INSIGHT

Issue 19
June 2012

An Official Journal Of Kerala Govt. Optometrists' Association

Reg. No. 285

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Printed at
GP Offset
Thiruvananthapuram

എഡിറ്റോറിയൽ

സുഹൃത്തുക്കളേ,

കേരളാ ഗവൺമന്റ് ഒപ്റ്റോമെട്രിസ്റ്റ് അസ്സോസിയേഷന്റെ അഭിമാനമായ ജേർണൽ ഇൻസൈറ്റിന്റെ മറ്റൊരു ലക്കം കൂടി നിങ്ങളേവർക്കും മുന്നിൽ സമർപ്പിക്കുന്നു. കഴിഞ്ഞ ലക്കത്തിലാരംഭിച്ച ഒപ്റ്റോടൂൾസ് എന്ന പംക്തി വളരെയേറെ പേർ ശ്രദ്ധിച്ചെന്ന് മനസ്സിലാക്കിയതിൽ സന്തോഷം.

മേജർ ആശുപത്രികളിലെ തസ്തിക സീനിയർ സിസ്റ്റ് (ഗസറ്റഡ് ഓഫീസർ) അസ്സൈൻ ചെയ്യണമെന്ന് കാലാകാലങ്ങളായി സർക്കാർ സർവ്വീസിലുള്ള സീനിയർ ഒപ്റ്റോമെട്രിസ്റ്റുമാർ ആവശ്യപ്പെട്ട് വന്നിരുന്നത് സംഘടനയുടെ സമയോചിതമായ ഇടപെടലോടെ നടപ്പാക്കപ്പെട്ടതിന്റെ സന്തോഷത്തിലാണ് നാമേവരും. അരോഗ്യ വകുപ്പിൽ ഒപ്റ്റോമെട്രിസ്റ്റുകളുടെ പുതിയ തസ്തികകൾ ശൃഷ്ടിക്കുന്നതിനും മെഡിക്കൽ വിദ്യാഭ്യാസ വകുപ്പിന് കീഴിൽ നടക്കുന്ന ഒഫ്ത്താൽമിക്ക് അസിസ്റ്റന്റ് ഡിപ്ലോമ കോഴ്സിന്റെ പഠന നിലവാരം ഉയർത്തുന്നതിനുമുള്ള തീവ്ര ശ്രമങ്ങളും നടന്നുകൊണ്ടിരിക്കുന്നു.

ചില വിമർശനാത്മകമായ ഇ-മെയിലുകൾ എഡിറ്റർക്ക് ലഭിച്ചത് എഡിറ്റോറിയൽ ബോർഡ് ചർച്ച ചെയ്ത് കഴിവിന്റെ പരമാവധി പ്രശ്നരഹിതമായി ഈ ലക്കം പ്രസിദ്ധീകരിക്കാൻ സാധിച്ചു എന്നാണ് വിശ്വാസം. തുടർന്നും ക്രിയാത്മകമായ ആശയങ്ങളും അഭിപ്രായങ്ങളും പ്രതിക്ഷിച്ചു കൊണ്ട്

അരുൺ ആർ ജെ
എഡിറ്റർ

For internal circulation only

PRESIDENT'S VOICE

പ്രിയ സുഹൃത്തേ,

ഈ പ്രസിദ്ധീകരണം നിങ്ങളിലെത്തുമ്പോൾ നാമോരുരുത്തരും ഒപ്റ്റോമീറ്റിന്റെ വേദിയിലായിരിക്കുമല്ലോ. വീണ്ടും എന്നെ കേരളാ ഗവൺമന്റ് ഒപ്റ്റോമെട്രിസ്റ്റ് അസ്സോസിയേഷന്റെ പ്രസിഡന്റ് പദവിയിലെത്തിച്ച ഓരോരുത്തരോടുമുള്ള എന്റെ ഹൃദയം നിറഞ്ഞ നന്ദി അറിയിക്കട്ടെ. ഇതുവരെയും സത്യസന്ധമായി ഈ സംഘടനയെ നയിക്കാൻ സഹായിച്ച ഭാരവാഹികളോടും ഓരോരോ അംഗങ്ങളോടും ഞാനെന്റെ കടപ്പാടും നന്ദിയും വീണ്ടും അറിയിക്കുന്നു.

നമ്മുടെ ഔദ്യോഗിക ജേർണലായ ഇൻസൈറ്റിന്റെ പ്രസിദ്ധീകരണം ആനുകാലിക വിവരങ്ങൾ മനസ്സിലാക്കാൻ വളരെ സഹായിക്കുന്നു എന്നത് ഒരു വസ്തുത തന്നെയാണ്. അതിന് വേണ്ടി പ്രവർത്തിക്കുന്ന ഏവരോടുമുള്ള നന്ദിയും ഈ അവസരത്തിൽ സമർപ്പിക്കുന്നു. സംഘടനയുട പൂർവ്വ ചരിത്രം പരിശോധിക്കുമ്പോൾ കഴിഞ്ഞ കുറച്ച് കാലം പ്രതികൂലങ്ങളുടെ സമയമായിരുന്നു. എന്നാൽ ഈ അവസരങ്ങളിലൊക്കെയും ശക്തിയോടെ പ്രവർത്തിക്കാൻ കഴിഞ്ഞത് അസ്സോസിയേഷന്റെ ഒരു നേട്ടം തന്നെയാണ്. വളരെ കാലമായി നമ്മളാഗ്രഹിച്ച സീനിയർ ഗ്രേഡ് പോസ്റ്റ് അസ്സൈന്മെന്റും സീനിയർ മോസ്റ്റ് ജില്ലാ ഒഫ്ത്താൽമിക്ക് കോർഡിനേറ്ററെ അരോഗ്യ വകുപ് ആസ്ഥാനത്ത് പോസ്റ്റ് ചെയ്യനുമുള്ള ഉത്തരവ് ആയെങ്കിലും അത് പ്രാവർത്തികമാക്കാൻ കഴിഞ്ഞത് സംഘടനയുടെ ഇടപെടൽ കൊണ്ടാണെന്ന് നിസ്സംശയം പറയാം.

കേരളത്തിലെ ഒപ്റ്റോമെട്രിസ്റ്റുകളുടെ കൂട്ടായ്മ എടുത്ത് പറയേണ്ട ഒന്നുതന്നെയാണ്. നമ്മുക്ക് ഇനിയും വളെരെയധികം നേട്ടങ്ങൾ കൈവരിക്കേണ്ടതായിട്ടുണ്ട്. പുതിയ തസ്തികകൾ ശൃഷ്ടിക്കൽ, കൂടുതൽ വിഷൻ സെന്റർ തുടങ്ങുക, ശമ്പള കമ്മീഷൻ അപാകതകൾ പരിഹരിക്കുക, BSc ഒപ്റ്റോമെട്രി അഡ്മിഷൻ നേടുന്നതിന് ഡെപ്യൂട്ടേഷൻ നേടിയെടുക്കുക, എല്ലാ മെഡിക്കൽ കോളേജുകളിലും BSc ഒപ്റ്റോമെട്രി കോഴ്സ് തുടങ്ങുക, മെഡിക്കൽ വിദ്യാഭ്യാസ വകുപ്പിൽ തസ്തിക ഒപ്റ്റോമെടിസ്റ്റായി പുനർ നാമകരണം ചെയ്യിക്കുക തുടങ്ങി നിരവധി ആവശ്യങ്ങളിൽ വരും കാലങ്ങളിൽ ഒത്തൊരുമയോടെ പ്രവർത്തിച്ച് സർക്കാരിൽ നിന്നും അനുകൂലമായ ഉത്തരവുകൾ നേടിയെടുക്കേണ്ടതായിട്ടുണ്ട്.

നമ്മുടെ നാടിന്റെ അന്ധതാനിയന്ത്രണ പരിപാടിയുടെ ഊർജ്ജിതമായ പ്രവർത്തനത്തിനും നമ്മുടെ ന്യായമായ ആവശ്യങ്ങൾ നേടിയെടുക്കാനും ഓരോ അംഗങ്ങളും ഒരൊറ്റ മനസ്സോടെയും അർപ്പണ ബോധത്തോടെയും മുന്നേറാൻ ദൈവം സഹായിക്കട്ടെ എന്ന പ്രാർഥനയോടെ നിർത്തുന്നു.

സി എം ജൈസ്സി
പ്രസിഡന്റ്

FROM SECRETARY'S DESK

മാന്യ സുഹൃത്തേ,

കേരളത്തിലെ പ്രബുദ്ധരായ ഒപ്റ്റോമെട്രിസ്റ്റുകൾ ഒന്നടങ്കം പങ്കെടുത്ത് ആവേശോജ്വലമായി സമാപിച്ച 20ാം സംസ്ഥാന സമ്മേളനത്തിൽ കേരളാ ഗവൺമന്റ് ഒപ്റ്റോമെട്രിസ്റ്റ് അസ്സോസിയേഷന്റെ ജനറൽ സെക്രട്ടറിയായി എന്നെ തെരഞ്ഞെടുത്തതിന്റെ നന്ദി വാക്കുകൾക്കതീതമാണ്. ഭിന്നതയല്ല 'ഒരുമയാണ്' കേരളത്തിലെ ഒപ്റ്റോമെട്രിസ്റ്റുകൾ ആഗ്രഹിക്കുന്നതെന്ന് ദുഷ്പ്രചാരണങ്ങളെ അവഗണിച്ച് 20ാം സംസ്ഥാന സമ്മേളനത്തിൽ തിങ്ങി നിറഞ്ഞ സദസ്സ് തെളിയിച്ചു. നിങ്ങൾ എന്നിലർപ്പിച്ച വിശ്വാസവും, ഏൽപ്പിച്ച കർത്തവ്യവും എത്രമാത്രം വലുതാണെന്ന് തിരിച്ചറിയുന്നു.

സംഘടനയുടെ അംഗബലം 80 ശതമാനത്തിനടുത്ത് എത്തിക്കാൻ കഴിഞ്ഞു എന്നതിൽ അഭിമാനമുണ്ട്. ഇതിനായി പ്രവർത്തിച്ച സംഘടനയുടെ എല്ലാ ഭാരവാഹികളേയും പ്രവർത്തകരേയും അഭിനന്ദനം അറിയിക്കുന്നു. നേരത്തേ തന്നെ സംഘടന തുടങ്ങിവച്ചിരുന്ന പല കാര്യങ്ങളും പൂർത്തീകരിക്കാൻ കഴിഞ്ഞതിൽ ചാരിതാർത്ഥ്യമുണ്ട്. കേരളത്തിലെ ഒപ്റ്റോമെട്രിസ്റ്റുകളുടെ ചിരകാലാഭിലാഷമായിരുന്നു സീനിയർ പോസ്റ്റ് അസ്സൈനിംഗ്. ഉത്തരവിറങ്ങിയിട്ടും നടപ്പിലാക്കാതെ മാറ്റിവച്ചിരുന്ന പോസ്റ്റ് അസ്സൈനിംഗ് നമ്മുടെ ശക്തമായ ഇടപെടൽ കൊണ്ട് അവസാനം നടപ്പിലായി.

കൗൺസിലിംഗ് പ്രക്രിയയിലൂടെ സുതാര്യമായി, കാര്യമായ പരാതികൾക്കൊന്നും ഇട നൽകാതെ സീനിയർ പോസ്റ്റ് അസ്സൈൻ ചെയ്ത് മാറ്റി നിയമിക്കാൻ പ്രയത്നിച്ച ആരോഗ്യവകുപ്പ് ഡയറക്ടറേറ്റിലെ ബന്ധപ്പെട്ട ഉദ്യോഗസ്ഥരെ എത്ര അഭിനന്ദിച്ചാലും മതിയാകില്ല. ചിലരുടെ സ്ഥാപിത താത്പര്യങ്ങൾ നടപ്പിലാവതെ പോയതിലുള്ള ഏതാനും പരാതികൾ മാത്രമാണ് ഉയർന്ന് വന്നത്. എന്നിരുന്നാലും വിരലിലെണ്ണാവുന്ന ചില സീനിയർ ഒപ്റ്റോമെട്രിസ്റ്റുകൾക്ക് ബുദ്ധിമുട്ട് ഉണ്ടായിട്ടുള്ളതിൽ ഖേദമുണ്ട്. വരും വർഷങ്ങളിൽ തീർച്ചയായും ഈ ബുദ്ധിമുട്ടുകൾ പരിഹരിക്കപ്പെടും. മാത്രമല്ല, മിക്കവാറും അതിൽ എല്ലാവർക്കും സ്ഥലം മാറ്റം മുഖേന സംഭവിച്ച ബുദ്ധിമുട്ടുകൾ ലഘൂകരിക്കുന്നതിനുള്ള താത്ക്കാലിക സംവിധാനങ്ങൾ ഉണ്ടാക്കിക്കൊടുക്കാൻ നമുക്ക് കഴിഞ്ഞു. സാങ്കേതിക കാരണങ്ങളാൽ മാറ്റിവച്ചിരിക്കുന്ന അപേക്ഷയിലും ഉടനടി നടപടി കൈക്കൊള്ളുമെന്ന് പ്രതീക്ഷിക്കുന്നു.

2012ലെ പൊതു സ്ഥലം മാറ്റവും കാര്യമായ പരാതികളൊന്നും ഇല്ലാതെ നടന്നു. ഇതിൽ സംഘടനയ്ക്കുള്ള പങ്ക് ചെറുതായി കാണരുത്. കഴിഞ്ഞ കുറേ വർഷങ്ങളുടെ കണക്കെടുത്താൽ, ഏറ്റവും കുറവ് കോടതി വ്യവഹാരങ്ങൾ ഉണ്ടായത് ഈ വർഷമാണെന്നുള്ളത് ശ്രദ്ധേയമാണ്.

