Erasmus MC
University Medical Center Rotterdam

# Omgaan met lichamelijk onverklaarde klachten
## Training voor specialisten en aios

Syllabus
Juni 2014

# Verantwoording

Deze syllabus en training is ontwikkeld in het kader van het onderzoeksproject 'Effectiviteit en doelmatigheid van een training consultvaardigheden voor medisch specialisten gericht op patiënten met somatisch onvoldoende verklaarde klachten (SOLK)'. In dit project is samengewerkt door de afdelingen Inwendige Geneeskunde en Neurologie van het Erasmus MC, het Instituut Psychologie van de Faculteit Sociale Wetenschappen van de Erasmus Universiteit Rotterdam, de afdeling Huisartsgeneeskunde/EMGO+ van het VUmc en het NIVEL.
De resultaten van dit onderzoek verwachten we eind 2014 beschikbaar te hebben.
Deze training bouwt voort op de NHG - cursus 'Beleid bij patiënten met onverklaarde klachten, cognitief gedragstherapeutische technieken door de huisarts' uit 2003. Voor het gebruik van de bouwstenen is door het NHG toestemming verleend. Ook is met toestemming gebruik gemaakt van onderdelen uit de vernieuwde versie van de trainingssyllabus voor huisartsen door Quartz Helmond (maart 2011).


**Auteur en eindredacteur:**
Drs. A. Weiland, als onderzoeker en trainer verbonden aan de afdeling Inwendige Geneeskunde van het Erasmus MC Rotterdam en het Instituut voor Psychologie van de Erasmus Universiteit Rotterdam.

**Contactgegevens:**
a.weiland@erasmusmc.nl
http://www.anneweiland.nl/contact.
http://www.lichamelijkonverklaardeklachten.nl

**Redactie:**
Prof. Dr. J.L.C.M. van Saase, opleider en afdelingshoofd Inwendige Geneeskunde Erasmus MC Rotterdam
Prof. Dr. H.T. van der Molen, decaan Faculteit Sociale Wetenschappen en hoogleraar psychologie aan het Instituut voor Psychologie van de Erasmus Universiteit Rotterdam
Dr. A.H. Blankenstein, huisarts en hoofd Huisartsopleiding VUmc Amsterdam
Prof. Dr. L.R. Arends, psycholoog en hoogleraar methoden en technieken aan het Instituut voor Psychologie en het Instituut voor Pedagogiek van de Erasmus Universiteit Rotterdam
Drs. M.H.A. Willems, neuroloog Erasmus MC Rotterdam

Juni 2014

# Inhoudsopgave

# 1. Inleiding

## Relevantie

Voor veel lichamelijke klachten kan geen medische verklaring gegeven worden. Het percentage poliklinische patiënten met onverklaarde klachten bedraagt voor neurologen, gynaecologen en reumatologen tenminste 40%. Goede arts-patiënt-communicatie is dé sleutel tot doelmatige medische zorg voor patiënten met somatisch onvoldoende verklaarde klachten. Effectieve consultvoering bij deze patiëntengroep leidt tot preventie van somatoforme stoornissen, verbetering van welzijn van patiënten en kostenbesparing door het voorkomen van overbodig medisch specialistisch ingrijpen. Een training consultvaardigheden voor specialisten, toegespitst op deze omvangrijke patiëntengroep, kan de kwaliteit van zorg verbeteren.
Het onderzoeksproject "Effectiviteit en doelmatigheid van een training consultvaardigheden voor medisch specialisten gericht op patiënten met somatisch onvoldoende verklaarde lichamelijke klachten (SOLK)" is erop gericht een dergelijke training te ontwikkelen én te testen op effectiviteit en doelmatigheid. De training 'Omgaan met lichamelijk onverklaarde klachten' is hiervan het resultaat en is als interventie onderwerp van studie. In een pilot training op de afdeling Neurologie van het Erasmus MC in 2007 en 2008 is al veel ervaring opgedaan met de trainingsopzet en de toepassing van de geleerde consultvaardigheden voor neurologen in de praktijk. In 2012 en 2013 is de interventie in 6 ziekenhuizen uitgetest, waaraan artsen van 11 verschillende specialismen hebben meegedaan.

## Regie huisarts

De huisarts heeft conform de nieuwe richtlijn de regie in de zorg voor patiënten met SOLK. De medisch specialist komt veelal in beeld als de huisarts de patiënt met onverklaarde klachten verwijst voor aanvullend onderzoek, diagnostiek en beleid. Vanuit dat perspectief is de verwijzing naar de specialist idealiter een beperkte en essentiële stap in een langdurig beleid van de huisarts. Soms is de verwijzing van de huisarts naar de specialist er op gericht om (nogmaals) vast te stellen dat er geen ziekte aan de onbegrepen klachten ten grondslag ligt. De wijze waarop de specialist een normale testuitslag meedeelt is van invloed op de mate van geruststelling bij de patiënt. Wanneer u als specialist een doorverwijzing van een patiënt met SOLK naar een andere specialist of hulpverlener noodzakelijk acht is het wenselijk daarin de huisarts vooraf te betrekken om te voorkomen dat er onnodige, medische handelingen worden verricht zonder een verbetering van de onbegrepen klachten van de patiënt.

## Leerdoelen

De specialist en aios heeft na afloop van de training:

- Een brede kennis aangaande theorie, achtergronden, epidemiologie, etiologie, behandelmodellen en behandelmogelijkheden voor patiënten met SOLK en somatoforme stoornissen.
- De vaardigheid cognitief gedragstherapeutische technieken in te zetten in consulten met patiënten met lichamelijk onverklaarde klachten voor wat betreft exploratie, uitleg, vervolgbeleid en terugrapportage:
  - Exploratie van lichamelijke, emotionele, cognitieve, gedragsmatige en sociale aspecten van de lichamelijk onverklaarde klachten van een patiënt via de SCEGS analyse.
  - Uitleg geven aan patiënten met lichamelijk onverklaarde klachten op basis van de bevindingen uit exploratie, anamnese en onderzoek over de oorsprong en het beloop van hun klachten.
  - Uitleg geven over de rol van vicieuze cirkels en neerwaartse spiralen bij het in stand houden van de lichamelijke klachten.
  - Uitleg geven aan patiënten met onverklaarde klachten wat ze van aanvullend specialistisch onderzoek kunnen verwachten.
  - Effectief geruststellen van patiënten met lichamelijk onverklaarde klachten.
  - Adequaat reageren op onzinnige verwijzingen.
  - Adequaat een terugrapportage naar de huisarts maken, waarin de verwijsvraag van de huisarts zowel als de hulpvraag van de patiënt met lichamelijk onverklaarde klachten beantwoord wordt met een duidelijke uitleg over bevindingen en advies.

Daarnaast vergroot u met deze training ook uw competenties op het taakgebied 'arts-patiënt communicatie':

- U leert hoe uw eigen opvattingen en attitude uw consulten met patiënten met lichamelijk onverklaarde klachten beïnvloeden.
- U bevordert een actieve inbreng en zelfwerkzaamheid van uw patiënt met lichamelijk onverklaarde klachten en betrekt uw patiënt in de besluitvorming.
- U ontwikkelt effectieve behandelrelaties met patiënten met onverklaarde klachten.

## Werkwijze

De huidige training bevat 4 bijeenkomsten van in totaal 14 uur  met een interval van 4 tot 6 weken, die in groepen van 12 deelnemers wordt gegeven onder leiding van 2 ervaren trainers.
Elke bijeenkomst heeft een eigen thema en is erop gericht deelnemers vertrouwd te maken met het toepassen van cognitief gedragstherapeutische technieken in de consultvoering gericht op patiënten met SOLK. De training bestaat uit het aanbieden van korte theoretische fragmenten in combinatie met veel ruimte voor het oefenen van vaardigheden met eigen casuïstiek. Het programma is zo opgebouwd, dat er steeds na elk dagdeel een aantal weken ruimte zit om de vaardigheden in de eigen praktijk toe te passen. De MDR "SOLK en somatoforme stoornissen" en de syllabus bevatten ondersteunende informatie ter voorbereiding van de afzonderlijke bijeenkomsten. Van u als cursist wordt verwacht voorafgaand aan elke bijeenkomst het opgegeven huiswerk voor te bereiden, zodat u tijdens de bijeenkomsten de vaardigheden optimaal kunt oefenen.

