients. A different sampling strategy such as random home visits might have yielded different prevalence data, and this would in fact be the ideal method for future work.
Secondly, the reliance on echocardiographic technicians to make the diagnosis of RHD raises the possibility that cases were missed or, alternatively, that those cases diagnosed will subsequently be refuted upon review by cardiologists certified in echocardiography. It seems more likely that cases were missed, leading to underestimation of true prevalence.

Despite these limitations, it is clear than there is an undiagnosed burden of rheumatic heart disease in western Kenya, and echocardiographic screening is a feasible means by which to identify at least a portion of these cases. I next outline currently ongoing and suggested future efforts to follow upon this study.

### 4.1 Future Directions

#### 4.1.1. Core lab review of the echocardiograms

As alluded to above, it will be necessary to confirm the accuracy of the technician interpretation of the study echocardiograms. All echocardiograms will be reviewed in a blinded fashion by two qualified cardiologists. Where there is discrepancy between reviewers regarding the presence or absence of rheumatic heart disease, a third cardiologist will make the final determination, blinded to the first two reports. Interrater reliability of echocardiographic assessment will be determined using the kappa statistic. This will increase confidence in the point estimate and may in fact lead to the diagnosis of more cases.
4.1.2. Next steps in prevention of rheumatic heart disease

Having demonstrated that there is a substantial burden of rheumatic heart disease in Western Kenya, the logical next step is to focus efforts on prevention of its consequences. As this study demonstrates, it requires substantial effort to identify a relatively small number of RHD cases via echocardiographic screening. Clinical screening (by history and physical exam) would be easier to employ, but has proven inferior to echocardiography for the detection of RHD, even when trained cardiologists perform the physical exam. [5] A more effective, and cost-effective, approach may be to aim our efforts at different steps in the pathogenesis of RHD. Possibilities range from big picture approaches to alleviate poverty, sanitation, and crowding, thereby decreasing the intensity of streptococcal transmission and incident cases of streptococcal pharyngitis. This has been dubbed “primordial prevention” of RHD, and is undoubtedly a worthwhile pursuit – alleviating poverty is clearly laudable for a host of reasons that are independent of the effect on rheumatic heart disease.

The next step beyond primordial prevention would be to identify and treat cases of streptococcal pharyngitis to prevent acute rheumatic fever, or, failing that, to identify and treat cases of acute rheumatic fever. Institution of secondary prophylaxis with monthly penicillin injections can, as noted previously, prevent progression to rheumatic heart disease and its attendant morbidity and mortality.

With a number of collaborators in Kenya and at Duke, we have begun to look for these streptococcal pharyngitis and acute rheumatic fever cases. The first challenge in this effort is to figure out when and where the cases are. Since there is rheumatic heart disease in western Kenya, it follows that there must be cases of group A streptococcal pharyngitis and acute rheumatic fever, and that these are being inadequately treated.
Our suspicion is that patients with these diagnoses are actually presenting to healthcare providers, but are being misdiagnosed – usually as malaria. Indeed, it is known that acute febrile illness is among the most common conditions prompting a visit to a healthcare provider in the developing world, and it is also the leading cause of childhood mortality. [12] Given malaria’s historic preeminence in these presentations, the WHO Integrated Management of Childhood Illness (IMCI) guidelines were developed to guide treatment at first-level facilities, emphasizing antimalarial administration. Recently, reports have emerged describing a decline in the burden of malaria in several countries, [13] including Kenya. [14,15] However, despite recent WHO guidelines advocating parasitological confirmation of malaria infection prior to initiation of treatment, [16] more than 90% of outpatients with fever in western Kenya are prescribed antimalarials (O’Meara, unpublished observations). This practice persists in part due to a lack of evidence regarding other pathogens contributing to acute febrile episodes. In the wake of this changing malaria burden, it is expected that other pathogens are emerging as important contributors to the burden of pediatric illness. It is not yet known whether group A streptococcus, either in the form of acute pharyngitis or the post-infectious acute rheumatic fever, is one of the other pathogens leading to presentations to local health facilities. The alternative hypothesis is that, while there must be streptococcal pharyngitis and acute rheumatic fever, patients may not be ill enough to present for care with these diagnoses. Thus, in work that continues at the time of this writing, we are enrolling a group of febrile patients (and an afebrile comparison group) and looking for non-malarial pathogens, including group A streptococcus, among those presenting to a rural district hospital.