സംസ്ഥാനത്തെ മുഴുവൻ ഒഴിവുകളിലും നിയമനം നടത്തിക്കാനും, ജില്ലാ ഒഫ്ത്താൽമിക്ക് കോർഡിനേറ്റർ, സീനിയർ ഒപ്റ്റോമെട്രിസ്റ്റ് പ്രമോഷനുകൾ യഥാസമയം നടപ്പിലാക്കിക്കാനും കഴിഞ്ഞു. മന്ദഗതിയിലായിരുന്ന പോസ്റ്റ് ക്രിയേഷൻ സംബന്ധിച്ച നടപടികൾ ത്വരിതപ്പെടുത്താൻ സാധിച്ചിട്ടുണ്ട്. എന്നാൽ ഇക്കാര്യത്തിൽ ആരോഗ്യവകുപ്പിലെ ഒഫ്ത്താൽമിക്ക് സെൽ കുറേക്കൂടി കാര്യക്ഷമത കാണിക്കേണ്ടിയിരിക്കുന്നു. ശമ്പള പരിഷ്ക്കരണ അനോമലി സംബന്ധിച്ച് നാം സമർപ്പിച്ച നിവേദനം ഇപ്പോൾ സർക്കാരിന്റെ സജീവ പരിഗണനയിലാണ്. ഉടനടി അനുകൂല നടപടി ഉണ്ടാകും എന്ന് പ്രതീക്ഷിക്കാം.

ജനറൽ സെക്രട്ടറി എന്ന നിലയിൽ എന്നെ അറിയിക്കുന്ന ഒപ്റ്റോമെട്രിസ്റ്റുകളുടെ ന്യായമായ എല്ലാ പ്രശ്നങ്ങളിലും യഥാസമയം ഇടപെട്ട് പരിഹാരം കാണാൻ പരമാവധി ശ്രമിച്ചിട്ടുണ്ട് - തുടർന്നും അതുണ്ടാവുമെന്ന് ഉറപ്പ് തരുന്നു.

ഒരുവശത്ത് ഭിന്നതയുടെ സ്വരങ്ങൾ നേർത്ത് വരുമ്പോൾ സംഘടനയെ തുരങ്കം വയ്ക്കാൻ മറ്റൊരുവശത്ത് പുതിയ പേരുകളിൽ ചില ഉയിർത്തെഴുന്നേൽപ്പുകൾ നടക്കുന്നത് നിങ്ങൾ തിരിച്ചറിയണം. ഒപ്റ്റോമെടിസ്റ്റുകളെ ഭിന്നിപ്പിക്കുന്നതിന് ചിലർ ബോധപൂർവ്വം നടത്തുന്ന ഈ ശ്രമങ്ങളെ നാം ഒറ്റക്കെട്ടായി നിന്നെതിർത്ത് തോൽപ്പിക്കണം. സ്ഥാപിത താത്പര്യങ്ങൾ നടപ്പിലാക്കി കിട്ടുന്നതിന് വേണ്ടി സംഘടനയ്ക്കും, വകുപ്പിനും, സർക്കാരിനും അതീതരാണെന്ന് 'സ്വയം പ്രാധാന്യം' ഭാവിച്ച് അഹങ്കരിച്ച് സംഘടനകളെ 'തകർത്ത് തരിപ്പണമാക്കാൻ' ഇറങ്ങിത്തിരിച്ചിരിക്കുന്ന ചിലർക്കെതിരേ പ്രതികരിക്കുകയല്ല, സഹതപിക്കുകയാണ് വേണ്ടത്. താനിരിക്കുന്ന കസേരയുടെ 'വലിപ്പം' ഈ സംഘടനയുടെ നിരവധി വർഷങ്ങളുടെ പ്രവർത്തനഫലമാണെന്നോർക്കണം.

കഴിഞ്ഞ 20 കൊല്ലമായി അജയ്യമായി നിലകൊള്ളുന്ന കേരളത്തിലെ സർക്കാർ ഒപ്റ്റോമെട്രിറ്റുകളുടെ ഏക അംഗീകൃത സംഘടനയെ കൂടുതൽ ശക്തമായി നിലനിർത്തുന്നതിന് നമുക്ക് ഒത്തൊരുമിച്ച് പ്രവർത്തിക്കാം. ഒപ്റ്റോമെട്രിസ്റ്റുകളുടെ അന്തഃസ്സ് കാത്ത് സൂക്ഷിക്കാം

പി ശ്രീകുമാർ
ജനറൽ സെക്രട്ടറി

സംസ്ഥാന സമ്മേളനം

കേരളാ ഗവൺമന്റ് ഒപ്റ്റോമെട്രിസ്റ്റ് അസ്സോസിയേഷന്റെ 20ാം സംസ്ഥാന സമ്മേളനം 13-11-2011ന് 10 മണിക്ക് എറണാകുളം കച്ചേരിപ്പടി ആശീർ ഭവനിൽ (വി സീമ നഗർ) നടന്നു. പ്രസിഡന്റ് ശ്രീമതി.സി എം ജെസ്സിയുടെ അദ്ധ്യക്ഷതയിൽ കൂടിയ സമ്മേളനം ശ്രീ.ഡൊമിനിക്ക് പ്രസന്റേഷൻ MLA ഉത്ഘാടനം ചെയ്തു. ശ്രീ.ഹൈബി ഈഡൻ MLA മുഖ്യ പ്രഭാഷണം നടത്തി. എറണാകുളം ജനറൽ ആശുപത്രി സൂപ്രണ്ട് ഡോ.ജുനൈബ് റഹ്മാൻ ആശംശകൾ നേർന്നു. വിശിഷ്ടാതിഥിയായി ഐഡിയ സ്റ്റാർ സിങ്ങർ ഫെയിം അഞ്ചു ജോസഫ് പങ്കെടുത്തു. നേത്രാരോഗ്യ ബോധവത്കരണ മേഖലയിൽ സുപ്രധാന സംഭാവനകൾ നൽകിയ ശ്രീ.റഹീം പാലാറ (ഒപ്റ്റോമെട്രിസ്റ്റ്, വളവന്നൂർ പ്രാഥമികാരോഗ്യ കേന്ദ്രം), കവി ശ്രീ.സന്തോഷ് കോട്ടുകൽ എന്നിവരെ ചടങ്ങിൽ ആദരിച്ചു. ശ്രീ.ആർ ബിനോയ് സ്വാഗതവും ശ്രീ.ജി സാബു നന്ദിയും പറഞ്ഞു.

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LOGMAR VISION CHARTS

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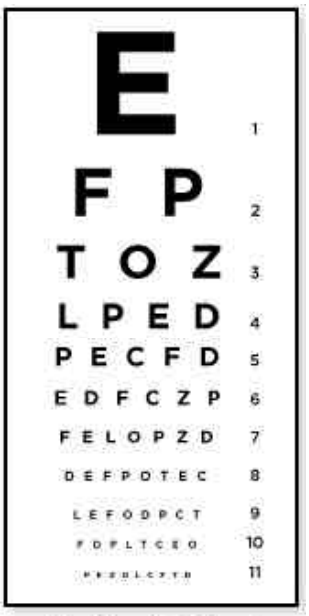
Whenever we think of Visual Acuity, the first thing that comes to our mind is the Snellen's chart. Though this time tested chart has been in service for more than 1 ½ century, newer, more scientific vision charts are replacing the Snellen's chart.

The term 'Visual Acuity' has been coined by Donders in 1861. It is the ability of the eye, to resolve a spatial pattern, separated by a visual angle of one minute of an arc. The Snellen's chart was designed by Hermann Snellen in 1862. The size of the letter in the chart and its constituent lines are calculated by mathematical principles. The size of the letters is such that the whole letter will subtend an angle of 5 minutes at the nodal point, at the given distance. Breadth of each line will subtend an angle of one minute at the nodal point. The Snellen's Chart is conventionally designed to be kept at 6 meters. This is done considering the fact that at 6M, divergence of the rays which enter the pupil is so slight that the rays can be considered parallel. The long lasting bond between the Snellen's Chart and the ophthalmic fraternity is now becoming slightly lax.


Hermann Snellen

Problems with the Snellen's Chart

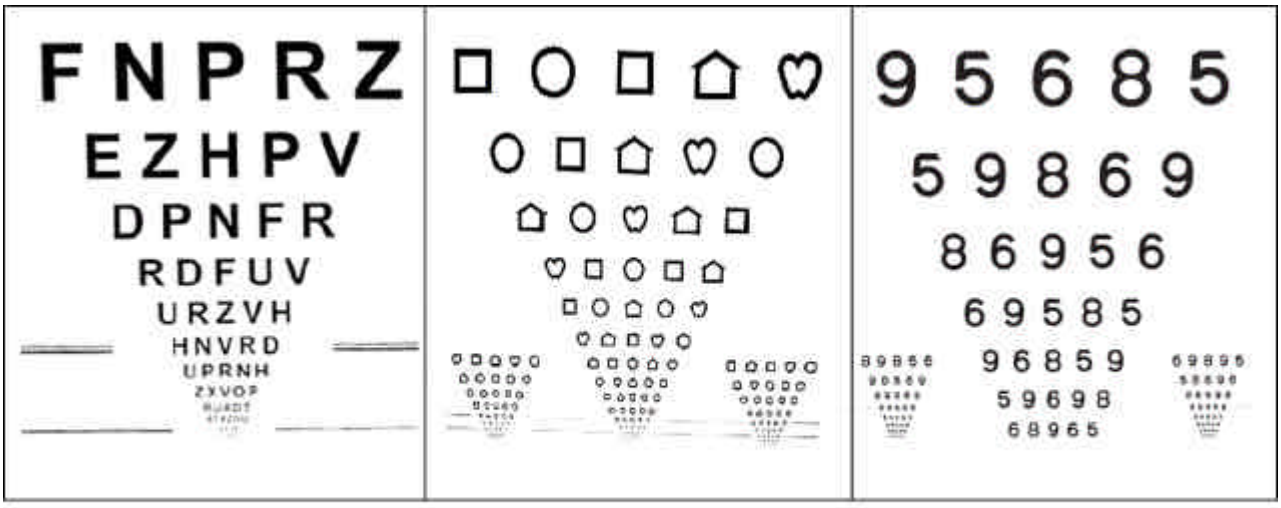


Snellen's Chart

* The Snellen's lines are not related to each other in a logarithamatic sense ie, difference in size of letter from 6/60 to 6/36, is different from 6/36 to 6/24
* Certain letters like C, G, and O etc are difficult to recognize than the other.
* Due to different number of letters in each row, the 'the crowding phenomenon' is different and is more in the lower row of letters.

To overcome these demerits, Log MAR charts has been designed. MAR (minimum angle of resolution) is arrived by dividing the denominator, by the distance at which the letters are read. Eg.6/12 or 20/40 corresponds to 2 min of arc. Logaritham of MAR is the Log MAR. Chart prepared on the basis of this, overcomes the demerits of Snellen's chart and it has got research/statistical significance.

Ian Bailey and Jan Lovie, of Australia designed the first log MAR chart in 1976. It used to be called the 'Bailey- Lovie chart'. This was modified and popularized by Ferris etal (of National Eye institute, USA) in 1982 for ETDRS.



LOGMAR Charts

In its original form, the Bailey- Lovie chart was made with 10 letters known to have relatively equal legibility. ie, D, E, F, H, N, P, R, U, V, Z .

The log MAR chart has five letters, in each row. There is a constant geometric progression in the size of letters, in the subsequent rows. Spacing between the letters and rows, is equal to the letter size. The layout is created in such a way that the crowding effect is standardized. There are are ten lines between the 6/60 row and 6/6 row. The score of 6/60line is '1' and the score of 6/6 line is '0'. Each line in the chart has five letters and the value of one letter is 0.02. Starting from 6/60(score1.0), for each line, the person reads, 0.1(ie, 5 letters X 0.02) is substracted from 1. If the person is not able to read all the letters in a row, but only some letters, 0.02 is substracted for each letter read correctly. For eg: If the person reads 8 lines fully and 3 letters in the next line- his Visual acuity will be

1-(8 X 0.1) – (3 X 0.02) = 1- 0.86 = 0.14

Score of '0' means 6/6 vision. Score below '0' ie, negative values indicate better than 6/6 vision. (ie, 6/5, 6/4 etc). Values above '1' indicate less than 6/60 vision.

To assess visual acuity properly with log MAR chart, it may take a little more time than with conventional Snellen's chart.

Gradually the log MAR charts are replacing the Snellen's chart from the rooms of Ophthalmologists and Optometrists.

ഉണരാത്ത മിഴികൾ

അമ്മിഞ്ഞയൂട്ടിയ അമ്മയെ കാണുവാനായില്ല,
തോളിലേറ്റിയ താതനേ കണ്ടില്ല
കൂട്ടുകൂടി കളിക്കുവാൻ സോദരൻ
ചാരെയുണ്ടെന്ന് കാണാതെ കേൾക്കുന്നു
കേണു ചൊല്ലിഞാൻ മാതാവിനോടന്ന്
അരുമയെന്തേ വരുന്നില്ലയെൻ മുന്നിൽ
അമ്മയെന്നോടു ചൊല്ലിയ വാക്കുകൾ
അന്നെനിക്കു മനസ്സിൽ പതിച്ചില്ല
അന്ധനായി ജനിച്ചതിൻ വേദന -
ഇന്ന് ഞാനേറെ അനുഭവിച്ചീടുന്നു.