## Literatuur

Voor de training wordt de Multi Disciplinaire Richtlijn "SOLK en somatoforme stoornissen" (2010) gebruikt als inhoudelijke standaard en naslagwerk. Deze MDR is gratis te downloaden via www.ggzrichtlijnen.nl. De MDR is ook als boek verkrijgbaar en te bestellen via de webwinkel van het Trimbos-instituut: www.trimbos.nl/webwinkel; artikelnummer AF0945, prijs excl. verzendkosten 22,95 euro. Daarnaast is er een syllabus met aanvullende theorie, artikelen, praktijkopdrachten en oefenmateriaal.

## Huiswerk ter voorbereiding 1ste dagdeel:

Voor de eerste bijeenkomst is deelnemers aan de training gevraagd zich als volgt voor te bereiden:
uit de MDR hoofdstuk 1 t/m paragraaf 1.7. + paragraaf 2.4 ('Diagnostisch stappenplan voor de eerste en tweede lijn' ) te lezen, uit de syllabus de hoofdstuk 1 t/m 5 te lezen, oefening 1 te doen uit de syllabus (hoofdstuk 9) en een casus van een patiënt met lichamelijk onverklaarde klachten mee te brengen via het ingevulde casuïstiekformulier (hoofdstuk 8).

# Programma overzicht

| Dagdeel 1 | Thema 'Exploratie en uitleg' |
|---|---|
| 13.00 – 13.30 | ▪ Introductie: link training/onderzoek, docenten stellen zich voor<br>▪ Voorstelronde: naam, specialisme, ervaring met SOLK-pt, leervraag<br>▪ Programma en huishoudelijke mededelingen |
| 13.30 – 13.50 | ▪ Oefening 1: Herkenning eigen gedachten, emoties en gedrag tav SOLK<br>▪ Soorten patiënten met SOLK, over welke pt gaat het in de training? |
| 13.50 – 15.00 | ▪ Exploreren: uitleg over SCEGS + informatie uit verwijsbrief<br>▪ Illustratie mbv Dvd-consult (fragment pt Zwaan, SCEGS)<br>▪ Reacties op Dvd-consult<br>▪ Uitleg werkwijze en oefeningen<br>▪ Oefening 2: Doorvragen over klachten (met eigen casus)<br>▪ Nabespreken plenair |
| 15.00 – 15.15 | ▪ Thee pauze |
| 15.15 – 16.30 | ▪ Informeren: uitleg algemeen<br>▪ Oefening 3: Informeren; Samenvatten van bevindingen<br><br>▪ Informeren: uitleg neerwaartse spiralen<br>▪ Oefening 4: Informeren; Benoemen en tekenen van neerwaartse spiralen<br>▪ Nabespreken oefening 3 en 4 |
| 16.30 – 17.00 | ▪ Ruimte voor vragen, opmerkingen;<br>▪ Indeling in koppels voor presentaties in de 3e bijeenkomst en introductie bijbehorende opdracht<br>▪ Huiswerk voor volgende keer: leeswerk; SCEGS oefenen; uitleg neerwaartse spiralen oefenen |
| 17.00 | ▪ Afsluiting |

| Dagdeel 2 | Thema 'Afstemmen van verwachtingen' |
|---|---|
| 13.00 – 13.40 | ▪ Welkom en programma<br>▪ Uitwisseling ervaringen met toepassen vaardigheden |
| 13.40 – 14.15<br><br><br><br><br>14.15 – 14.40 | ▪ Oefening 5: Warming up<br>▪ Wat draagt bij aan effectieve geruststelling? Groepsgesprek<br>▪ Oefening 6: Effectief geruststellen<br>▪ Nabespreken oefening<br><br>▪ Afstemmen van verwachtingen, uitleg<br>▪ Illustratie mbv Dvd-consult (fragment pt Zwaan, final test)<br>▪ Bespreken reacties |
| 14.40 – 14.55 | ▪ Thee pauze |
| 14.55 – 15.40<br><br><br><br>15.40 – 16.10<br><br><br><br>16.10 – 16.50 | ▪ Verwachtingen over onderzoek vooraf bespreken met pt, uitleg<br>▪ Oefening 7: Afstemmen van verwachtingen<br>▪ Nabespreken<br><br>▪ Reageren op verwijzingen, die je zelf onzin vindt<br>▪ Oefening 8: casus over 'onzin-verwijzing'<br>▪ Nabespreken<br><br>▪ Afronding en antwoord op verwijzing huisarts en hulpvraag pt, uitleg<br>▪ Oefening 9: Terugrapportage naar huisarts<br>▪ Nabespreken |
| 16.50 – 17.00 | ▪ Ruimte voor vragen en opmerkingen<br>▪ Huiswerk voor volgende bijeenkomst: alle technieken oefenen; terugrapportage maken en meenemen; voorbereiden presentatie eigen casuïstiek in koppels |
| 17.00 | ▪ Afsluiting |

| Dagdeel 3 | Thema 'Verwijzing en implementatie' |
|---|---|
| 13.00 – 13.05 | ▪ Welkom en programma; hoe ziet de cursus er vandaag uit?<br>▪ Inventarisatie aantal presentaties |
| 13.05 – 14.05 | ▪ Oefening 10: Integratie vaardigheden -patiënt met lastige SOLK |
| 14.05 – 14.20 | ▪ In tweetallen lezen van elkaars terugrapportage naar huisarts<br>▪ Essentiële punten in terugrapportage, uitleg<br>▪ Oefening 11: Bespreking van eigen terugrapportage in tweetallen<br>▪ Nabespreken |
| 14.20 - 14.30 | ▪ Behandeling van SOLK met CGT in de GGZ, uitleg |
| 14.30 – 14.45 | ▪ Klaar zetten presentaties en theepauze |
| 14.45 – 16.45 | ▪ Oefening 12: Presentaties en discussie |
| 16.45 – 17.00 | ▪ Ruimte voor vragen en opmerkingen<br>▪ Huiswerk voor laatste bijeenkomst: Oefening 13: Zelfevaluatie maken eventueel mbv filmpje van SOLK-consulten gemaakt voorafgaand aan de training: op welke punten ben ik vaardiger geworden? |
| 17.00 | ▪ Afsluiting |

| Dagdeel 4 | Thema 'Verdieping en verfijning' |
|---|---|
| 15.00 – 15.05<br><br>15.05 – 15.30 | ▪ Welkom en programma van vandaag<br><br>▪ In tweetallen, uitwerking oefening 13:<br>▪ wat gaat er al goed (nav zelfevaluatie)?<br>▪ wat vind je nog lastig in consulten met SOLK-pt?<br>▪ vertaling naar oefensituatie |
| 15.30 – 16.40 | ▪ Indeling in subgroepen; oefenen met situaties die lastig gaan |
| 16.40 – 17.00 | ▪ Voornemens voor de toekomst<br>▪ Evaluatieformulier invullen<br>▪ Uitreiken van certificaten<br>▪ Korte nabespreking van cursus |
| 17.00 | ▪ Afsluiting |

# 2. Lichamelijk onverklaarde klachten

Dit hoofdstuk is grotendeels een bewerkte versie van hoofdstuk 1 uit het 'Handboek Somatisatie. Lichamelijk onverklaarde klachten in de eerste en tweede lijn' van CM van der Feltz-Cornelis en HE van der Horst.[1]