Preliminarily, it has been interesting (and surprising) to learn that group A
streptococcus has been very rarely present, as determined by rapid antigen detection test or by bacterial culture, from pharyngeal samples. In fact, less than 1% of those enrolled have had positive streptococcal tests of either kind. Conversely, we have been able to detect influenza A in 15.9% of 157 patients tested via nasopharyngeal swabs. Efforts are ongoing to test for other pathogens, including rickettsiae, leptospirosis, dengue fever, Q fever, and brucellosis.

If group A streptococcus is not detectable in pediatric febrile illnesses cases at a district hospital during a one year period, then where might they be? Possibilities include: 1) febrile patients who are ill enough to present to the hospital are heavily pretreated with over-the-counter medications prior to presentation, including antimalarials and often beta-lactam antibiotics that might have led to false-negative streptococcal testing; 2) streptococcal transmission is cyclic and we are sampling at a time of low incidence; 3) children with streptococcal pharyngitis or acute rheumatic fever are not ill enough to present to the hospital and are instead being managed at home. More study will be required to elucidate this seeming paradox of prevalent RHD without detectable group A streptococcal infections.
5. Conclusion

As suspected based on anecdotal experience and non-population based data, there is a substantial burden of undiagnosed rheumatic heart disease in western Kenya. Echocardiographic screening can identify at least a portion of these cases. A multipronged approach, including enhanced RHD screening efforts as well as research into the precursor conditions of streptococcal pharyngitis and acute rheumatic fever, will hopefully yield a menu of possible interventions to decrease the devastating burden of this all-too-common illness.
Appendix A

Rheumatic Heart Disease Study
DATA COLLECTION FORM AND INTAKE QUESTIONNAIRE

<table>
<thead>
<tr>
<th>General</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Patient is on which ward?</td>
</tr>
<tr>
<td>2. Reason for admission</td>
</tr>
<tr>
<td>3. Year of birth</td>
</tr>
<tr>
<td>4. Date of birth (if known):</td>
</tr>
<tr>
<td>_ _ / _ _ day / month</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Demographics</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Sex:</td>
</tr>
<tr>
<td>8. Residence:</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Historical Elements</th>
</tr>
</thead>
<tbody>
<tr>
<td>9. Do you own any of the following?:</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>Own an automobile?</td>
</tr>
<tr>
<td>Have a flushing toilet in your home?</td>
</tr>
<tr>
<td>Have a television in your home?</td>
</tr>
<tr>
<td>Have electricity (current) in your home?</td>
</tr>
<tr>
<td>Have a refrigerator in your home?</td>
</tr>
<tr>
<td>10. How many people (including yourself) live in your home?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Historical Elements</th>
</tr>
</thead>
<tbody>
<tr>
<td>11. Do you have any personal or documented history of:</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>Rheumatic fever?</td>
</tr>
<tr>
<td>Rheumatic heart disease?</td>
</tr>
<tr>
<td>Other heart disease?</td>
</tr>
</tbody>
</table>

(continued) Any personal or documented history of:
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>Stroke?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other neurologic disease? If yes, specify:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Kidney disease?</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>HIV?</td>
</tr>
</tbody>
</table>

12. Does anyone in your immediate family (parents, brothers, sisters, children) have known rheumatic heart disease?

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>Don't know</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>If yes, how many people?</td>
</tr>
</tbody>
</table>

Signature

Research Staff Print Name and Sign:

- Clinical Officer
- Research Assistant
- Physician

[Pick the date]

Rheumatic Heart Disease Study v1.0  Page 2 of 2
Appendix B

RHD STUDY ECHOCARDIOGRAM ACQUISITION PROTOCOL

I. Patient Identifiers

Patient Identifiers must be obtained and verified with patient including:

- Study ID number
- Enter Study ID Number for Family Name
- Sex
- Full Date of birth if known or Year
- Technicians name

II. Machine Settings: Gain should be between 40 and 55.

III. Acquisition Protocol

All images must be ECG-gated; Obtain as clean an ECG signal as possible.

The results of this study will only be as good as the images that are acquired!!

All acquired loops should be 2-beats (or 2 seconds if ECG signal is poor)

<table>
<thead>
<tr>
<th>1. Parasternal Long Axis</th>
<th>2. RV Inflow</th>
</tr>
</thead>
<tbody>
<tr>
<td>2D optimizing LV endocardial borders including left atrium</td>
<td>2D loop</td>
</tr>
<tr>
<td>2D Zoom of mitral valve and aortic valve</td>
<td>Color Doppler of TV to show TR</td>
</tr>
<tr>
<td>Color Doppler of Aortic Valve for AI</td>
<td>CW Doppler of tricuspid valve. Optimize scale so TR jet takes up as much of display as possible but do not cut-off the tip of the jet</td>
</tr>
<tr>
<td>Color Doppler of MV to show MR</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. RV Outflow</th>
<th>4. Parasternal SAX of the LV</th>
</tr>
</thead>
<tbody>
<tr>
<td>2D view with bifurcation (if possible)</td>
<td>2D loop of the MV in SAX (good quality for planimetry)</td>
</tr>
<tr>
<td>Color Doppler to show PR</td>
<td>Color Doppler of MV to show MR</td>
</tr>
<tr>
<td>CW of PV to show Pulmonic outflow</td>
<td></td>
</tr>
<tr>
<td>PW of PV at level of valve – ONLY IF CW velocity is &gt; 200cm/sec</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>5. Parasternal SAX of the Aortic Valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>2D loop</td>
</tr>
<tr>
<td>2D Zoom on Aortic Valve</td>
</tr>
<tr>
<td>Color Doppler of the Aortic Valve for AI</td>
</tr>
</tbody>
</table>
6. **Apical 4 Chamber**
- 2D image showing all four chambers - Try not to foreshorten
- Show a loop with decreased depth such that LV occupies most of the imaging sector ensuring all walls are visualized
- Color Doppler of MV
- CW Doppler of the MV for LV inflow
- PW Doppler of MV inflow at tips of MV
- Color Doppler of the AV for AI
- CW Doppler of AV showing AI
- CW Doppler of AV showing LV outflow
- PW Doppler of the LVOT
- Color Doppler of TV
- CW Doppler of TV

7. **Apical 2 Chamber**
- 2D optimizing LV endocardial borders: Need to see all aspects of the Anterior Wall, Inferior Wall, and Apex
- Color Doppler of MV for MR
- CW Doppler of MV for LV inflow
- PW Doppler of MV inflow at tips of MV

9. **Subcostal 4-chamber view**
- 2D of IVC with respiration (4-beat loop; Need to see change with respiration)

10. **Suprasternal View**
- Color Doppler ascending aorta
- CW ascending aorta
- PW at LVOT

8. **Apical 3 Chamber View**
- 2D loop
- Color Doppler of MV
- CW Doppler of MV for LV inflow—ONLY IF velocity is greater than 200 cm/sec
- PW Doppler of MV inflow at tips of MV – ONLY IF velocity is greater than 200 cm/sec
- Color Doppler of AV for AI
- CW Doppler of AV showing AI
- CW Doppler of AV showing LV outflow
- PW Doppler of LVOT – ONLY IF CW velocity greater than 200 cm/sec

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References