ഈശ്വരൻ നൽകിയ പൊൻമണിക്കുഞ്ഞിനെ -
യൊന്നു കാണുവാനാകാതെ കേഴുന്നു.
എൻ വിരലിലെ സ്പർശനം കൊണ്ടു ഞാൻ -
പൊൻമണിയുടെ സൗന്ദര്യം കാണുന്നു.

കാലമേറെ കഴിഞ്ഞു പോകയെങ്കിലും,
കണ്ണുമാത്രം തുറക്കുവാനായില്ല.
മന്ന് ജീവന് ഭൂമിയിൽ വാഴുവാൻ
അന്തിനേരത്തെ പാട്ടുകൊണ്ടാവില്ല.
പാഠശാലയിൽ പോകാത്ത പൊൻമകൾ
കൂട്ടിനായി വരുന്നു കവലയിൽ
കൈപിടിച്ചു കയറ്റുന്നു വണ്ടിയിൽ
കൈകൾ കൂപ്പി യാചിച്ചീടുന്നു
അന്ധനായി പിറന്നു പോയാൽ പിന്നെ
അച്ഛനമ്മമാർ ദു:ഖിതരാകുന്നു.

എത്രനാളിനി ഭൂമിയിൽ തങ്ങണ-
മെന്നു മാത്രമെനിക്കുമറിയില്ല.
ഇത്രകാലം വസിച്ചൊരു ഭൂമിയെ -
യൊന്നു കാണുവാനാഗ്രഹമുണ്ടെനിക്ക്
തന്നു കൂടെ എനിക്കുമൊരു മിഴി
ഞാനും കണ്ടോട്ടെ ഭൂമി.... ദേവിയേ.

മണ്ണ് കണ്ട് വളർന്ന നിങ്ങൾ തൻ നേത്രങ്ങൾ -
മണ്ണിൽ നാളെ പുഴുവരിച്ചീടുന്നു

മൺമറഞ്ഞ് നാം പോയാലും നമ്മുടെ
നേത്രമെങ്കിലും വാഴട്ടെ ഭൂമിയിൽ
നേത്രദാനം നടത്തുവിൻ നാമെല്ലാം
അന്ധരില്ലാത്തതാകട്ടെ ഭൂമിയിൽ.

സന്തോഷ് കോട്ടുക്കൽ.

S/o. സുരേഷ് കുമാർ. ആർ
ഒപ്റ്റോമെട്രിസ്റ്റ്
സി.എച്ച്.സി. അഞ്ചൽ, കൊല്ലം

Biochemistry Of Vision

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The human eye belongs to a general group of eyes found in nature called "camera-type eyes." Instead of film, the human eye focuses light onto a light sensitive membrane called the retina.The study about the biochemistry behind vision started very early. Now the story gets revealed. The detection of light, smells, and tastes (vision, olfaction, and gustation, respectively) in animals is accomplished by specialized sensory neurons that use signal transduction mechanisms fundamentally similar to those that detect hormones, neurotransmitters, and growth factors. An initial sensory signal is amplified greatly by mechanisms that include gated ion channels and intracellular second messengers; the system adapts to continued stimulation by changing its sensitivity to the stimulus (desensitization); and sensory input from several receptors are integrated before the final signal goes to the brain.

How vision occurs in vertebrates …

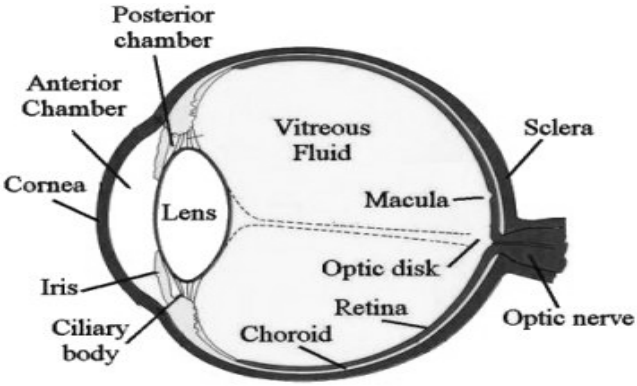
Light Hyperpolarizes Rod and Cone Cells of the Vertebrate Eye.

In the vertebrate eye, light entering through the pupilis focused on a highly organized collection of light sensitive neurons .The light-sensing cells are of two types: rods (about 109 per retina), which sense low levels of light but cannot discriminate colors, and cones (about 3 _ 106 per retina), which are less sensitive to light but can discriminate colors. Both cell types are long, narrow, specialized sensory neurons with two distinct cellular compartments: the outer segment contains dozens of membranous disks loaded with the membrane protein rhodopsin, and the inner segment contains the nucleus and many mitochondria, which produce the ATP essential to photo transduction.

Two types of vision:-

Rod vision

Rods are good for monochrome vision in poor light.Rhodopsin is the light-absorbing pigment of the rods. This G-protein-coupled receptor is incorporated in the membranes of disks that are neatly stacked (some 1000 or more of them) in the outer portion of the rod, which can sense low levels of light.



Cone Vision

Cones are used for color and for the detection of fine detail. Cones(about 3 million cones) are packed into a part of the retina directly behind the retina called the fovea**.**

Light

Light reception in the vertebrate eye.

The lens of the eye focuses light on the retina, which is composed of layers of neurons. The primary photo sensory neurons are rod cells (yellow), which are responsible for high-resolution and night vision, and cone cells of three subtypes (pink), which initiate color vision. The rods and cones form synapses with several ranks of interconnecting neurons that convey and integrate the electrical signals. The signals eventually pass from ganglion neurons through the optic nerve to the brain.

Like other neurons, rods and cones have a trans membrane electrical potential (Vm), produced by the electro genic pumping of the Na^+K^+ ATPase in the plasma membrane of the inner segment. Also contributing to the membrane potential is an ion channel in the outer segment that permits passage of either Na^+ or $Ca2^+$ and is gated (opened) by cGMP. In the dark, rod cells contain enough cGMP to keep this channel open.The membrane potential is therefore determined by the net difference between the Na^+ and K^+pumped by the inner segment (which polarizes the membrane) and the influx of Na^+ through the ion channels of the outer segment (which tends to depolarize the membrane). The essence of signaling in the retinal rod or cone cell is a light-induced decrease in the concentration of cGMP, which causes the cGMP-gated ion channel to close. The plasma membrane then becomes hyperpolarized by the Na^+K^+ATPase. Rod and cone cells synapse with interconnecting neurons that carry information about the electrical activity to the ganglion neurons near the inner surface of the retina. The ganglion neurons integrate the output from many rod or cone cells and send the resulting signal through the optic nerve to the visual cortex of the brain.

Biochemical changes during process of vision

Stages in visual transduction

First stage :- Light Triggers Conformational Changes in the Receptor Rhodopsin

Visual transduction begins when light falls on rhodopsin, many thousands of molecules of which are present in each disk of the outer segments of rod and cone cells. Rhodopsin (Mr 40,000) is an integral protein with seven membrane-spanning _ helices, the characteristic serpentine architecture. The amino-terminal domain projects into the disk, and the carboxyl-terminal domain faces the cytosol of the outer segment. The light-absorbing pigment (chromophore) 11-cis-retinal is covalently attached to opsin, the protein component of rhodopsin, through a Schiff base to a Lys residue. The retinal lies near the middle of the bilayer oriented with its long axis approximately in the plane of the membrane. When a photon is absorbed by the retinal component of rhodopsin, the energy causes a photochemical change; 11-cis-retinal is converted to alltrans- retinal. This change in the structure of the chromophore causes conformational changes in the rhodopsin molecule—the first stage in visual transduction.

Second stage:- Excited Rhodopsin Acts through the G ProteinTransducin to Reduce the cGMP Concentration

In its excited conformation, rhodopsin interacts with a second protein, transducin, which hovers nearby on the cytoplasmic face of the disk membrane,which reduces cGMP concentration.

Third stage :- Amplification of the Visual Signal Occurs in the Rod and Cone Cells

Several steps in the visual-transduction process result in great amplification of the signal.these results the generation of electrical impulses which get passed to the brain through the optic nerve and we get the vision. And finally the Visual Signal Is Quickly Terminated.

The overall process behind the vision is when light strikes either the rods or the cones of the retina; it's converted into an electric signal that is relayed to the brain via the optic nerve.The brain then translates the electrical signals into the images we see.

* * * * * * * *

Do you know……

Vitamin A is very much essential for proper vision. Lack of this vitamin causes night blindness. Humans cannot synthesize retinal from simpler precursors and must obtain it in the diet in the form of vitamin A .Given the role of retinal in the process of vision; it is not surprising that dietary deficiency of vitamin A causes night blindness (poor vision at night or in dim light).

ബാധ്യത ആർക്കും വേണ്ടേ?

-ചാർത്തൻ-

1976ൽ അഞ്ചാം പഞ്ചവത്സര പദ്ധതിക്കാലത്ത് ഭാരതത്തിൽ ആരോഗ്യ രംഗത്തും ഒരു പദ്ധതി നിലവിൽ വന്നു. ദേശീയ അന്ധതാ നിവാരണ പദ്ധതി അഥവാ NPCB. ലോകാരോഗ്യ സംഘടനയുടെ ആശയം. കാറ്റഗറി Aയിൽ 100% കേന്ദ്ര സഹായം. 2000ാം ആണ്ട് ആകുമ്പോഴേക്കും അന്ധത 1.4% നിന്ന് 0.3% ആക്കുകയായിരുന്നു ലക്ഷ്യം. 2012 ആയെങ്കിലും ഇതുവരെ അവിടെ എത്താൻ കഴിഞ്ഞെല്ലെന്നതാണ് സത്യം. നേത്രരോഗ വിദഗ്ദ്ധരുടെ ദൗർലഭ്യം അന്ധതാ നിവാരണ പദ്ധതിക്ക് തടസ്സമാണെന്ന് മനസ്സിലാക്കി അത് ഒരു പരിധി വരെ പരിഹരിക്കുന്നതിന് ഒരു പുതിയ കോഴ്സിന് കേന്ദ്ര സർക്കാർ തുടക്കമിട്ടു. ഡിപ്ലോമ ഇൻ ഒഫ്താൽമിക് അസിസ്റ്റന്റ്സ് കോഴ്സ് (DOA). സിലബസ്സ് കണ്ടാൽ തൊട്ടും. 1980 ആകേണ്ടി വന്നു ആ കോഴ്സിന് കേരളത്തിൽ ക്ലച്ച് പിടിക്കാൻ. ഭാരതത്തിലെ അന്ധതാ നിവാരണ പരിപാടിയുടെ പൂട്ട് തുറക്കുന്ന Key Personnel ആണ് ഒഫ്താൽമിക് അസിസ്റ്റന്റുമാർ എന്ന് കേന്ദ്ര സർക്കാർ പറയുന്നു.

ഇപ്പോൾ കേരളത്തിലെ അഞ്ച് സർക്കാർ മെഡിക്കൽ കോളേജുകളിൽ നിന്നും പ്രിയദർശിനി ഇൻസ്റ്റിട്യൂട്ടിൽ നിന്നും 80 കുട്ടികളും അതിന്റെ പലമടങ്ങ് സ്വയാശ്രയങ്ങളിലും ഈ കോഴ്സിന് പഠിക്കുന്നു. സർക്കാർ മെഡിക്കൽ കോളേജുകളിലെ പ്രത്യേകിച്ചും തിരുവനന്തപുരം മെഡിക്കൽ കോളേജിലെ ഈ കോഴ്സ് നടത്തിപ്പ് വെറും നാഥനില്ലാ കളരിയായി മാറിയിരിക്കുന്നു. തിരുവനന്തപുരം RIO, കോഴിക്കോട് മെഡിക്കൽ കോളേജ് എന്നിവിടങ്ങളിൽ ഒപ്റ്റോമെട്രിസ്റ്റ് ഡിഗ്രി കോഴ്സും വന്നു. അതോടെ തുടങ്ങി DOA കുട്ടികളുടെ കഷ്ടകാലം. പാരാമെഡിക്കൽ കോഴ്സുകളിൽ തന്നെ ഏറ്റവും ആകർഷകമായ ഈ കോഴ്സിന് പ്ലസ് 2വിന് ഉന്നത മാർക്ക് വാങ്ങിയവർക്ക് മാത്രമാണ് പ്രവേശനം. പക്ഷെ, പ്രവേശനത്തിനുള്ള കടമ്പ കഴിഞ്ഞാൽ തീർന്നു. കാറ്റുപോയ ബലൂൺ. ഒരു വർഷം മെഡിക്കൽ കോളേജുകളിലെ വരാന്തകളിൽ നിരങ്ങണം. മിക്കവാറും അറ്റൻഡർ പണി തന്നെ. ആരെങ്കിലും തിയറി പഠിപ്പിക്കാൻ വന്നാൽ ഭാഗ്യം. പക്ഷെ പരീക്ഷയിൽ യാതൊരു മായവുമില്ല. സ്വയം പഠിച്ചെഴുതണം എന്നായിരിക്കും. "You should observe and study" എന്ന് പണ്ടൊരു RIO ഡയറക്ടർ പറഞ്ഞത് ഓർക്കുന്നു.