## 2.1. Vooraf

De patiënt die met een veelheid van lichamelijke klachten, die stuk voor stuk moeilijk te duiden zijn, voor de zoveelste keer de spreekkamer van zijn arts betreedt, is van alle tijden en komt overal ter wereld voor.[2,3]
Naar schatting blijft 20-50 procent van de klachten en problemen waarmee patiënten hun huisartsen consulteren lichamelijk onverklaard.[4,5,6]
Niet zelden leidt dat tot een eindeloze rondgang in het medisch circuit, zodat ook in tweedelijns setting regelmatig patiënten met lichamelijk onverklaarde klachten worden gezien. In een Nederlands onderzoek onder een poliklinische populatie werd vastgesteld dat er bij 25 procent van de patiënten sprake is van lichamelijk onverklaarde klachten.[7] Engelse cijfers komen nog hoger uit: rond de 35 procent van nieuwe verwijzingen zou patiënten met lichamelijke onverklaarde klachten betreffen.[8]
In een deel van de gevallen kan de arts een onschuldige, fysiologische verklaring van de klachten geven, waarmee de patiënt zich getroost en gerustgesteld weer naar huis laat sturen. In een ander deel van de gevallen gaat dat niet op, dan is er een wat meer hardnekkige ongerustheid over de klachten, of een duidelijke overtuiging dat de klachten toch echt veroorzaakt worden door een ziekte.
Ook wanneer er wel sprake is van een lichamelijke ziekte, kunnen er klachten zijn waar geen goede verklaring voor is. De patiënt kan zijn of haar klachten toeschrijven aan de aandoening die hij of zij heeft, terwijl de arts van mening is dat die klachten daar eigenlijk niet mee te maken kunnen hebben, of dat zij, te oordelen naar de ernst van de aandoening, niet zo ernstig kunnen zijn als de patiënt beschrijft.

De huisartsgeneeskundige zorg voor deze groep patiënten is niet gemakkelijk: alert blijven op mogelijke achterliggende ziekte, openingen voor psychosociale hulp blijven verkennen, terwijl de huisarts soms met tegenzin aan het consult begint. Met name langdurige of veelvuldig terugkerende lichamelijk onverklaarde klachten kunnen een wissel trekken op de arts-patiëntrelatie.
De patiënt heeft lichamelijke klachten, die dagelijks hinder, onwelbevinden, bezorgdheid en beperkingen met zich meebrengen. Hij bezoekt zijn huisarts voor een duidelijke diagnose en behandeling van de lichamelijke klachten. Deze kan de huisarts in de gegeven situatie niet bieden. Omdat de klachten niet verminderen, ondanks doorverwijzingen en behandelingen, bezoekt de patiënt zijn huisarts steeds weer met een somatisch gerichte hulpvraag en bijpassende verwachtingen. Geruststelling, een fysiologische of psychosociale verklaring door de huisarts, sluiten daar niet op aan en geven dan ook geen soelaas. Integendeel: de huisarts raakt geïrriteerd door het herhaalde en dwingende appèl, dat hij niet kan beantwoorden. De patiënt voelt zich een aansteller, een klager (het is 'psychisch'), onbegrepen, of zelfs afgewezen. Bovendien merkt hij de machteloosheid van zijn dokter. Het resultaat is, dat de huisarts en de patiënt zich machteloos en ontevreden voelen, wat op den duur ook de vertrouwensbasis van de arts-patiëntrelatie ondermijnt.

Een cognitief gedragstherapeutische aanpak door de huisarts kan in veel gevallen de impasse doorbreken, of nog beter, voorkomen. Deze technieken kunnen het gereedschap zijn voor de huisarts met een 'lege handen'-gevoel, dat sommige patiënten teweeg kunnen brengen.

## 2.2. Terminologie

Het is niet verwonderlijk dat er voor een fenomeen dat zo vaak en zo wereldwijd voorkomt vele benamingen in omloop zijn. De meest neutrale term is *lichamelijk onverklaarde [of: onbegrepen] klachten*. Het voordeel van deze term is, dat hij precies weergeeft waarvan er sprake is, terwijl veel andere termen hetzij verwijzen naar specifieke mechanismen, hetzij voor velerlei uitleg vatbaar zijn.
Andere termen die veel gebruikt worden of tot voor kort gebruikt zijn, passeren hieronder de revue.

Het begrip *vage klachten* is een benaming die nogal eens gebruikt wordt, overigens zowel door artsen als soms ook door patiënten, om weer te geven dat er voor de klachten geen lichamelijke oorzaak te vinden is. Nadeel van deze term is dat veel patiënten zich ermee afgescheept voelen, en dat de term ook niet goed weergeeft waar het om gaat. De klachten zijn vaak voor patiënten allerminst vaag, zij weten waar ze last van hebben en hoeveel last ze ervan hebben. Dat de arts ze niet kan duiden is een andere zaak.

*Functionele klachten* is een term die artsen vaak hanteren om aan te geven dat er naar hun mening geen verklaring is voor de lichamelijk klachten. Vaak wordt er tevens mee bedoeld dat de oorzaak 'dus' psychologisch bepaald is. Het woord functioneel zou men op twee manieren kunnen opvatten. Enerzijds kan 'functioneel' verwijzen naar de functie die de klachten hebben, bijvoorbeeld het indirect kenbaar maken van allerlei ongenoegens of van onvrede met een bestaande situatie in het leven van de patiënt, of het verschaffen van een 'alibi' om zich gelegitimeerd te kunnen onttrekken aan verplichtingen. Anderzijds kan het begrip 'functionele klacht' opgevat worden als een klacht over een bepaalde lichamelijke functie: zoals bij het prikkelbaredarmsyndroom waar er naast pijnklachten ook sprake is van klachten over de darmfunctie: diarree of verstopping, winderigheid etcetera.

In Nijmegen is in de jaren tachtig van de vorige eeuw het begrip *nerveus-functionele klachten* ontwikkeld. Om de 'diagnose' nerveus-functionele klachten te mogen stellen hoort er niet alleen sprake van functionele klachten te zijn, maar dient er ook een duidelijke aanwijzing te zijn dat de klachten samenhangen met psychosociale problematiek. Hoewel de term in Nijmeegse publicaties, onder andere voortkomend vanuit de Continue Morbiditeits Registratie, nog steeds opduikt, is zij in de rest van de Nederland niet echt in zwang gekomen.[4]

De term *somatische fixatie* komt eveneens uit de Nijmeegse school. Er wordt gesproken van somatische fixatie 'als mensen door een proces van voortdurend inadequaat omgaan met en reageren op ziekte, onlustgevoelens, klachten of problemen, door henzelf, door hun sociale omgeving of door vertegenwoordigers van de gezondheidszorg, meer dan nodig afhankelijk worden van anderen, met name van [medische] hulpverlening of zelfs gaan vastlopen in het medisch kanaal.'[9]
Hoewel de term nog regelmatig gebruikt wordt, heeft hij geen brede ingang gevonden. Vanuit psychiatrische hoek is er bezwaar geuit tegen de term somatische fixatie, omdat het weer net iets anders beschrijft dan de gangbare termen. In huisartsenkringen is het een niet onomstreden begrip, waarover voor- en tegenstanders een tijdlang in discussie zijn geweest. Eén van de bezwaren was, dat het gaat om een ingewikkeld proces dat empirisch niet of nauwelijks valt te onderbouwen.[10]

*Psychosomatiek* of *psychosomatische klachten* zijn termen die nog steeds terugkeren in publicaties of nascholingen rond het onderwerp lichamelijk onverklaarde klachten. De term psychosomatiek verwijst naar een hypothese die in de jaren 50 van de vorige eeuw onder andere door de internist Groen werd geopperd. De aanhangers van de psychosomatiek gingen ervan uit, dat specifieke intrapsychische conflicten en persoonlijkheidsconstellaties kunnen leiden tot specifieke lichamelijke aandoeningen. De maagzweer, astma, colitis ulcerosa golden bij uitstek als voorbeelden van een dergelijke psychosomatose. De hypothese is onhoudbaar gebleken, dat wil zeggen is nooit aangetoond. Inmiddels is duidelijk, dat bij de genoemde ziektebeelden diverse factoren een rol spelen, waar een eventuele psychische factor maar een klein onderdeel van uitmaakt.

Het begrip *somatisatie* werd in het begin van de vorige eeuw geïntroduceerd door Stekel. Hij verstond hieronder een diep gewortelde neurose, verwant aan het conversiemechanisme.[10] Dit is een geheel andere betekenis dan daaraan tegenwoordig in het algemeen gegeven wordt. Lipowski is in 1988 met een omschrijving gekomen die sinds die tijd vrij algemeen gebruikt wordt. Hij beschrijft somatisatie aldus: 'Somatization is a tendency to experience, and communicate somatic distress and symptoms, unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them'.[11] Belangrijke elementen van deze omschrijving zijn:

- het ervaren van klachten en ze uiten;
- het toeschrijven aan een lichamelijke oorzaak, waar die onaannemelijk is en niet gevonden wordt;
- het zoeken van medische hulp voor de klachten.

Het fenomeen dat mensen lichamelijke klachten ervaren ook als daar geen lichamelijke ziekte aan ten grondslag ligt is een alledaags gebeuren. Van de bevolking ervaart 85-95 procent in een willekeurige periode van een paar weken minimaal één somatische klacht.[12] Het merendeel van de mensen raadpleegt daarvoor echter geen arts en schrijft de klachten toe aan: stress, een verkeerde beweging, verkeerd eten, slecht slapen, de menstruatie of wat er ook maar in aanmerking kan komen als verklaring. Goldberg en Bridges stellen dat somatiseren een wereldwijd voorkomend fenomeen is, en waarschijnlijk beschouwd kan worden als een basismechanisme van mensen, dat optreedt in reactie op stress.[13]

Somatisatie kan ook beschouwd worden als een min of meer normaal psychologisch en biologisch adaptatie-mechanisme.[11] Somatisatie wordt dan ook wel als een continuüm gezien: aan de ene kant van het spectrum zitten de mensen die nauwelijks de neiging hebben om klachten te ervaren, laat staan ze te uiten of aan artsen te rapporteren; aan de andere kant van het spectrum bevinden de zich de mensen die voldoen aan de criteria van een somatoforme stoornis conform de DSM-IV,*.[14] Daartussenin zitten de mensen bij wie er volgens Lipowski's omschrijving sprake is van somatisatie, en die het grootste deel van de somatisatieproblematiek bij de huisarts representeren.[15] Slechts een klein deel van de mensen die in de huisartsenpraktijk als somatiseerders gekenschetst worden, heeft een somatoforme stoornis conform de DSM-IV.
Rosen heeft een onderverdeling voorgesteld in drie vormen van somatisatie: acute somatisatie, subacute en chronische somatisatie. Acute somatisatie is een kortdurende reactie van dagen tot weken op een manier van omgaan met stressvolle life-events. Bij de subacute vorm is er sprake van somatisatie gedurende een aantal maanden, die in het algemeen gepaard gaat met een behandelbare psychiatrische stoornis of met een oplosbaar psychosociaal probleem. Chronische somatisatie duurt jaren of zelfs levenslang, en voldoet in het algemeen ook aan de criteria van een somatoforme stoornis conform de DSM-IV.[16]

*Hypochondrie* is ook een term die artsen nogal eens hanteren voor patiënten waarbij geen lichamelijke verklaring voor hun klachten kan worden gevonden. Tegenwoordig wordt de term hypochondrie gereserveerd voor één van de zeven strikt omschreven somatoforme stoornissen in de DSM-IV.
Kenmerkend voor hypochondrie is de preoccupatie met de angst of het idee een ernstige ziekte te hebben, en dat deze preoccupatie aanhoudt ondanks adequaat medisch onderzoek en geruststelling. Die overtuiging, die minimaal zes maanden aanwezig dient te zijn, is gebaseerd op een onjuiste interpretatie van één of meer lichamelijke signalen of symptomen.
De preoccupatie leidt tot een verstoring van beroepsmatige en sociale activiteiten en creëert een grote lijdensdruk. Er is echter geen sprake van een waan. De prevalentiecijfers uit onderzoeken variëren van 1-5 procent. Hypochondrie komt bij zowel mannen als vrouwen voor, zonder duidelijke voorkeur voor één van beide geslachten. Meestal ontstaat hypochondrie op jongvolwassen leeftijd, maar ook op latere leeftijd kan hypochondrie zich voor het eerst manifesteren. Het beloop is in het algemeen chronisch, waarbij perioden van heftige symptomen afgewisseld worden met perioden waarin er slechts weinig symptomen zijn. Soms treedt er een blijvende, volledige remissie op van de hypochondrie. Prognostisch gunstige factoren zijn: acuut begin, milde hypochondrische symptomen, de aanwezigheid van somatische comorbiditeit en de afwezigheid van psychiatrische comorbiditeit en van secundaire ziektewinst.
Voor een beschrijving van de overige somatoforme stoornissen die de DSM-IV onderscheidt, wordt verwezen naar hoofdstuk 1 van het Handboek Somatisatie of de DSM-IV.[1,14]

In deze syllabus wordt gekozen voor de term 'lichamelijk onverklaarde klachten', omdat hiermee een brede groep patiënten binnen de definitie valt. Soms zal hier en daar nog wel eens de term *somatisati*e gebruikt worden. De definitie van *somatisatie* volgens Lipowski is hierbij het uitgangspunt.

* Zie bijlage I van de MDR 'SOLK en somatoforme stoornissen".

## 2.3. Het beloop van somatisatie

Het beloop van somatisatie is uiteraard afhankelijk van de ernst van de somatisatie enerzijds en de eventueel aanwezige comorbiditeit anderzijds. Als somatisatie zich voordoet als een reactie op een stressvolle situatie (acute somatisatie) dan is het beloop in het algemeen gunstig en neemt het somatiseren vaak af zodra het verband tussen beide duidelijk is, en vooral wanneer de uitlokkende factor weer onder controle is. Bij een aantal patiënten is er waarschijnlijk sprake van een neiging tot somatiseren, al dan niet op grond van aangeleerd gedrag. In dat geval valt te verwachten dat ze bij een volgende stressvolle periode waarschijnlijk weer zullen somatiseren. Het uiteindelijke beloop zal dan afhangen van de mogelijkheden die ze hebben om het verband te zien tussen klachten en uitlokkende stressvolle situaties, maar ook van hun mogelijkheden om de stressvolle situaties het hoofd te bieden. Het beloop van somatisatie als begeleidend verschijnsel van een angststoornis of een depressie is afhankelijk van het beloop van de onderliggende stoornis. Als de onderliggende psychiatrische stoornis adequaat behandeld en begeleid wordt, neemt het somatiseren vaak af, of vormt in elk geval niet meer de reden voor ongerustheid en herhaald doktersbezoek.
In het algemeen kan gesteld worden dat de somatoforme stoornissen volgens de DSM-IV een langdurig beloop hebben, waarbij de klachten en de beperkingen tengevolge daarvan soms sterk kunnen variëren in de tijd. Slechts zelden treedt er een volledige, blijvende remissie van de stoornis op.

### Eén functioneel syndroom of velen?

Niet alleen komen lichamelijke onverklaarde klachten veel voor, ook worden ze nogal eens als 'syndromen' gelabeld. Veel patiënten met deze 'functionele syndromen' voldoen aan de somatisatie definitie van Lipowski. Het lijkt erop dat elk specialisme zijn eigen syndroom heeft: een cluster van klachten waarvoor vaak ondanks uitgebreid onderzoek naar een mogelijke etiologie geen oorzaak wordt gevonden. Het chronische vermoeidheidssyndroom, het prikkelbaredarmsyndroom, het postwhiplashsyndroom, het premenstruele syndroom, fibromyalgie zijn voorbeelden van dergelijke syndromen. De laatste jaren wordt steeds vaker de vraag opgeworpen of het hier wel gaat om *verschillende* syndromen.[12,17,18] De overlap in definities van de verschillende syndromen en het feit dat veel patiënten die voldoen aan de criteria van het ene syndroom ook blijken te voldoen aan de criteria van een ander syndroom, pleiten tegen het bestaan van onderscheiden syndromen. Ook het gegeven dat patiënten die verschillende syndromen zouden hebben, in een aantal karakteristieken overeenkomen, maakt dat minder waarschijnlijk.[17] Veel patiënten die een syndroomdiagnose hebben gekregen als prikkelbaredarmsyndroom of fibromyalgie zijn van het vrouwelijke geslacht, de enige lichamelijk onverklaarde klachten die vaker door mannen worden gemeld zijn niet-cardiale pijn op de borst en problemen met lopen. Een andere gedeelde karakteristiek is dat psychologische distress en psychiatrische stoornissen, zoals angst en depressie, vaker voorkomen bij mensen met een functioneel syndroom dan in vergelijkbare controlegroepen zonder het betreffende syndroom. Daarbij is uiteraard de vraag naar wat oorzaak en wat gevolg is, niet goed te beantwoorden: leidt het hebben van lichamelijk onverklaarde klachten wellicht tot angst en depressie of is de relatie vooral vice versa? Er komen steeds meer aanwijzingen, dat bij functionele syndromen subtiele verstoringen in het centrale zenuwstelsel een rol spelen, met name in het serotonerge systeem en in de hypothalamus-hypofyse-bijnier-as (HPA-as).