ഡിഗ്രി കോഴ്സ് വന്നപ്പോൾ RIO അധികാരികൾക്ക് ഡിപ്ലോമയോട് പുച്ഛം. DOA കുട്ടികളോട് അത് അധികാരികൾ പരസ്യമായി പ്രകടിപ്പിക്കാനും തുടങ്ങി. കോഴ്സ് നിർത്തികളയും അല്ലെങ്കിൽ മറ്റെവിടേക്കെങ്കിലും ഷിഫ്റ്റ് ചെയ്ത് കളയും എന്ന് ഭീഷണിയും. അവസാനം സംഘടനയ്ക്ക് ഇടപെടേണ്ടി വന്നു.

എങ്ങനെയെങ്കിലും ആ വർഷം കഴിഞ്ഞാലും തീർന്നില്ല കഷ്ടകാലം. പിന്നെ ജില്ലാ ആശുപത്രി, സഞ്ചരിക്കുന്ന നേത്രം എന്നിങ്ങനെ. ആ ആശുപത്രികളിലൊക്കെ തൃശ്ശൂർ പൂരത്തിന്റെ തിരക്കാണ്. കൂടെ DOA ട്രെയിനിങ്ങും. ട്രെയിനീസിന് അകത്ത് കയറാൻ സ്ഥലമില്ല. അകത്ത് കടന്ന് കൂടിയാൽ തന്നെ മിക്കവാറും അറ്റൻഡർ പണി തന്നെ.. തിരുവനന്തപുരം ജനറൽ ആശുപത്രി, പേരൂർക്കട മോഡൽ ജില്ലാ ആശുപത്രി എന്നിവിടങ്ങളിൽ ചെന്നാൽ ആർക്കും ഇത് നേരിട്ട് കാണാം. പ്രായോഗിക പരിശീലനത്തിനയക്കുന്ന ഈ സ്ഥലങ്ങളിലും പരീക്ഷയും പരീക്ഷണങ്ങളുമൊക്കെ തീയറിയിൽ തന്നെ അവതരിക്കുന്നു. ജില്ലാ ആശുപത്രികളിലും സഞ്ചരിക്കുന്ന നേത്ര വിഭാഗങ്ങളിലും DOA കുട്ടികളുടെ വിലയിരുത്തൽ എങ്ങനെ നടത്തണം എന്ന് വ്യക്തമായ ഗൈഡ് ലൈൻ നിലവിലുണ്ട്. പക്ഷെ, അതിനൊ ആര് കാണാൻ? മലപ്പുറം, കോഴിക്കോട് ജില്ലകളിൽ DOAക്കാരുടെ പരീക്ഷ നടത്തിപ്പുമായി ബന്ധപ്പെട്ട് ഉണ്ടായ 'ഉടക്കിലും' അവസാനം സംഘടനയ്ക്ക് ഇടപെടേണ്ടി വന്നു.

പക്ഷെ, സ്വാശ്രയക്കാർക്ക് ഇത് പ്രശ്നമേ അല്ല. ഇന്റേണൽ അസസ്സ്മന്റ് പോലുള്ള ലൂപ്പ് ഹോളുകളിൽ പിടിച്ച് പ്രാക്ടികൽ മാർക്കൊക്കെ സ്വന്തമായി ഇടും. സർക്കാർ കുട്ടികളുടെ മാർക്കിന്റെ ഗതി ട്രെയിനിംഗ് എടുക്കുന്ന സർക്കാർ ആശുപത്രിയിലെ ഡോക്ടർ തലവന്റെ മനോഗതം പോലെ തന്നെ. ട്രെയിനിംഗ് കൊടുക്കുന്ന ഒപ്റ്റോമെട്രിസ്റ്റിനെ അതേൽപ്പിക്കാൻ പലർക്കും ഒരു കുറച്ചിൽ.

ഇങ്ങനെയൊക്കെ തന്നെ മതിയോ മെഡിക്കൽ വിദ്യാഭ്യാസ ഡയറക്ടറുടെ കീഴിലുള്ള ഒരു പ്രധാന കോഴ്സ് നടത്തിപ്പ്? സിലബസ്സ് കൈയ്യിലുണ്ടെങ്കിൽ ഒന്നൊടുത്ത് വായിച്ചു നോക്കണം. ശരിയായി ഈ കോഴ്സ് നടത്തിക്കാൻ ആർക്കും ബാധ്യതയില്ലേ?

* * * * *

സർക്കാർ മെഡിക്കൽ കോളേജുകളിൽ BSc (ഒപ്റ്റോമെട്രി) കോഴ്സ് തുടങ്ങിയിട്ട് 2 വർഷം കഴിഞ്ഞു. അതും DOAയുടെ മറുപുറമായി തന്നെ മുന്നേറുന്നു. 2 വർഷം കഴിഞ്ഞിട്ടും ലാബ് സൗകര്യം ഉണ്ടാക്കാൻ കഴിഞ്ഞിട്ടില്ല. പഠിപ്പിക്കാൻ ആളെ നിയമിക്കാനോ, ട്യൂട്ടർ തസ്തിക ഉണ്ടാക്കാനോ കഴിഞ്ഞിട്ടില്ല. ട്യൂട്ടർ നിയമനത്തിലെ സ്വജന പക്ഷപാതവും വ്യക്തി താത്പര്യങ്ങളും ഈ കോഴ്സിനേയും തകർക്കാൻ തുടങ്ങിയിരിക്കുന്നു. ഒരു ഡിഗ്രി കോഴ്സ് പഠിപ്പിക്കാൻ ട്യൂട്ടർമാരില്ലാത്ത ഏക കോഴ്സും ഇപ്പോൾ BSc ഒപ്റ്റോമെട്രി ആയിരിക്കും. ഈ കോഴ്സിന്റെ നടത്തിപ്പിലും ജൂനിയർ - സീനിയർ അടി 'പൊടിഞ്ഞു' നടക്കുന്നു. ഇച്ഛയുണ്ടെങ്കിൽ ശരിയായി കോഴ്സ് നടത്താവുന്നതേയുള്ളു. 'വേണമെങ്കിൽ ചക്ക വേരിലും കായ്ക്കും'. ട്യൂട്ടർ തസ്തിക ശൃഷ്ടിക്കണം. മതിയായ യോഗ്യതയുള്ളവരെ അതിൽ നിയമിക്കണം. അല്ലാതെ മറ്റുള്ളവരുടെ പ്രമോഷൻ പോസ്റ്റ് തട്ടിപ്പറിക്കുകയോ ഉദ്യോഗസ്ഥരെ തരം താഴ്ത്തുകയോ, ചെവിക്ക് പിടിച്ച് പുറത്താക്കുകയോ ചെയ്തിട്ട് വേണോ പുതിയ ട്യൂട്ടർ തസ്തിക ഉണ്ടാക്കാൻ. പ്രതികാരം തീർക്കാനാണെങ്കിൽ വഴി വേറെ നോക്കണം. ഭാരതത്തിൽ സുപ്രീമസി ഭരണഘടനയ്ക്കാണ്.

Duochrome Test

Anju T N
Optometrist

We can see two rows of black letters in red and green background in the lower portion of the Snellen's illuminated visual acuity chart. These are read by the patient while doing the duochrome test. The letter sizes are from 6/12. So if vision is less than 6/12 duochrome test is not done.

It is a subjective test, means it is based on how the patient is responding. This test is used to refine spherical endpoint while doing acceptance. For example if the patient is reading 6/6 line with +2.00 DS, +1.50 DS, and +1.00DS, this test helps to easily decide which lens should be given. Duochrome test helps to balance two eyes with regard to chromatic variation in refraction.

PRINCIPLE

It is based on chromatic aberration. When light passes through glass lens, prisms etc, it get dispersed or split into colours. Light with different wavelengths are bend to different extent. Longer wavelength is refracted less. So each colour is focused at different point. Blue-green has shorter wavelength and red has longer wavelength. Green is refracted more than red. So green light focuses slightly in front of the retina and red behind the retina. So hyperopes see green end of spectrum well whereas myopes see red end of spectrum well. If over plused red light focus on retina and it becomes clearer. If over minused green light focus on retina and it becomes clearer.

PROCEDURE

a) **Hyperopes**: Suppose RE unaided vision is 6/18 and we got a dry retinoscopy value of +3.00 in both meridians. So we can start fogging with +3.00DS in trial frame and occluding left eye. While placing +3.00 DS we need to tell patient that letters in the chart will not be clear now and it will become clearer slowly. Ask the patient how much he can read. Reduce in terms of +0.50 DS. Always make sure one lens is put and then only the other one is removed (in case of plus lenses) so that accommodation is relaxed. From +1.50 reduce in terms of +0.25 DS. Suppose patient reads 6/6 line with + 1.75 DS, put off room illumination and ask patient whether letters in green is better or red is better. If patient says letters in red are better we need to reduce power. With +1.25 DS patient says letters in red and green are equally clear. It means duochrome is balanced. We need to prescribe this power. If we reduce plus power, ie at + 1.00 DS patient can read 6/6 but duochrome is green better, then hyperopia is under corrected. If cylinder is also present, put the cylinder power in the trial frame along with the fogging lenses.

b) **Myopes**: Start from the low minus power and slowly increase it. For example RE unaided vision is 6/18. Suppose dry retinoscopy shows -2.00 in both meridians. Start with -0.50 DS. Ask patient how much he can read. As before we need to tell the patient before that vision will slowly become clearer. Then put -1.00 DS after removing -0.50 DS from the trial frame (in case of minus lenses). Ask the patient how much he can read. Give the minimum minus power with maximum vision. Once 6/6 vision is got, do duochrome test. In case of myopes duochrome should always be red better, ie patient should read letters in red better. If over minused letters in green become better.

It is ideal to do duochrome test for optimal refractive correction. Finally we need to do the test binocularly also.

AUTOMATED REFRACTION

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The human eye is an organ which reacts to light for several purposes. As a conscious sense organ the mammalian eye allows vision. Rods and Cones in the retina allow conscious light perception and vision including colour differentiation and the perception of depth.

Measurement of accurate Visual Acuity has been a challenging procedure to Optometrists and Ophthalmologists. Correction of refractive errors and prescription of apt and comfortable glass or contact lenses is very important in the practice of Optometry today.

Refraction being the most commonly performed optical procedure has been widely developed. Though the conventional technique of retinoscopic refraction is an excellent method of objective refraction. Retinoscopic Refraction is a time consuming procedure and not every practitioner manages to accomplish it accurately.

Refractometry is an alternative method of finding out the error of refraction by use of optical equipment called refractometer or optometer.

Optical Principles used in Refractometers

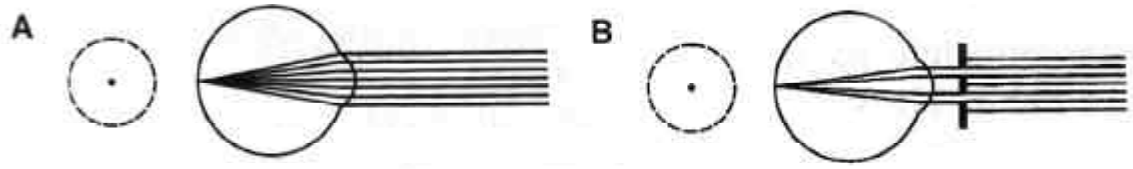
Optometers are essentially based on following two principles.

1.**The Scheiner principle (Scheiner double pin-hole refraction)**

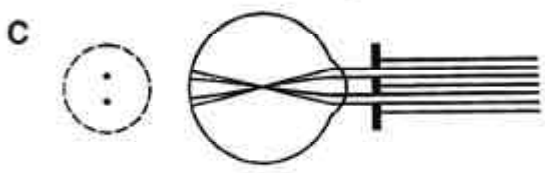
Scheiner in 1619 observed that refractive error of the eye can be determined by using double pin-hole apertures before the pupils.

Following were his observations:

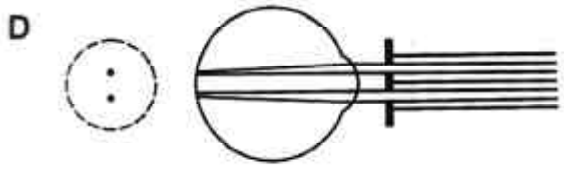
a. The parallel rays of light entering the eye from a distant object, which are normally focused on a point on the retina in an emmetropic patient, are limited to two small bundles when double pin hole apertures are placed in front of the pupil



b. In a Myopic eye the two ray bundles cross each other before reaching the retina and two small spots of light are seen.



c. In a hypermetropic eye the ray bundles are intercepted by the retina before they meet and thus again two small spots of light are seen.

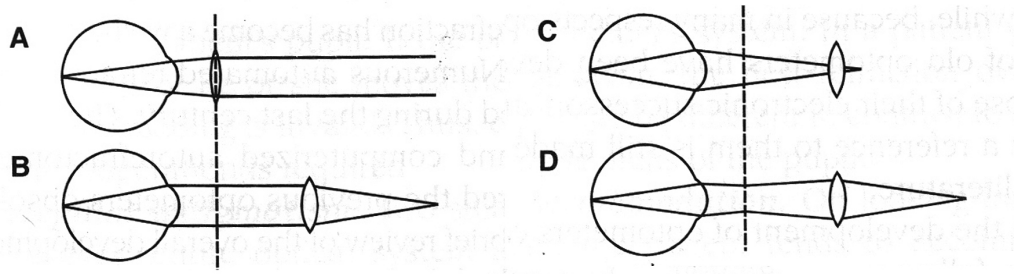


These two points of light can be coalesced to a single point by moving the double pin-hole to the far point of eye.

Thus from the far point of the eye the refractive error of the eye can be determined.

2**. The Optometer Principle**

Porter field in 1759, coined the term optometer to describe an instrument for measuring the limits of distinct vision.The optical principle on which this instrument was based is now known as the optometer principle. The principle permits continues variation of power in the refracting instruments.

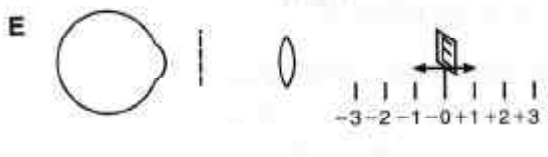


As shown in (fig A), the autorefractometers based on this principle use a single converging lens placed at its focal length from the eye (or the spectacle plane) instead of interchangeable trial lens.

Light from the target on the far side of the lens enters the eye with vergence of different amounts, ie, [zero(fig B),minus (fig C) or plus (fig D) depending on the position of the target.

The vergence of light in the focal plane of the optometer lens is linearly related to the displacement of the target.

A scale with equal spacing can thus be made which would show the number of diopters of correction.



Development of optometers (in brief)

Development of optometers can be grouped as follows:

* Early subjective optometers
* Early objective optometers
* Modern Objective Autorefractometers
* Modern Subjective Autorefractometers

An early refractometer
(Rodenstock)

Early subjective optometers

The earliest Optometers developed during 1895- 1920 were all subjective.These optometers required the patient to adjust the instrument for best focus or best alignment of parts of the target. These subjective Optometers were unsuccessful because of the instrument accommodation.

Examples of early subjective Optometers:

1.Badal Optometer

2.Young's Optometer

Early Objective Optometers

Objective Optometers were developed to offer an alternative means for evaluating the optical correction of the eye. These instruments were all based on the optometer principle or scheiner principle

Eg: 1 Rodenstock Refractometer

2 Zeiss- Jena coincidence refractometer

3 Finchain coincidence optometer

Limitations of Optometers:

Three basic factors responsible for the limited acceptance of optometers in clinical refraction include the following:-

1. Alignment problem

As per the requirement of scheiner's principle, both pin-hole apertures must fit within the patient's pupil. If the patient's fixation wanders or he moves the head even slightly, the reading is invalid. Thus, considerable patient co-operation is required.

2. Irregular Astigmatism

Two small apertures of the eye's entire optical system are used by the Scheiner's system. In a patient with irregular astigmatisam, the best refraction over the whole pupil may be different in contrast to the two small pinhole areas of the pupil.

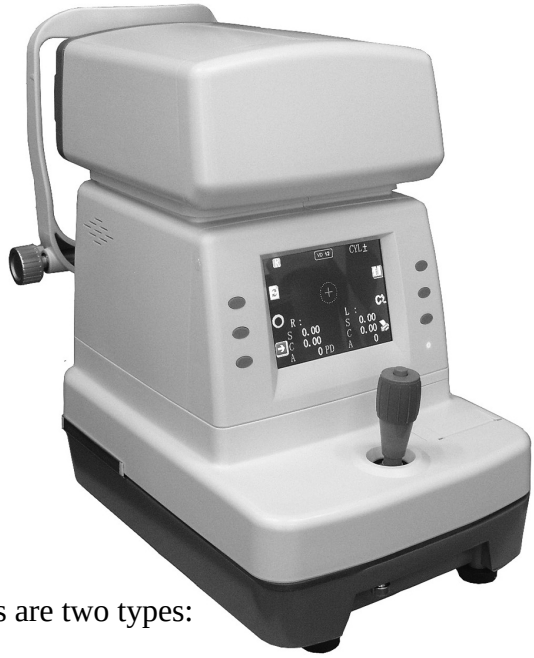
3. Accomodation

On looking into the instrument, the patient tends to accommodate. This is known as instrument Myopia and this alters the actual refractive status of the patient. Factors affecting accommodation include- attention, fatigue, direction of gauze, illumination, image detail, blur of the retinal image and psychological factors.

AutoRefractor or Automated Refractor or ARM

It is a micro computer controlled machine used during an eye examination to provide an objective measurement of a person's refractive error and prescription for glasses or contact lenses.

MODERN AUTOREFRACTOMETERS



* Rapid development in electronics and micro computers, a number of innovative methods and instruments for automated clinical refraction have appeared since 1960.

* Efforts have been made to eliminate the limitations of old refractors.

Based on operational method used, electronic optometers fall into three main classes:

* **Analysis of image quality** (Dioptron, Canon, Hoya)

* **Retinoscopic Scanning** (Ophthalmetron, Humpry, Nikon 5000 and 7000)

* **Scheiner Disc Refraction** (Nidek, Topcon)

Based on instrument design and method the instruments are two types:

1. Objective Refractometers
2. Subjective Refractometers

Objective instrument design and methods

Patients' whole pupil is used in this system, thus avoiding some of the alignment and partly the irregular astigmatism problems. The Operator focuses a single spot of light on the patient's retina and measures the astigmatism by successively focusing the two focal lines.

Automated infrared optometers

- Refraction is performed automatically by using infrared light which is invisible to patients.

- A visible fixation light is provided in each instrument in order to control the patients fixation and accommodation.

- Wavelength of infrared radiation 800-900nm.(Nikon, Canon, Nidek,Humphrey, shin- Nippon)

* Photo Refraction

* Electrophysiologic methods

Subjective instrument design and methods

* Laser speckle pattern refraction

* Multimeridional refractometry

* Computer – actuated refraction

* Instruments having continuously variable sphero cylindrical power

Both objective and subjective modern autorefractometers are available commercially

General comparison of subjective and objective instruments

	Objective	subjective
1	**Source of light** Invisible infrared light used to perform refraction	Visible light using.
2	**Time required for refraction** 2-4 minutes	4-8 minutes
3	**Information provided** NOT provide visual acuity except Humphrey ARM	Supply more information Corrected visual acquity obtained
4	Patient co-operation factors * Require less co-operation * Children above 5 years old can be refracted.	* Require patients co-operation * Children above 8 years old can be refracted.
5	**Ocular factors:** Ocular diseases may limit the performance of the refractometers as below. * Give better results in the presence of macular diseases with clear ocular media. * In the presence of hazy ocular media causing a drop of visual acuity of more than 6/18, the objective refractometers not function properly * Performance equal in the presence of hazy ocular media which cause decreased Snellen's Visual acuity up to 6/18	* NOT giving better results when compared with objective refractometers * Rough refraction often still may be obtained. * Performance equal in the presence of hazy ocular media which cause decreased Snellen's Visual acuity up to 6/18
6	**Over refraction capability** The over refraction in patients using spectacles, contat lenses or IOL is comparatively difficult	No such problem
7	**Expected results** Provide only preliminary refractive findings	Provide refined subjective results.

Objective Autorefractometers

* The automatic infrared optometers have aroused the greatest interest

* These instruments perform the refraction automatically, using infrared light which is invisible to the patients.

*A visible fixation target is provided in each instrument to help control the patients fixation and accommodation.

All current infrared objective optometers used any one of the following basic principles:

1. Scheiner disc principle- eg. Topcorn, Nidek

2. Grating focus principle- eg. Dioptron, Canon

3. Retinoscopy illumination- eg. Nikon, Ophthalmetron

Note:-

* **O**bjective infrared **ARMs** perform sphero cylindrical refraction

* These instruments finds best focus in 2 to more meridians

* Scans 180‘-360‘ in 0.5 to 2 seconds

* Micro computer calculates the refractive correction by fitting a sine 2 curve to the best data points.

To conclude:

*The Scheiner principle and optometer principle and their modifications have been used time and again to automated refraction have become a well established technique.

*The automated refraction technique is quick, simple , painless and repeatable.

*No feedback is required from the patient during this process.

*Within seconds an approximate measurement of a person’s prescription can be made by the machine.

*Useful when dealing with non- communicative people such as young children or those with disabilities.

*All ARMs now use the fogging technique to relax accommodation prior to objective refraction.even with this fogging technique , micro fluctuations in accommodation occur up to 0.50 Ds.

Recent studies report that autorefractometer measurements without application of cycloplegia can result in significant overestimation of Myopia. The result of autorefraction post refractive surgery and in eyes with corneal distortion should always be viewed with suspicion.Aberrrometers may help to provide a better starting point for refraction in these instances.

BINOCULAR SINGLE VISION

Binoy R
Optometrist
PHC Manalur

The image formed on the retina of each eye is fused in to one and a single image is perceived by the subject, then the person is said to have binocular single vision. There are three essential necessary for attaining BSV.

1. There must be a healthy macula in each eye sub served by an efficient focusing mechanism so that two clear and equally distinct images can be formed.

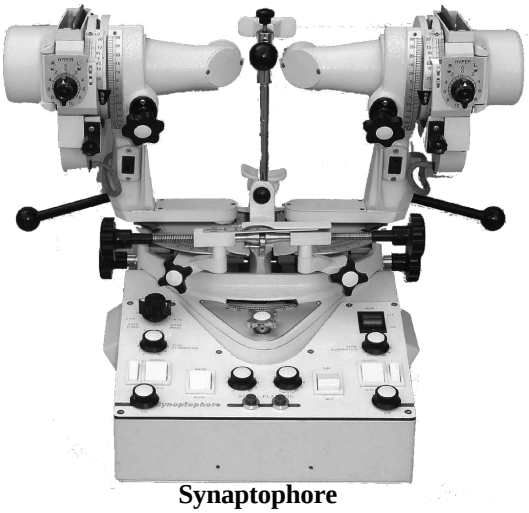
2. Normally functioning set of ocular muscles which are competent to bring about fine adjustments which is necessary.

3. Efficiently working nervous mechanism which can receive the two impressions and blend them psychologically in to one.

GRADES OF BSV

1.Simultaneous perception

The ability to see the objects clearly with each eye is called simultaneous perception and if the visual axis is in proper alignment the two images are superimposed into one. This can be tested by an instrument called synaptophore. Two slides of dissimilar objects are presented before each eye and if the person has SP he can see both pictures.



Synaptophore

2.Fusion

The ability to fuse two dissimilar objects into one is called fusion. A composite picture is presented so that each half of it is complete; the part missing in one eye is accurately superimposed by the other when fusion is present. If there is no fusion as soon as the axes are moved from the position of parallelism the picture becomes broken up. The extent to which tubes of synaptophore may be separated or brought together can be taken as a measurement of the development of fusion faculty.

The amount of effort which can be put forth to maintain fusion can also be measured by using prisms. It is found that very large prisms up to 30^{o} can be overcome by convergence. Weaker ones from 10^{o} to 15^{o} can be overcome by divergence. 2^{o} to 4^{o} can be overcome by vertical deviation of the eye. This power of suppressing an artificially produced diplopia is called verging power.

3.Stereopsis

It is otherwise called three dimensional view. It is the process in visual perception leading to the perception of depth from two slightly different projections of world into the retinas of the two eyes.The difference in the two retinal images are called horizontal disparity arise from the eyes different positions in the head. Stereopsis is commonly referred to as depth perception.Stereopsis was first described Charles wheatson in 1838.

Field of view and Eyes movements.

Some animals have their two eyes positioned on opposite side of their head to give widest possible field of view. Eg. Rabbits and buffaloes. In human beings ,eagle ,wolves etc, the two eyes positioned on the front of their head thereby allowing for BSV and reducing field of view in favor of stereopsis.

In whales, the two eyes are positioned on opposite sides of their heads. In animals with forward facing eyes the eyes usually move together. When the eyes movelaterally in the same direction it is called version. When the eyes move in the opposite direction, it is called vergence.

BINOCULAR INTERACTION

1.Pupillary Diameter: Light falling in one eye affects the pupil in both eyes.

2.Accommodation and convergence are linked by a reflex, so that one evokes the other.

3.Intraocular transfer: The state of adaptation of one eye can have a small effect on the state of light adaptation of the other.

DISORDERS OF BSV.

1. PHORIA: Slight difference in the length or insertion, position or strength of the same muscles (eom) in the two eyes can lead a tendency to deviate from normal position especially when one is tired. One way to reveal it is cover uncover test.

2. Tropia: Manifest squint is more problematic disorder of binocular vision.

3. Anisometropia: It is a condition wherein the refraction of the eyes is unequal. Vision in anisometropia may be alternating, binocular or may be uniocular. In anisometropia binocular vision is rarely perfect and the attempt at fusion frequently brings on symptoms of accommodative asthanopia.

With higher degrees of error fusion is impossible and one of two alternatives occurs.

1.Alternating vision in which case each of the two eyes is used on at a time. If the defect in one eye is high and if its visual acuity is not good, it may be excluded altogether from vision at an early stage in life.

2.Aniseikonia: a condition wherein the size and shape of the images in the two eyes are unequal. It may be due to anisometropia or may by a difference in the distribution of the retinal elements.

DIPLOPIA

Diplopia is the simultaneous perception of two images of a single object that may be displaced vertically or horizontally in relation to each other. It is usually the result of impaired function of the EOM, due to mechanical problems , disorders of the neuromuscular junction or disorders of the cranial nerves 3rd, 4th , and 6th that stimulates the muscles and occasionally disorders involving the supra nuclear oculomotor pathways.

Diplopia is often one of the first signs of a systemic disease, particularly to a neurological or muscular process.

Classification

1.Binocular, 2. Monoocular, 3. Temporary, 4. Voluntary

If the fovea of one eye corresponds to the fovea of the other images, falling on the two foveas are projected to the same point in space.

Binocular diplopia arises as a result of misalignment of the two eyes relative to each other occurs as in tropia. In such cases while fovea of one eye is directed at the object of regard the other eye an extra foveal area of the retina, is directed towards the object of regard.