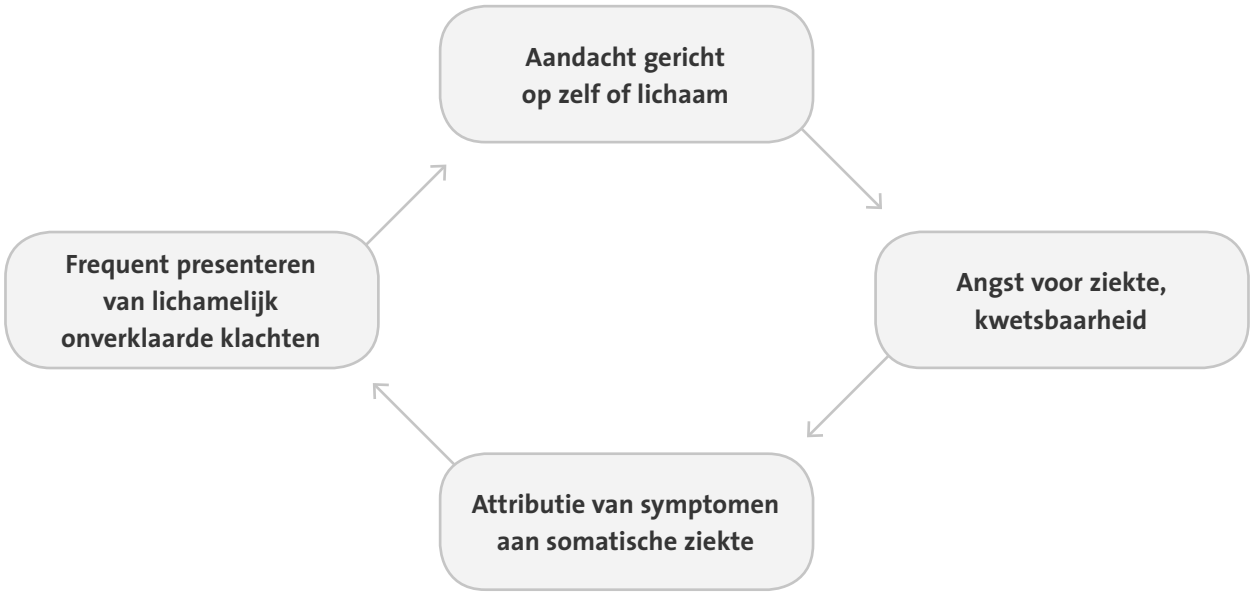
Ook hier kan weer de vraag worden opgeworpen of de genoemde verstoringen oorzaak of gevolg zijn. Waarschijnlijk is er sprake van een wisselwerking tussen klachten, gedrag, psychologische factoren enerzijds en verstoringen van de genoemde systemen anderzijds. Een laatste overeenkomst tussen de functionele syndromen is, dat er aanwijzingen zijn dat ze alle gunstig reageren op dezelfde soort therapieën. Cognitieve gedragstherapie en antidepressiva lijken in de meeste gevallen een beter effect te sorteren dan geen behandeling.[17]
Een nadeel van het 'opdelen' van patiënten in verschillende syndroomcategorieën is, dat de overtuiging versterkt wordt dat er iets aan de hand is, waarvoor uiteindelijk door de medische wetenschap een oplossing gevonden zal worden, als er maar lang en goed genoeg gezocht wordt. Deze overtuiging lijkt naast allerlei andere cognities een belangrijke barrière te zijn in het genezingsproces. Daarnaast is het goed te benadrukken, dat niet alle patiënten die van hun behandelend arts het label diagnostisch syndroom hebben gekregen, voldoen aan de criteria voor somatisatie.

## 2.4 Verklaringsmodellen

Er zijn verschillende verklaringsmodellen beschreven voor het ontstaan en het in stand blijven van somatisatie.

Robbins en Kirmayer beschrijven een *circulair proces* (zie figuur 1).[19]

**Figuur 1**



Fysiologische veranderingen die samenhangen met de spijsvertering, de ademhaling, uitputting en hormonale variaties, resulteren in allerlei diffuse maar ook specifieke lichamelijke reacties. Ook allerlei minimaal pathologische gebeurtenissen kunnen lichamelijke sensaties veroorzaken, zoals misselijkheid en buikkrampen. Lichamelijke veranderingen zijn vaak een begeleidend verschijnsel van emoties, zoals angst die gepaard gaat met onder andere een sneller hartritme en transpireren. Een toegenomen activiteit van het autonome zenuwstelsel kan leiden tot een toegenomen spierspanning, en endocriene processen tijdens emotionele opwindingstoestanden kunnen somatische symptomen veroorzaken zonder dat er sprake is van ziekte of lichamelijke schade.
De eerste stap is dat de aandacht van de patiënt zich richt op zichzelf en zijn lichaam, waardoor lichamelijk sensaties versterkt worden waargenomen. Dit veroorzaakt een gevoel van lichamelijke kwetsbaarheid (hypochondrie). Deze gevoelens geven aanleiding tot met angst beladen cognities: de lichaamssensaties worden toegeschreven aan een ziekte (attributie). Vervolgens bezoekt de patiënt een arts met klachten, waarvoor de arts vaak geen somatische verklaring kan geven. De symptomen blijven echter bestaan en trekken nog meer de aandacht van de patiënt naar zijn lichaam, waardoor een vicieuze cirkel van somatisatie kan ontstaan.

Een ander verklaringsmodel is het *biopsychosociaal (BPS) model*. Dit model is gebaseerd op de theorie van Engel die begin jaren tachtig van de vorige eeuw de biopsychosociale benadering introduceerde.[20]
Het BPS-model gaat er van uit dat de (lichamelijke) sensaties of klachten die iemand heeft, niet alleen maar op een somatisch niveau te verklaren zijn, maar ook betekenis hebben op het niveau van cognities, emoties, gedrag en omgevingsreacties.
Als iemand pijn ervaart, is er niet alleen de pijn, maar er is vaak ook een, in het algemeen negatieve, emotie die met die pijn gepaard gaat. Ideeën die iemand heeft over de oorzaak van de pijn, kunnen de pijn in stand houden, bijvoorbeeld het toeschrijven van de pijn aan een bedreigende ontsteking, of juist verminderen, doordat de pijn gelabeld wordt als voorbijgaand en onschuldig.
Het gedrag dat mensen als gevolg van lichamelijke klachten vervolgens vertonen, wordt eveneens niet alleen beïnvloed door de klachten zelf, maar ook door hun emoties en cognities. Als iemand met nek- en hoofdpijn denkt dat sporten schadelijk is voor de nek, zal hij dat in het algemeen niet gaan doen. Het laatste element van het BPS-model is de omgeving. Reacties uit de omgeving zijn vaak bepalend voor wat ervaren mag worden (buikpijn hebben mag wel, je angstig en incompetent voelen mag niet) en voor de wijze waarop de patiënt met zijn klachten omgaat (lichamelijke klachten zijn een goede reden om thuis te blijven of bedreigende situaties te vermijden).

In het *draagkracht/draaglastmodel* wordt ervan uit gegaan dat mensen individueel verschillen in hun mogelijkheden om allerlei vervelende situaties en gebeurtenissen het hoofd te bieden.[21] Die mogelijkheden, waaronder allerlei vaardigheden en copingstijlen, bepalen, naast de mate van sociale steun en de lichamelijke conditie, de omvang van de draagkracht. De draaglast wordt bepaald door allerlei factoren als persoonlijke, materiële, werkgebonden en sociale problemen. Als de draaglast te groot wordt voor de draagkracht van de persoon in kwestie zal dat in het algemeen leiden tot klachten van zowel lichamelijke als ook psychische aard. Afhankelijk van de interpretatie van deze klachten zal iemand hulp zoeken voor zijn lichamelijke klachten (somatisatie) of voor zijn psychische of sociale problemen.