The image falling on the fovea is seen as being directly ahead, while those falling on retina outside fovea may be seen as above, below,right, or left of straight ahead depending upon the area of retina stimulated. Thus, when the eyes are misaligned, the brain will perceive two images of one target object producing diplopia/double vision.

In an attempt to avoid double vision brain can sometimes ignore the image from one eye, process known as suppression. The ability to suppress is more in childhood when the brain is still developing. This ability to suppress can prevent the proper development of vision in the affected eye resulting in amblyopia.

Monocular Diplopia: When a patient perceives more than two images with one eye it is called monocular polyopia.It occurs in conditions like keratoconus, subluxated lens,structural defect within the eye etc.

Temporary Diplopia can be caused by alcohol intoxication or head injuries such as concussion. It can also be caused by a tired EOM in phoria.Side effects of some medicines can also cause temporarydiplopia.eg. epileptic drugs phenytoin,zonisamide,and hypnotic drugs like zolpidem can cause temporary diplopia

Reference:-

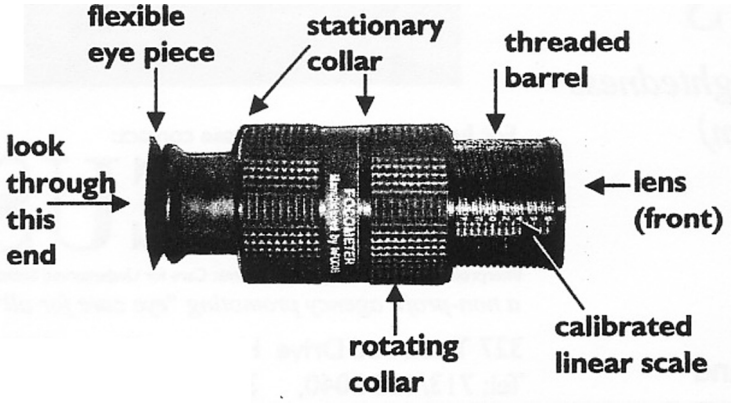
Duke Elder's Practice of Refraction
Wikipedia, the free encyclopedia
A.K.Khurana, Text book of Ophthalmology

OPTOTOOLS - FOCOMETER

A focometer is an instrument that measures refractive errors and is intended to provide rural or economically disadvantaged populations spherical eyeglass prescriptions without the need for complicated protocols, expensive equipment, or electricity. The focometer is monocular and hand-held, and is normally used in natural lighting. Patients rotate a collar on the focometer until the best focus is achieved. The individual's refractive power is then read off a linear dioptre scale.

The focometer was developed by Drs. Ian Berger and Larry Spitzberg at the University of Houston, College of Optometry in Houston, Texas, to provide a simple, inexpensive means for measuring refractive error in human vision. The portable, hand-held instrument is highly appropriate for use in remote and poor areas.

Focometers measure spherical refractive errors. Astigmatism can also be measured using a "clock target" supplied with the device. A study has found, however, that the focometer is less effective for identifying astigmatism than an autorefractor, and that its axis accuracy is limited to 15°.



The advantages of a focometer over other methods for use in developing countries are that it is lightweight, compact, relatively inexpensive, fairly quick, and easy to use with minimal training. A clinical trial compared the repeatability, validity, and ease of use of the focometer with an autorefractor. It found that the focometer results were within 0.75 dioptres of the autorefractor value 84% of the time.

The design eliminates the need for many pieces which could be lost or broken and may be difficult to replace. Based on Badal optics, the focometer allows the patient to view a real, unmagnified target, and bring it into focus, with a direct reading of spherical correction on a linear diopter scale.

Strengthening Keratoconus Management

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Keratoconus is a visually debilitating ocular condition, characterised by progressive degeneration of the corneal stroma. At present, the pathogenesis of the disease is poorly understood and, until recently, there has been no effective treatment for delaying the progression of the disease.

Corneal collagen cross-linking (CXL) treatment, using riboflavin and ultraviolet (UV) light, is a novel procedure that has been developed to address this clinical need. We review the clinical characteristics of keratoconus and provide an evidence-based update on the efficacy and safety of CXL.

Keratoconus, first described in detail in 1854,[1] derives from the Greek words Kerato (cornea) and Konos (cone); it is a progressive, bilateral but typically asymmetric, non-inflammatory ectasia of the cornea that is characterised by progressive thinning of the axial corneal stroma. The condition has both physical and chemical markers that arise from the cellular malformation of the corneal stroma. Epidemiological studies indicate that the prevalence of keratoconus is 5.4 per 10,000; the condition can occur in all ethnic groups with no male or female preponderance.[4] While keratoconus typically occurs in isolation, the most common recognised systemic associations are generalised atopy,[5] Down syndrome[6] and Marfan's syndrome.[7] Ocular comorbidities include Leber's congenital amaurosis[8] and retinitis pigmentosa.[9] Keratoconus can vary significantly in its clinical course, but classically manifests at puberty and is progressive until the third or fourth decade, when it usually arrests. It may however, commence later in life and progress or stabilise at any age.[4]

Approximately 15 per cent of affected individuals will require a corneal transplant, due to inadequate vision correction with contact lenses, contact lens intolerance and/or visually debilitating corneal scarring.[10] At present, the aetiology of keratoconus is uncertain but may involve genetic, biochemical and environmental factors.

Clinical Features

The clinical hallmark of keratoconus is progressive thinning of the axial corneal stroma, resulting in protrusion of the corneal apex to assume a conical shape. The thinner apex is typically downwardly displaced, leading to irregular astigmatism and visual distortion. Although keratoconus affects both eyes it can be highly asymmetric, with the condition far more advanced in one eye compared with the other.

Patient symptoms are variable and depend upon the severity of the disease. Early symptoms typically include monocular diplopia, mild photophobia and a history of deteriorating vision with spectacles.[11,12] Advanced keratoconus is associated with more significant visual impairment and displays such symptomology as: poor best-corrected spectacle acuity, haloing around lights, a rapidly changing subjective refraction and generalised asthenopia.

Fortunately, much of this visual distortion can be corrected with appropriately-fitted rigid gas permeable lenses which are the mainstay of optometric management. Hybrid, mini-scleral and scleral contact lenses are further contact lens modalities that may be appropriate for patients with keratoconus.

Biomicroscopic indicators of keratoconus may include one, or more, of the following signs :

1. Central or paracentral corneal thinning (usually inferior or infero-temporal);

2. A complete, or incomplete, iron line at the base of the cone (Fleischer ring) is observed in approximately half of patients; it represents the accumulation of iron deposits from the tear film onto the cornea as a result of severe corneal curvature changes and/or modification of the normal epithelial slide process;

3. Fine vertical lines located in the deep corneal stroma and Descemet's membrane that parallel the axis of the cone (Vogt's striae), produced by compression of Descemet's membrane;

4. Increased visibility of the corneal nerves;

5. Apical corneal scarring, which may be due to ruptures in Bowman's membrane or the result of a flat-fitting contact lens;

6. A V-shaped deformation of the lower lid produced by an advanced, ectatic cornea in down-gaze (Munson's sign);

7. Corneal hydrops, caused by an acute rupture in Descemet's membrane which results in a sudden, abnormal accumulation of fluid in the corneal stroma. Corneal hydrops is estimated to occur in 5 to 15 per cent of keratoconus patients. The corneal oedema may take weeks to months to resolve, resulting in residual stromal scarring that is accompanied by a relative flattening of the corneal curvature; this can in some cases, allow contact lenses to be more readily fitted.

Clinical indicators that can assist with a diagnosis of keratoconus include:

1. Subjective refraction: myopia with, or without, high astigmatism (typically oblique or against the rule) and near acuity better than expected from refraction and age, due to the multifocality of the cornea;

2. Retro-illumination: scissoring reflex on retinoscopy and/or the 'Charleux' oil-droplet sign evident with ophthalmoscopy;

3. Pachymetry: reduced central corneal thickness (< 450µm is suspicious of keratoconus, however due to significant variability of central pachymetry measures within the normal population it cannot be solely relied upon);

4. Photokeratoscopy: distortion or steepening of keratometry mires centrally or inferiorly;

5. Videokeratoscopy (corneal topography): is regarded as useful for both the detection and monitoring of keratoconus. This technique, which provides a graphical representation of the physical configuration of the cornea, can accurately reveal the shape, size and location of the cone.

While corneal topography is the most commonly used device to accurately detect and monitor keratoconus, the Pentacam instrument (Oculus, Wetzlar, Germany) is an alternative optical instrument for assessing the anterior ocular surface. The Pentacam takes multiple images of the cornea at different angles using a rotating camera. As such, it allows for the evaluation of disease severity and progression based upon changes in corneal volume and anterior chamber angle, depth and volume.

Classification of Keratoconus

Several classification systems for keratoconus have been proposed in the literature. These systems categorise the condition based upon different criteria, including corneal morphology, disease evolution, ocular signs and corneal indexes.

Morphological Classification

The classification of keratoconus using morphological criteria is based upon corneal topographical data; three major sub-groups of keratoconus have been described (Figure 2):

- Centred (nipple) cones are small (i.e. a cone diameter d" 5mm), round in shape and are positioned central or just inferior to the visual axis; the peripheral cornea remains relatively normal in curvature. Approximately 45 per cent of cones are reported to be of this morphology. Correction with rigid gas permeable lenses is relatively straightforward owing to the centralised location of the corneal steepening.

- Oval (sagging) cones are larger in size (i.e. a cone diameter >5mm), and are displaced inferonasally or inferotemporally, inducing high degrees of irregular astigmatism. It is recognised that approximately half of cones are of an oval morphology. Since contact lenses tend to naturally centre over the apex of the cone, centration and adequate pupillary coverage can be more difficult to achieve than for the centred cones.

Globus cones are the least common morphology, constituting less than five per cent of presentations. As the cone involves at least 75 per cent of the cornea, these are the most challenging cones to fit with contact lenses; typically, large intra-limbal or sclera lenses will be required.

Disease evolution

Disease evolution refers to the classification of keratoconus based upon the severity of the clinical signs.[13]

Stage one is defined as forme fruste or sub-clinical keratoconus and is characterised by normal slit lamp findings, best-corrected spectacle acuity of 6/6 (or better) combined with the presence of subtle corneal irregularity on corneal topography. Stage two represents early keratoconus, in which mild corneal thinning may be evident on biomicroscopy, but corneal scarring is absent. In Stage three (moderate keratoconus), slit lamp signs such as Vogt's striae and Fleischer's ring are more common. Best-corrected spectacle acuity is reduced to below 6/6, with irregular astigmatism between 2.00 and 8.00 dioptres. Stage four is the most severe form of keratoconus, with a maximal curvature value in excess of 55.00D, the presence of stromal corneal scarring, severe corneal ectasia and acuity below 6/7.5 even with contact lens correction.

Index-based systems

Several index-based systems have been described for the detection of keratoconus (Table 1). These systems have the advantage of being objective and are designed to be sensitive for detecting, early sub-clinical forms of the disease.

Corneal Collagen Cross-linking

Corneal collagen cross-linking (CXL) is the most recent clinical intervention that demonstrates promise for arresting the progression of keratoconus. The treatment was inspired by the German ophthalmologist Professor Theo Seilor in the 1990s, whom during a visit to his dentist noted the use of ultraviolet (UV) radiation to harden a synthetic filling and proposed that a similar process may have the potential to stiffen a weakened keratoconic cornea. The procedure aims to increase the biomechanical stability of the keratoconic cornea, thereby potentially slowing, or even halting progressive corneal ectasia and postponing, or even negating, the need for future corneal transplantation.

Mechanism of Action

In the normal cornea, covalent bonds (or cross-links) exist between collagen fibrils, imparting structural integrity and rigidity to the tissue. In keratoconus, a reduced number of cross-links between the collagen layers reduce the mechanical strength of the cornea by up to 30 per cent. CXL involves the use of the photosensitisation agent riboflavin (Vitamin B2) and ultra-violet A (UVA) irradiation, to induce photo-oxidative cross-linking of the collagen within the corneal stroma in vivo.

Riboflavin has an absorption peak for UVA at a wavelength of approximately 370nm. When the riboflavin-saturated cornea is exposed to radiation of this wavelength, the riboflavin molecules fluoresce, resulting in the generation of singlet oxygen and superoxide radicals; these reactive oxygen species lead to the formation of covalent bonds between collagen molecules. Studies indicate that CXL induces physical changes within the cornea that include: an increase in Young's modulus, increased bending stiffness, larger collagen fibre diameter[22] and an enhanced resistance to enzymatic degradation.

Clinical Technique

The CXL treatment is conducted under sterile conditions in an operating theatre. Pre-operatively, the patient's eye is anaesthetised with topical anaesthetic (e.g. Proxymetacaine hydrochloride 0.5 per cent drops) and pilocarpine is instilled to induce pupillary constriction in order to minimize UV exposure to the lens and retina. The central 7-9mm of corneal epithelium is then debrided to allow the diffusion of riboflavin into the corneal stroma.

A 0.1 per cent riboflavin solution (10mg riboflavin-5-phosphate in 10ml dextran 20 per cent solution) is applied to the eye approximately every five minutes, commencing five minutes prior to the first irradiation. The irradiation is performed from a one centimetre distance for 30 minutes using UVA at 370nm and an irradiance of $3mW/cm^2$. The required irradiance is carefully controlled in each patient directly before the treatment to avoid a potentially dangerous UVA overdose.

At the completion of the procedure, the patient is commenced upon topical, broad-spectrum antibiotics and a

bandage contact lens is applied to the eye. To minimise the ocular inflammatory response, topical corticosteroid eye drops are commonly used upon the third post-operative day, when the bandage contact lens is also removed.

Clinical Results

Cross-linking pre-clinical studies began in 1993 and included laboratory work and experimental trials. The first in vivo clinical study to be published on CXL was conducted by the German research group, Wollensak and colleagues in 2003.[24]

Since this pilot study, a growing number of papers have described the clinical efficacy of CXL for keratoconus (Table 2). Although differences exist in the study methodology, inclusion and exclusion criteria, treatment parameters and outcome measures, data from all of these studies consistently demonstrate varying degrees of improvement in visual acuity and a reduction in maximal keratometry values with CXL treatment.

The world's first prospective, randomised, controlled clinical trial on CXL for keratoconus was conducted by the Centre for Eye Research Australia (CERA) at the Royal Victorian Eye and Ear Hospital, Melbourne, Victoria.

Commencing in 2006, this study involved the recruitment of one hundred patients who had demonstrated clinically-significant progression of their keratoconus over the prior six to twelve months. The preliminary findings of this study, published in 2008, indicated statistically significant differences between the CXL treatment and control (untreated) groups for changes to maximum (steepest) simulated keratometry values and best spectacle-corrected acuity.[26] A larger, multi-centre treatment trial is currently being conducted in the USA; the findings of this important study are eagerly anticipated.

Patient Selection

With the advent of CXL, it is now more important than ever that optometric management of keratoconus involves the careful documentation and monitoring of younger patients (less than 35 years or age) with established disease; these patients are at the highest risk of progression and therefore may benefit most from CXL. Moreover, as primary eye care providers, optometrists play a pivotal role in identifying patients with the early signs and symptoms of keratoconus, including sub-clinical manifestations, through appropriate clinical examination and corneal topography. Within practices that are not equipped with a corneal topographer, referral for corneal mapping is warranted in patients with a family history of the condition in order to identify keratoconus suspects at an early age. Younger patients may require reviews every few months if there is a suspicion of progression or incipient keratoconus.

It is recommended that patients should be referred for ophthalmologic assessment for CXL if there is demonstrable progression in their keratoconus, as evident clinically through one or more of: topographical/ keratometric changes (e" 1.0 dioptral change in maximum keratometry value), repeated changes of contact lens base curve (if topography is unavailable) and/ or deteriorating vision.

Treatment is currently contraindicated in the following scenarios:

- Corneal thickness less than 400ìm
- Prior ocular herpetic infection
- Concurrent corneal infection
- Severe corneal scarring or opacification
- A history of poor wound healing
- Severe ocular surface disease (excluding dry eye)
- Auto-immune disorders
- Pregnancy

Careful ophthalmologic management of- CXL patients is required over the first few weeks post-treatment. A return to contact lens wear is recommended approximately four to six weeks after CXL, at which time the patient is generally returned to optometric care for ongoing monitoring at 6-12 monthly intervals.

Safety

CXL involves a highly localised photopolymerisation reaction that is generated in vivo, within the corneal stroma. Its application therefore requires consideration with regard to the potential effect on surrounding ocular structures, in particular the corneal endothelium, lens and retina.

Guidelines regarding optimal practice for CXL have recently been published.[37]The safety of the procedure is primarily dependent upon the appropriate delivery of the irradiation and ensuring a sufficient stromal riboflavin concentration. Of particular concern is the potential for oxygen radical damage to the corneal endothelium. Control of the depth of the reaction is primarily governed by the pre-operative corneal thickness. It is accepted that in order to minimise risk of permanent endothelial damage, a minimum corneal thickness of 400ìm is necessary following the debridement of the epithelium. The use of hypo-osmolar solutions of riboflavin can temporarily enhance corneal thickness in patients who do not meet the minimum thickness requirement[39], however further research is necessary to determine if this procedure provides adequate endothelial protection.

Although the theoretical risk exists, there is currently no evidence for CXL causing a reduction in corneal endothelial cell density. Published studies that have examined the endothelium using specular microscopy, one-year after CXL treatment have reported no quantitative change to endothelial cell numbers.[24,26,30]

Side-effects and Risks

Mild anterior- and mid-stromal corneal haze is the most common side-effect of CXL; the effect has been documented to persist for up to twelve months but has been noted to not significantly affect visual acuity.[27] Complications related to the debridement of the corneal epithelium include sterile corneal infiltrates40 and infectious keratitis secondary to bacteria[41], acanthamoeba[42] and Herpes simplex virus[43]; all of these complications were reported within the first week post-treatment.

While most of the published literature describes relative improvements in visual acuity (VA) post-CXL, a loss of two lines or more best-corrected VA was described in 2.9 per cent of treated eyes, one year post-operatively;[35] this should however be taken in context with the overall results of this study which reported progressive corneal ectasia (treatment failure) in 7.6 per cent of eyes.

Other Applications

CXL may also have a therapeutic role in the treatment of other corneal conditions, including pellucid marginal degeneration, post-LASIK keratoecastia, bullous keratopathy and microbial keratitis; definitive published evidence to support these applications is still required.[38]

Until recently, the management of keratoconus has been limited to supportive optical measures, primarily spectacles and contact lenses. The current scientific literature demonstrates that CXL has a significant arresting effect on the progression of keratoconus. Although further research into the long-term effects of CXL is still warranted, this exciting new treatment demonstrates the potential to transform the lives of patients with keratoconus, reducing their risk of progressive ectasia and visual impairment.

Dr. Laura Downie, BOptom, PhD(Melb), PGCertOcTher, DipMus(Prac), AMusA is an optometrist who specialises in contact lenses. She has been published in scientific journals and is a clinical instructor to undergraduate optometry students.

PROCEEDINGS OF THE ADDITIONAL DIRECTOR OF HEALTH SERVICES(MEDICAL), DIRECTORATE OF HEALTH SERVICES THIRUVANANTHAPURAM

Sub: Estt – HSD - General Transfer 2012 – Transfer & Posting of Optometrists - orders issued

Order No EF4- 16054/2012/DHS dated 30.3.2012

The following Optometrists Gr I & II (as shown in the annexure) are transferred and posted to the stations noted against each. The date of relief and joining should be reported promptly.

Sd/-

Dr.Kumari.G.Prema,

Addl.Director of Health Services

To

The Incumbents (through the head of institution)

Copy to

1.District Medical Officer of Health, ...

2. The Accountant General (Kerala), Thiruvananthapuram

3.The Superintendent,Taluk Headquarters/District/General Hospital .....

4. The Medical Officer in-charge, CHC/PHC .....

5.File / Stock File

// Forwarded //

Superintendent

ANNEXURE

Sl.No	Name and Present Station	Station to which transferred
1.	Flora Sheeja, General Hospital, Thiruvananthapuram.	PHC, Venpakal Thiruvananthapuram (Vice J. Sudhadevi, Senior Optometrist transferred)
2.	Remani.R.S, Government Model District Hospital, Peroorkada, Thiruvananthapuram	CHC, Vellanad, Thiruvananthapuram (Jayesh Thomson.T, Senior Optometrist transferred)

3	Sujatha R Nair THQH Chirayinkil, Thiruvananthapuram	PHC Anchuthengu, Thiruvananthapuram (Vice Sri.Sreekumar transfered)
4	Umakumari S THQH Neyyattinkara, Thiruvananthapuram	PHC Pulluvila, Thiruvananthapuram (Vice Geetha Kumari R transfered)
5	Sunil Kumar S THQH Parassala, Thiruvananthapuram	CHC Manabur, Thiruvananthapuram (Voce Naseera Beegum A transfered)
6	B R Shaji THQH Karunagapalli, Kollam	District Hospital, Kollam (Vice Retnakumari L transfered)
7	A Sujatha General Hospital, Pathanamthitta	District Hospital Kollam (Vice G Usha Kumari transfered)
8	Lekha Sivaraman THQH Mallapalli, Pathanamthitta	PHC Kanjeettukara, Pathanamthitta (Vice Rajeswari C K transfered)
9	Prasanna Kumari s THQH Tiruvalla, Pathanamthitta	CHC Thumbamon, Pathanamthitta (Vice Muhammed Rafeek transfered)
10	Ambili k S THQH Pala, Kottayam	PHC Paika, Kottayam (Vice Sushama E N transfered)
11	Biju Kumar R THQH Changanasseri, Kottayam	PHC Kumarakom, Kottayam (Vice Viswasanthi C S transfered)
12	Sreelekha K N THQH Vaikkom, Kottayam	CHC Edamaruku, Kottayam (Vice Ajitha K transfered)
13	Jitha Varghese General Hospital, Ernakulam	CHC Chengamanad, Ernakulam (Vice S Usha Kumari transfered)
14	Bindhu V Sidiq THQH Muvattupuzha, Ernakulam	CHC Kalady, Ernakulam (Vice Jaisree M V transfered)
15	Kavitha P THQH North Paravur, Ernakulam	PHC Thrikkakkara, Ernakulam (Vice T Anithakumari transfered)
16	Jeeja P Sadasivan THQH Perumbavoor, Ernakulam	PHC Nettoor, Ernakulam (Vice Mercy V transfered)
17	Ambily P Kumar THQH Thodupuzha, Idukki	PHC Muttom, Idukki (Vice Shamy Varghese transfered)
18	Anitha Mathew THQH Nedumgandam, Idukki	CHC Arakkulam, Idukki (Vice Asha Devi M transfered)
19	Vincent J THQH Wadakkancheri, Thrissur	CHC Mattathoor, Thrissur (Vice Rajila Beevi J transfered)
20	Remya C S THQH Chavakkad, Thrissur	CHC Mullasseri, Thrissur (Vice Thresiamma K M transfered)
21	Priji C S THQH Irinjalakkuda, Thrissur	PHC Piravom, Ernakulam (Vice Usha Kumari V transfered)
22	Leena Jacob District Hospital, Palakkad	PHC Kadampazhippuram, Palakkad (Vice Sathianesan V G transfered)
23	Shajahan S THQH Mannarkkad, Palakkad	CHC Puthenchira, Thrissur (Vice Thankamani J transfered)
24	Julie B l CHC Cheruvannore, Kozhikkode	General Hospital, Kozhikkode (Vice Rekha C D transfered)
25	Mansoor R THQH Tirurangadi, Malappuram	PHC Vengara, Malappuram (Vice Sheeba C S transfered)
26	Sheeba C S CHC Vengara, Malappuram	THQH Malappuram (Vice Bindhu C transfered)

27	Bindhu C THQH Malappuram	District Hospital Tirur, Malappuram (Vice Sudheesh B R transfered)
28	Shemeer M THQH Quilandy, Kozhikode	General Hospital, Kozhikode (Vice NAseema K transfered)
29	Pradeep Kumar P T THQH Kottarakkara, Kollam	PHC Chadayamangalam, Kollam (Vice Jayakumari K transfered)
30	Jayakumari K CHC Chadayamangalam, Kollam	CHC Kallara, Thiruvananthapuram (Vice Jasim V transfered)
31	Sasikala K S District Hospital, Kottyam	PHC Aryanad, Thiruvananthapuram (Vice Jayalekshmi transfered)
32	Sudheesh B R District Hospital, Tirur, Malappuram	General Hospital, Ernakulam (Vice Jidha Vardghese transfered)
33	Jayalekshmi PHC Aryanad, Thiruvananthapuram	CHC Kulathupuzha, Kollam (Vice Jaleela P transfered)
34	Faseena S PHC Edayazham, Kottayam	District Hospital, Kollam (Vice Subhash K S transfered)
35	Dussin S PHC Kadamanitta, Pathanamthitta	General Hospital Ernakulam (Vice Siby T P transfered)
36	Jaleela P PHC Kulathuppuzha, Kollam	PHC Kadamanitta, Pathanamthitta (Vice Dussin S transfered)
37	Suma M J THQH Alathur, Palakkad	PHC Panachikad, Kottayam (Vice P C Prasanna Kumari transfered)
38	Ajitha K CHC Edamaruku, Kottayam	PHC Nedumancavu, Kollam (Vice Ambika C J transfered)
39	Jaya V THQH Ottappalam, Palakkad	CHC Thuravoor, Alappuzha (Vice Santhamma M P transfered)
40	M J Sherly CHC Thiruvilamala, Thrissur	PHC Ochira, Kollam (Vice Bindu T S transfered)
41	Ambika C J PHC Nedumancavu, Kollam	General Hospital, Pathanamthitta (Vice Beena R transfered)
42	A Suresh Kumar General Hospital, Ernakulam	CHC Anchal, Kollam (Vice Binoy R transfered)
43	Binoy R CHC Anchal, Kollam	CHC Manaloor, Thrissur (Vice T T Valsa transfered)
44	Ramachandran Pillai General Hospital, Adoor, Pathanamthitta	CHC Nooranad, Alappuzha (Vice Sherly M transfered)
45	Rekha C D General Hospital, Kozhikode	CHC Cheruvannore, Kozhikode (Vice Julie B L transfered)
46	P T Letha THQH Kanjirappalli, Kottayam	General Hospital, Pathanamthitta (Vice Sujatha A transfered)
47	Abraham Varghese District Hospital, Manathawadi, Wayanad	PHC Seethathodu, Pathanamthitta (Vice Jacob B Reji transfered)
48	Rajan C THQH ponnani, Malappuram	PHC Pazhanji, Thrissur (Vice Sheeba Sebastian transfered)
49	P C Prassannakumari PHC Panachikkadu, Kottayam	CHC Vechoochira, Pathanamthitta (Vice Ameer Hamsath Beegum transfered)
50	Muhammed Rafeek PHC Thumbamon, Pathanamthitta	PHC Chunakkara, Alappuzha (Vice Beena V transfered)