Duidelijk is dat alle verklaringsmodellen als gemeenschappelijke element hebben dat niet de ervaren klachten, maar *de interpretatie daarvan en het daaruit volgende ziekte- of hulpzoekgedrag* centraal staan in het somatisatieproces. De modellen verschillen naar de mate waarin de lichamelijke klachten verklaard worden en de mate waarin bepaalde factoren (emoties, gedrag, invloeden uit de omgeving, doktersgedrag et cetera) een plaats krijgen.

Hoewel niet alle modellen expliciet *vicieuze cirkels* benoemen is duidelijk dat deze vaak een rol spelen bij het in stand houden van lichamelijk onverklaarde klachten, en daarmee bij het in standhouden van somatisatie.

**Enkele concrete voorbeelden zijn:**

## Rugpijn

**"bewegen is slecht"**

**Meer pijn /**
Letten op pijnsignaal

**Gaan liggen /**
Minder bewegen

*Spieren en gewrichten gaan meer vastzitten*

## Blozen

**"als ze maar niet zien dat.."**

**Rood in gezicht**

**Angst / Stress**

**Vegetatieve reactie:**
*Uitzetten van bloedvaten in gezicht*

Tot slot is het goed stil te staan bij verschillende patiënt – en artsgebonden factoren, die van invloed zijn op het ontstaan en/of in stand houden van lichamelijk onverklaarde klachten.

## Patiëntgebonden factoren zijn:

**Predisponerende factoren**

Mensen die in hun jeugd psychotrauma's hebben meegemaakt zoals affectieve verwaarlozing, geweld, seksueel misbruik zijn kwetsbaarder ten aanzien van het ontwikkelen van onverklaarde klachten.
Ook de manier van omgaan met lichamelijke klachten en ziek zijn in het stamgezin kunnen een gedrag aanleren waarbij de patiënt vaak met allerlei klachten naar de dokter gaat waar een ander dat niet zou doen. Onderzoek naar zowel psychologische als lichamelijke fenomenen laat zien dat patiënten met veel onverklaarde klachten een aantal kenmerken gemeenschappelijk hebben (Lucassen, 2010):

- *Negatief affect*, of neuroticisme, is een vrij stabiel persoonskenmerk. Het is de neiging te snel of te vaak last te hebben van negatieve gevoelens zoals irritatie, boosheid, angst, schaamte, schuld, somberheid, nervositeit of gespannenheid. Mensen met dit kenmerk ervaren meer klachten; ze hebben niet vaker een ziekte. Mensen met een hoge mate van negatief affect ervaren zowel in rust als bij stress veel meer klachten.
- *Hypervigilantie*. Dit is een toestand van verscherpte waakzaamheid voor lichamelijke sensaties. Patiënten met veel onverklaarde klachten scannen hun lijf als het ware om pijn op te sporen. Ze hebben meer aandacht voor wat zich in hun lijf afspeelt. Vaak is die aandacht gefocust op een specifiek onderdeel van dat lijf. Angst is daarbij richtinggevend.
- *Catastroferen*. Dit is de neiging om bij het waarnemen van lichamelijke verschijnselen meteen te denken aan een ernstige oorzaak. Catastroferen gaat gepaard met meer angst en met een kleinere kans dat de klachten vanzelf over zullen gaan. Anderzijds lijken positieve verwachtingen samen te gaan met een grotere kans op herstel. Hypervigilantie en catastroferen spelen een belangrijke rol bij het ontstaan van onverklaarde klachten, maar ook bij de instandhouding ervan.
- *Recall bias*. Dit is het verschijnsel dat een patiënt zich bepaalde dingen uit het verleden niet correct kan herinneren. Patiënten met veel onverklaarde klachten blijken zich veel meer klachten te 'herinneren' dan ze op het moment zelf hadden.
- *Veranderde waarneming* en doorgifte van prikkels in het centraal zenuwstelsel. Bij mensen met onverklaarde klachten blijkt een grotere gevoeligheid te bestaan voor het waarnemen en doorsturen van lichamelijke verschijnselen.

**Uitlokkende factoren**
Lichamelijke klachten, trauma's fysieke stress, vermoeidheid en overbelasting, maar ook psychische stress en life events kunnen allemaal het beginpunt zijn van een langdurige episode met onverklaarde klachten.

**In stand houdende factoren**
Factoren die predisponerend zijn voor het ontstaan van persisterende klachten zijn vaak ook factoren die de klachten in stand houden. Daarnaast zijn een slechte lichamelijke conditie, slaapgebrek en inactiviteit belangrijke 'in stand houders'. Tenslotte zijn juridische processen berucht om hun effect op het persisteren van de klachten.

## Artsgebonden factoren

Dokters worden vaak buiten beschouwing gelaten als in stand houder van lichamelijk onverklaarde klachten. Uit de definitie van somatisatie komt zelfs naar voren dat het hier gaat om een in de patiënt gelegen neiging. Toch is er een traditie binnen de geneeskunde geweest die structureel aandacht heeft gegeven aan de rol van de dokter. Het gaat om de lijn die aangeduid wordt met de term 'somatische fixatie' (Grol,1983). Met die term gaf men aan dat de dokter door al te eenzijdig somatisch te reageren op de klachten van de patiënt, die patiënt zou fixeren in het somatische spoor. Dit zou ertoe leiden dat de veronderstelde psychosociale oorzaak buiten het gezichtsveld van de patiënt bleef. De opvatting was dat psychosociale stress (in de zin van problemen in de relatie of op het werk) de achterliggende – en niet door de patiënt herkende – oorzaak is van onverklaarde klachten. Deze opvatting is nog steeds wijdverbreid onder artsen. (Lucassen, 2010). Dokters zien het vaak presenteren van onverklaarde klachten als uiting van een psychiatrisch probleem of persoonlijkheidsstoornis. Dokters denken vaak dat de klachten niet het eigenlijke probleem zijn, maar dat er iets achter of onder zit. Ze zijn sceptisch als het gaat om klachten waarvoor geen objectieve oorzaak wordt gevonden. Daarnaast hebben veel dokters het gevoel dat patiënten met onverklaarde klachten druk op hen uitoefenen en snel om allerlei medische interventies vragen zoals een recept, aanvullend onderzoek of een medische ingreep. Patiënten voelen de scepsis van de dokters. Zij willen niet zozeer medische interventies als wel goede communicatie en een goede verstandhouding. Uit onderzoek van consulten blijkt dat de verschillende gezichtspunten van arts en patiënt tot problemen kunnen leiden. Huisartsen en specialisten blijken bij patiënten met onverklaarde klachten veel minder goed te communiceren dan bij patiënten met wel verklaarde klachten. Ze exploreren de klacht minder, gevoelens en ideeën van de patiënt komen veel minder aan bod. Daarentegen blijken niet patiënten maar artsen de oorzaak te zijn van een groter percentage medische interventies: dokters bieden patiënten soms ongevraagd een recept, een verwijzing, of de mogelijkheid tot nader onderzoek aan. Patiënten met onverklaarde klachten willen vooral een dokter die empathisch is en goed communiceert, terwijl de dokters juist minder empathisch bleken te reageren bij mensen met onverklaarde klachten (Salmon, 2004). Het is goed denkbaar dat minder goed communiceren door dokters het hebben van klachten – en de geneigdheid om terug te komen – bevordert (Lucassen, 2010).