51	Sreeja V C THQH Pulinkunnu, Alappuzha	CHC Edayazham, Kottayam (Vice Faseena S transfered)
52	K C Mariamma PHC Pallippuram, Alappuzha	PHC Vakathanam, Kottayam (Vice D Ambika transfered)
53	D Ambika PHC Vakathanam, Kottayam	PHC Pallippuram, Alappuzha (Vice K C Mariamma transfered)
54	T T Valsa PHC Manaloor, Thrissur	CHC Arunoottimangalam, Kottayam (Vice Biju K R transfered)
55	Biju K R CHC Arunoottimangalam, Kottayam	Govt: Hospital, Chengannur, Alappuzha (Vice Annie Mathew transfered)
56	M Smitha PHC Ollur, Thrissur	PHC Vettackal, Alappuzha (Vice Laila Beegum transfered)
57	Saramma Abraham THQH Ranni, Pathanamthitta	PHC Kumbalangi, Ernakulam (Vice K J Manoj entered LWA)
58	Sasikala C C GMTHQH Karuvelpadi, Ernakulam	CHC Ollur, Thrissur (Vice Smitha M transfered)
59	Kumari Radhamony THQH Nedumangad, Thiruvananthapuram	CHC Vellarada, Thiruvananthapuram (Vice A Rajendran transfered)
60	Jyothi L R THQH Mavelikkara, Alapuzha	CHC Mannar, Alappuzha (Vice Santha D transfered)
61	Gliny S THQH Harippad, Alappuzha	CHC Muthukulam, Alappuzha (Vice Usha O transfered)
62	Saliha Beevi A THQH Kothamangalam, Ernakulam	CHC Malippuram, Ernakulam (Vice Laila Kumari K transfered)
63	Anjali C General Hospital, Thalasseri, Kannur	PHC Cheruvadi, Kozhikode (Vice Joseph T T transfered)
64	Sandhya C THQH Chittoor, Palakkad	District Hospital, Palakkad (Vice Asha P Nair transfered)
65	Asha P Nair District Hospital, Palakkad	CHC Vengoor, Ernakulam (Vice Mary Poulose transfered)
66	Sreekala Kumari THQH Thaliparamba, Kannur	PHC Irikkur, Kannur (Vice Aswathy Babu transfered)
67	Aswathy Babu CHC Irikkur, Kannur	CHC Vadanapalli, Thrissur (Vice B Sujatha transfered)
68	Sheeja Sebastian CHC Pazhanji, Thrissur	CHC Pazhampalacode (Vice N S Baburajan transfered)
69	Baaburaj N S CHC Pazhampalacode, Thrissur	CHC Thiruvilwamala, Thrissur (Vice Sherly M J transfered)
70	Omana M District Hospital, Idukki	CHC Elanthoor, Pathanamthitta (Vice Mary Kutty Philip transfered)
71	Soumya Devassia CHC Mulliyar, Kasargod	District Hospital, Idukki (Vice Omana M transfered)
72	Sreekala S PHC Kalackode, Kollam	PHc Edayirikkapuzha, Kottayam (Vice Vimal Roy transfered)
73	Prathibha S PHC Niranom, Pathanamthitta	District Hospital, Kollam (Vice Subhash K S transfered)
74	Bindhu T S CHC Ochira Kollam	PHC Niranom, Pathanamthitta (Vice Prathibha S transfered)

PROCEEDINGS OF THE ADDITIONAL DIRECTOR OF HEALTH SERVICES (MEDICAL), DIRECTORATE OF HEALTH SERVICES, THIRUVANANTHAPURAM

Sub:- Estt.- H.S.Dept.- Transfer and Re-posting of Senior Optometrists in Ophthalmology wing - Final list publishing of-

Read:- G.O.(Ms) No.145/2011/H&FWD Dt. 28.02.2011.

ORDER No.EF4-58098/2011/DHS DATED : 30.03.2012.

The following Transfer and re-posting of Senior Optometrists in Health Services Department are ordered as directed in the Government Order read above.

Sd/-
Dr.Kumari.G.Prema,
Additional Director of Health Services (Medical)

To

The incumbents

Copy to:

1. All District Medical Officer of Health (Circulation)
2. Web site

ANNEXURE

Sl. No.	Name and institution of Senior Optometrist	Station allotted
1.	Sri. Sreekumar, C.H. Centre, Anchuthengu, Thiruvananthapuram	General Hospital, Thiruvananthapuram. Vice Flora Sheeja transferred
2.	Geethakumari.R., C.H. Centre, Pulluvila, Thiruvananthapuram	Model District Hospital, Peroorkada, Thiruvananthapuram Vice Remani.R.S transferred
3.	Jasim.U., C.H. Centre, Kallara, Thiruvananthapuram	THQH, Nedumangad, Thiruvananthapuram Vice Kumari Rathamany transferred

4.	Naseera Beegom.A, C.H. Centre, Manamboor, Thiruvananthapuram	THQH, Chirayinkil, Thiruvananthapuram Vice Sujatha.R.Nair transferred
5.	J.Sudhadevi, C.H. Centre, Venpakal, Thiruvananthapuram	THQH, Neyyattinkara, Th iruvananthapuram Vice Umakumari.S. transferred
6.	A.Rajendran, C.H. Centre, Vellarada, Thiruvananthapuram	THQH, Parassala, Thiruvananthapuram Vice Sunil kumar.S. transferred
7.	Sheela.L., District Mobile Unit, District Hospital, Kollam	District Hospital, Kollam
8.	Ludhiyamma Chacko, THQH, Sasthamcottah, Kollam	THQH, Punaloor, Kollam Vice K.Sulochana transferred
9.	Retnakumari.L, District Hospital, Kollam	THQH, Sasthamcottah, Kollam Vice Ludhiamma Chako transfered
10.	K.S.Sabhash, District Hospital, Kollam	THQH, Karunagappally Vice Vice B.R.Shaji transferred
11.	K.Sulochana, THQH, Punaloor, Kollam.	THQH, Kottarakkara. Vice Pradep kumar.P.T transferred
12.	Abdul Jabharudeen.M. General Hospital,Pathanamthitta	General Hospital, Pathanamthitta
13.	Jacob.B.Reji, P.H. Centre, Seethathode, Pathanamthitta	General Hospital, Adoor, Pathanamthitta Vice Ramachandran Pillai transferred
14.	Laila Beegum.A., P.H. Centre,Vettackal,A lappuzha	THQH, Mallapalli, Pathanamthitta Vice Lekha Sivaraman transferred
15.	Rajeswary.C.K, C.H. Centre, Kanjeettukara, Pathanamthitta	THQH, Thiruvalla, Pathanamthitta Vice Prasannakumar.S transferred
16.	Beena.R, General Hospital, Pathanamthitta	THQH, Ranni, Pathanamthitta Vice Saramma Abraham transferred
17.	Ameer Hamsath Beegum.M.A, C.H. Centre, Vechuchira, Pathanamthitta	District Hospital, Kozhancherry, Pathanamthitta. Vice Saji.G transferred
18.	Ani Mathew, THQH, chengannoor, Alappuzha	General Hospital, Alappuzha Vice Venugopalan.P, Promoted

19.	Shaija.P.S. THQH, Cherthala, Alappuzha	THQH, Cherthala, Alappuzha
20.	Santhamma.M.P, C.H.Centre, Thuravoor, Alappuzha	THQH, Mavelikkara, Alappuzha Vice Jyothi.L.R transferred
21.	Sanitha.D, C.H.Centre, Mannar, Alappuzha	THQH, Pulikunnu, Alappuzha Vice Sreeja.V.C transferred
22.	Beena.V, C.H.Centre, Chunakkara, Alappuzha	THQH, Harippad, Alappuzha Smt.Gliny.S. transferred
23.	M.K.Radhamani, THQH, Chengannoor, Alappuzha	THQH, Chengannoor, Alappuzha
24.	Saji.G, District Hospital, Kozhencheri, Pathanamthitta	District Hospital, Kottayam Vice Sasikala.K.S transferred
25.	S.Ushakumari, C.H. Centre, Chengamanad, Ernakulam	THQH, Pala, Kottayam Vice Ambili.K.S transferred
26.	Marykutty Philip, C.H. Centre, Elanthoor, Pathanamthitta	THQH, Kanjirappalli, Kottayam Vice P.T. Latha transferred
27.	Vimal Roy.V.P, C.H.Centre, Edayirikkappuzha, Kottayam	THQH,Changanasseri, Kottayam Vice Bijukumar.R transferred
28.	Viswasanthy.C.S C.H.Centre, Kumarakom, Kottayam	THQH, Vaikkom, Kottayam Vice Sree letha.K.N. transferred
29.	Lailakumari.K, P.H. Centre, Malippuram, Eranakulam	General Hospital, Ernakulam Vice A.Suresh kumar transferred
30.	Jayesh Thompson.T.P., C.H. Centre, Vellanad, Thiruvananthapuram	Government Maharaja Hospital, Karuvelippadi, Ernakulam Vice Sasikala C.C. transferred
31.	Baiju.K.R, District Hospital, Aluva, Ernakulam	THQH, Thrippunithura, Ernakulam Vice Sabu promoted
32.	Mercy.V, P.H.Centre, Netoor, Ernakulam	THQH, Aluva, Ernakulam Baiju.K.R. tansferred
33.	Sushama.E.N, C.H. Centre, Paika, Kottayam	THQH, Muvattupuzha, Ernakulam Vice Bindu.V.Siddiqu transferred
34.	Siby.T.P., General Hospital, Ernakulam	T HQH, North Paravoor, Ernakulam Vice Kavitha transferred
35.	Mary Poulose, C.H.Centre, Vengoor, Ernakulam	THQH, Kothamangalam, Ernakulam Vice SalihaBeevi transferred
36.	Jayasree.M.V, C.H. Centre, Kalady, Ernakulam	THQH, Perumbavoor, Ernakulam Vice Jeeja.P.Sadasivan transferred
37.	Asha Devi.M, C.H. Centre, Arakulam, Idukki	District Hospital, Idukki Vice Sreeelatha.M.M transferred
38.	Geors Celin.K.George THQH, Adimaly, Idukki	THQH, Adimaly, Idukki

39.	Shamy Varghese, P.H. Centre, Muttom, Idukki	THQH, Thodupuzha, Idukki Vice Ambili.P.Kumar transferred
40	Sreeletha.M.M. District Hospital, Idukki	THQH, Nedumgandam, Idukki Vice Anitha Mathew transferred
41.	Sivadasan.P.K. District Hospital, Thrissur	District Hospital, Thrissur
42.	Leena.S THQH, Kodungalloor,Thrissur	THQH, Kodungalloor, Thrissur
43.	Moly Thomas, THQH, Chalalludi, Thrissur	THQH, Chalakkudi, Thrissur
44.	Rajila Beevi.J, C.H.Centre, Mattaathoor, Thrissur	THQH, Wadakkancherri, Thrissur Vice Vincent.V. transferred
45.	Sujatha.B, C.H.Centre, Vatanappally, Thrissur	THQH, Chavakkad, Thrissur Remya .C.S. transferred
46.	Thankamany.J, P.H. Centre, Puthenchira, Thrissur	THQH, Irinjalakkuda, Thrissur Vice Priji.C.S transferred
47.	V.Ushakumari, CH.Centre, Piravom, Ernakulam	District Hospital, Palakkad Vice Leena Jacob,transferred
48.	Narayanan.T.P., THQH, Pattambi, Palakkad	THQH, Pattambi, Palakkad
49.	G.Ushakumari, District Hospital, Kollam	THQH, Mannarkkad, Palakkad Vice Shajahan.S transferred
50.	Sathianesan.V.G, C.H.Centre, Kadambazhipuram, Palakkad	THQH, Ottappalam, Palakkad Vice Jaya.V. Transferred
51.	Thresiamma.K.M, C.H. Centre, Mullassery, Thrissur	THQH, Alathur, Palakkad Suma.M.J. transferred
52.	Joseph.T.T, C.H.Centre, Cheruvady, Kozhikode	General Hospital, Manjeri Sulu.E.P. transferred
53.	Vishnu Maya.T., District Hospital, Tirur, Malappuram	District Hospital, Tirur, Malappuram
54.	Sankara Narayanan.V.C, THQH, Perinthalmanna, Malappuram	THQH, Perinthalmanna, Malappuram
55.	Sherly.M., C.H. Centre, Nooranad, Alappuzha	THQH, Ponnani, Malappuram Vice Visvasanthi transfered
56.	Usha.O, C.H.Centre, Muthukulam, Alappuzha	THQH, Tirurangadi, Malappuram Vice Mansoor.R transferred
57.	Sulu.E.P., General Hospital, Menjeri, Malappuram	General Hospital, Kozhikode Krishna swamy Transferred
58.	Vijayan.M, General Hospital, Vadakara, Kozhikode	General Hospital, Vadakara, Kozhikode
59.	Naseema.K.K., General Hospital, Kozhikode	THQH, Quilandy, Kozhikode Vice Shamer.M transferred
60.	Binu Peter, District Mobile Unit, Kannur	District Hospital, Kannur
61.	Krishnaswami.T, General Hospital, Kozhikode	General Hospital, Thalassery Vice Anjali.C transferred
62.	T.Anithakumari, P.H. Centre, Thrikkakara, Ernakulam	THQH Chittoor, Palakkad Vice Santhya.C. transferred

Sd/-

Dr.Kumari.G.Prema,

Additional Director of Health Services (Medical)