# 3. Consultvoering met cognitief gedragstherapeutische technieken

## 3.1 Algemene principes en begrippen

In de modellen over het ontstaan en in stand houden van somatisatie spelen emoties, cognities en gedrag een belangrijke rol (hoofdstuk 2.4).
Het systematisch aandacht geven aan emoties, cognities en vermijdingsgedrag van een patiënt heeft tot doel: preventie of vroegtijdige onderkenning van een proces van somatisatie.[22,23]

Met technieken ontleend aan de cognitieve en de gedragstherapie (vaak in combinatie toegepast als cognitieve gedragstherapie) is het mogelijk om de circulaire processen die hieraan ten grondslag liggen te doorbreken.
De effectiviteit van deze therapievormen is, in tegenstelling tot andere psychotherapieën, uitgebreid onderzocht en voor verschillende toepassingsgebieden ook aangetoond.[24,25,26]
De op gedrag gerichte technieken zijn over het algemeen gebaseerd op *leertheoretische modellen*. Twee principes die hierin centraal staan zijn: gewenst gedrag wordt gestimuleerd door te belonen, en: ongewenst gedrag dooft niet uit door te straffen, maar wel door het te negeren.
*Vermijding* (een geconditioneerde gedragsrespons op een als onaangenaam ervaren stimulus) komt bij patiënten met lichamelijk onverklaarde klachten veel voor. Vermijdingsgedrag kan met gedragstherapie goed behandeld worden door middel van *exposure* (confrontatie met een als bedreigend ervaren situatie). Deze techniek berust op het gegeven dat bij langdurige blootstelling aan een stimulus (bijvoorbeeld een vermeden situatie) gewenning optreedt (*habituatie*) en de respons (de klacht) uitdooft (*extinctie*).
Uit de cognitieve hoek komt de notie dat specifieke opvattingen van de patiënt ten opzichte van de arts en diens hulpverlening evenals denkbeelden over de klachten, gerekend worden tot de belangrijkste factoren bij het blijven voortbestaan van klachten. Centraal punt in de cognitieve therapie is dat deze opvattingen over ziekte en gezondheid getoetst moeten worden. De patiënt leert dat er samenhang is tussen gedachten, gevoelens en gedrag.
Het bijhouden van een *klachtendagboek* waarin, naast de klachten, ook *cognities* (gedachten, overtuigingen, met emoties geladen ideeën) en gevoelens worden geïnventariseerd, is een techniek die daar meer inzicht in kan geven.
Door te vragen naar gedachten over de klachten kan men ook *klachtenverergerende cognities* (dramatiserende of catastroferende) op het spoor komen. De arts kan dan helpen om dramatiserende gedachten te veranderen in meer accepterende gedachten, of catastroferende gedachten uit te dagen. Zo wordt de patiënt aangemoedigd om catastroferende gedachten of misinterpretaties op realiteitswaarde te beoordelen en bij te stellen.

Ook voor de specialist blijkt het toepassen van deze cognitief gedragstherapeutische technieken  een waardevol instrument in de begeleiding van patiënten met lichamelijk onverklaarde klachten.

## 3.2. Cognitief gedragstherapeutische technieken in medische consultvoering

De laatste decennia is er veel onderzoek gedaan naar manieren om de gevolgen van lichamelijk onverklaarde klachten te beïnvloeden. Ook het omgaan van patiënten met chronische pijnklachten, zoals bijvoorbeeld bij artrose of reumatoïde artritis, is onderwerp van onderzoek geweest.
Waar 'somatische' aanpak van pijn en symptomen onvoldoende baat heeft, dient de behandeling zich vooral te richten op een optimaal functioneren van de patiënt *ondanks de klachten*. De hierbij toegepaste behandelvormen zijn vooral gebaseerd op cognitieve gedragstherapie. Cognitieve gedragstherapie wordt al langer toegepast bij de behandeling van stemmings- en angststoornissen en is daar aangetoond effectief.[27,28,29,30] Voor toepassing bij (chronische) whiplash, vermoeidheid of lage-rugpijn, fibromyalgie, IBS, RSI, het premenstrueel-, hyperventilatie- en globussyndroom en atypische thoracale bezwaren, zijn interventies ontworpen op basis van de belangrijkste behandelprincipes van cognitieve gedragstherapie.[31,32,33]

Interventies richten zich vooral op de factoren die het beloop van de klachten kunnen beïnvloeden:

- *Cognities* zijn in ruime zin alle gedachten van een patiënt over de klachten. Hieronder vallen attributies (het toeschrijven van de klachten aan lichamelijke aandoeningen) en verwachtingen over het beloop van de klachten en over de beheersingsmogelijkheden van de pijn. Patiënten met catastroferende gedachten schatten hun eigen mogelijkheden om de pijn te beïnvloeden vaak erg laag in, en leveren zich over aan de pijn. Dit kan gepaard gaan met *emoties* zoals ontmoediging.
- *Emoties* vormen vervolgens de drijfveren voor het gedrag.
- *Gedragsmatige gevolgen* bestaan vaak uit vermijding van activiteiten en situaties uit angst dat de klachten erger zullen worden.

Inadequate cognities en daarmee samenhangend vermijdingsgedrag kunnen in korte tijd leiden tot conditieverlies door verminderde activiteit, en tot disfunctioneren in gezin, werk en hobby's. Bij cognitieve gedragstherapie worden op een actieve, probleemgeoriënteerde wijze vooral die cognities, emoties en gedragsmatige gevolgen geïdentificeerd en aangepakt die de klachten in stand houden. Uit onderzoek bij patiënten met het prikkelbaredarmsyndroom bleek dat het bespreken van ongerustheid en vermijdingsgedrag leidde tot minder ongerustheid en vermijdingsgedrag en tot een afname van de medische consumptie.[24] Bij patiënten met chronische aspecifieke lage-rugpijn leidde een stapsgewijze, gedragstherapeutische benadering tot minder disfunctioneren, ziekteverzuim en medische consumptie.[34,35] Gedragstherapeutisch georiënteerde interventies met oefentherapie bewerkstelligden bij patiënten met gonartrose toegenomen pijntolerantie en grotere kennis van de aandoening, een adequate omgang met de klachten en verminderde medische consumptie.[36,37] Een fysiek actieve levensstijl heeft bij chronische pijnsyndromen gunstige effecten op het functioneren en de pijn, in tegenstelling tot een afhankelijke opstelling (passieve coping) met bijvoorbeeld veel rusten.[34,38] In het algemeen schaadt (aangepast) werken niet en vergemakkelijkt (arbeids)participatie het omgaan met de klachten door effecten zoals afleiding, satisfactie en behoud van conditie.

In richtlijnen die het Nederlands Huisartsen Genootschap de afgelopen jaren gemaakt heeft, zijn cognitief gedragstherapeutische principes steeds meer verwerkt in het aanbevolen beleid voor de huisarts.[39,40,41,42] Essentieel onderdeel van het beleid is dat de huisarts onjuiste cognities, angsten en vermijdingsgedrag identificeert en aanpakt, daarbij gebruik makend van zijn of haar kennis van de patiënt en zijn omgeving. Daarnaast adviseert de huisarts medicamenteuze, ergonomische of psychologische maatregelen ter verlichting van de klachten binnen een activerende en op zelfredzaamheid gerichte aanpak: op deze wijze worden gedragstherapeutische principes gecombineerd met ondersteunende en persoonlijke coaching.
De cognitief gedragstherapeutische technieken, die voor de medisch specialist in deze training geselecteerd zijn, beperken zich tot het exploreren en informeren van de patiënt.

# 4. Exploreren

## 4.1 Communicatie

Mevrouw Bruinsma heeft last van een veelheid aan lichamelijke klachten. Zo heeft zij al vele jaren last van pijnlijke spieren en gewrichten en de laatste tijd speelt vooral haar buik op. Daarnaast is ze duizelig, heeft ze regelmatig hoofdpijn en soms ook pijn op de borst. Voor deze klachten bezoekt zij vaak haar huisarts, dan eens voor haar spieren, dan voor haar buikpijn en een andere keer weer voor duizeligheid. De huisarts verwijst mevrouw Bruinsma diverse keren door naar een medisch specialist. Aanvullend specialistische onderzoeken leveren geen medische verklaring voor haar klachten op. De specialist verwijst mevrouw Bruinsma terug naar de huisarts.

Het blijkt dat artsen bij patiënten met onverklaarde klachten veel minder goed communiceren dan bij patiënten met verklaarde klachten: ze exploreren de klacht minder goed, en gevoelens en opvattingen van de patiënt komen minder aan bod. Artsen kunnen sceptisch zijn als het gaat om klachten waarvoor geen objectieve verklaringen gevonden worden. Daarnaast hebben veel dokters het gevoel dat patiënten met veel onverklaarde klachten druk op hen uitoefenen, en om allerlei medische interventies en aanvullend onderzoek vragen. Patiënten voelen de scepsis van de dokter, terwijl zij niet per se vragen om medische interventies, eerder verlangen ze naar patiëntgerichte zorg met goede communicatie over de klachten en een duidelijk advies. Het blijkt echter dat huisartsen vaker medische interventies aanbieden dan dat patiënten er om vragen. Aan u als specialist om de huisarts en de patiënt te ondersteunen met noodzakelijke informatie over de klachten en beleid.
Het verbeteren van de communicatie tussen de arts en de patiënt met onverklaarde klachten vermindert de kans op wederzijdse irritatie, heeft een betere arts- patiënt relatie tot gevolg en maakt de behandeling effectiever.
Een betere communicatie houdt in dat de dokter de patiënt serieus neemt en structureel en systematisch aandacht besteedt aan lichamelijke, emotionele, cognitieve, gedragsmatige en sociale aspecten van de klachten. Daarnaast is ook de fasering in een consult van belang: als de dokter goed heeft doorgevraagd kan deze vervolgens beter aansluiten met informatie. Mix je bij patiënten met onverklaarde klachten het exploreren met uitleg geven en advies dan is de kans groot dat de patiënt zich minder gehoord voelt en de dokter minder relevante informatie verkrijgt dan wenselijk is.

## 4.2 Exploreren volgens SCEGS analyse

In de exploratiefase vormt de specialist zich een zo gedetailleerd mogelijk beeld van de klachten van de patiënt en van zijn verwachtingen van de hulpverlening.
De verwijsbrief van de huisarts of collega biedt de specialist in een eerste consult informatie over de klachten en hulpvraag van de patiënt. Tijdens zo'n eerste consult is het van belang de patiënt goed uit te vragen zowel over zijn klachten, verwachtingen van het consult, gedachten over de klacht, als ook aandacht te besteden aan wat hij niet meer doet en hoe de omgeving reageert op de klachten.
Deze factoren spelen allemaal hun eigen rol bij het beloop van klachten en bij de daarvoor in te zetten behandeling. Ze dienen dan ook zo expliciet mogelijk aan de orde te komen. De SCEGS analyse die in Nijmegen is ontwikkeld, biedt een handvat voor deze fase.[43,44]

De S staat voor somatisch: het uitdiepen van de klachten met behulp van de speciële anamnese en gericht lichamelijk onderzoek. De arts kan zich hiermee een goed beeld vormen, en de patiënt voelt zich serieus genomen. Pas als de patiënt ervan overtuigd is dat de arts zijn klachten goed begrepen heeft, ontstaat er ruimte om ook andere aspecten van de klachten, zoals cognities, te bespreken.

De C staat voor cognities: wat ziet de patiënt als verklaring voor zijn klachten, heeft hij zelf ideeën over de mogelijke oorzaak? Ziet hij zichzelf als iemand die vatbaar is voor ziektes, denkt hij dat bepaald gedrag ziektebevorderend is, of de klachten verergert? (In de Engelse literatuur wordt hiervoor de term 'fear avoidance beliefs' gebruikt: bijvoorbeeld bewegingsangst vanuit de overtuiging dat bewegen slecht is bij rugklachten en de aandoening en de klachten verergert.)
Als de eigen ideeën van de patiënt over de klachten en hun oorzaak niet goed aan bod komen in de exploratieve fase dan is de kans groot dat de uitleg die de arts over de klachten geeft, niet aanslaat. Er blijven dan twee verklaringen naast elkaar bestaan. De patiënt heeft de uitleg van de arts wel gehoord, maar neemt hem niet aan omdat hij zijn eigen verklaring blijft koesteren en niet overtuigd is van de onjuistheid daarvan.

De E staat voor emoties. Wat doet de klacht met iemand? Wordt hij er wanhopig, moedeloos of juist opstandig van. Is hij erg ongerust over de klachten? Waarover maakt hij zich dan precies ongerust? Wat is de aanleiding voor die ongerustheid? Emoties rond de klachten, maar ook catastroferende gedachten ('deze pijn gaat nooit meer over') kunnen een negatieve invloed op het klachtenbeloop hebben zonder dat patiënt of arts zich dit bewust zijn. Er is maar één manier om erachter te komen: er actief naar vragen.

De G staat voor gedrag. Wat doet iemand als hij klachten heeft? En helpt dat of juist niet? Zijn er activiteiten die achterwege gelaten worden sinds er klachten zijn of toen de klachten toenamen? Welke activiteiten? Wat is de reden om ze achterwege te laten? Denkt de patiënt dat ze schadelijk zijn, of heeft hij ook daadwerkelijk gemerkt dat ze de klachten verergeren? Vermijdingsgedrag kan klachtenbestendigend werken en zal in de meeste gevallen niet spontaan verdwijnen. Veel patiënten noemen hun vermijdingsgedrag niet uit zich zelf. Mensen kunnen ook als het ware geruisloos hun activiteiten aanpassen en zijn zich daar niet eens echt bewust van. De enige manier om zeker te weten of er sprake is van vermijdingsgedrag is er actief naar vragen.

De tweede S staat voor sociaal. Welke gevolgen hebben de klachten in sociaal opzicht: hoe reageert de omgeving erop. Bezorgd? Overbezorgd? Negatief? Hoe gaat het op het werk en thuis? Kan de patiënt nog werken met de klachten, naar tevredenheid functioneren in allerlei opzichten? Soms vertellen patiënten spontaan hoe hun partner, kinderen of vrienden reageren op de klachten. Vaker wordt daar niets over gezegd, terwijl de invloed van deze reacties niet onderschat moet worden. Overbezorgdheid kan er toe leiden dat mensen meer ziektegedrag en vermijdingsgedrag vertonen dan nodig is. Onverschilligheid kan er toe leiden dat de klachten met meer kracht gebracht moeten worden om de gewenste aandacht te krijgen, met als onvermijdelijk gevolg dat ze ook als ernstiger ervaren worden.

## 4.3 Gespreksvaardigheden

Naast de specifieke inhoud van de exploratiefase, die wordt weergegeven door de SCEGS, zijn er ook specifieke gespreksvaardigheden die in deze fase van belang zijn. De actief luisterende houding vormt de basishouding voor de exploratieve fase. Meestal wordt ervan uitgegaan dat in deze fase de patiënt de agenda en de gespreksonderwerpen bepaalt. Dat is slechts ten dele het geval, want de arts zal in deze fase ook actief naar informatie over de vijf elementen van het SCEGS analyse moeten vragen. Hiervoor is nodig dat hij de vijf elementen en de vragen die daarbij horen in zijn hoofd heeft en ook weet waarvoor ze dienen.
De onderstaande gesprekstechnische vaardigheden kunnen van pas komen.

### Stimulerende gesprekstechnieken.

Op vragende toon letterlijk het laatste woord of de laatste paar woorden van de patiënt herhalen (papagaaien).
Letterlijk hele zinnen herhalen, roept irritatie op.
Knikken, hummen of andere instemmende gebaren of geluiden maken.
Stiltes laten vallen, niet zo lang dat ze de patiënt (of de arts) een onbehagelijk gevoel kunnen geven, maar lang genoeg om de patiënt de gelegenheid te geven zijn eigen verhaal aan te vullen of uit te diepen.

### Verhelderende gesprekstechnieken.

Laten specificeren: 'Hoe ging dat precies?'
Parafraseren, met andere woorden weergeven wat de patiënt gezegd heeft.
Voorbeelden vragen.
Open vragen stellen die geen voorgeprogrammeerd antwoord veronderstellen.

- Doorvragen: 'Wat bedoelt u met....'

### Ordenende gesprekstechnieken

Samenvatten. Als de samenvatting vragenderwijs geformuleerd wordt, krijgt de patiënt de gelegenheid correcties of aanvullingen te geven.
Structureren door te proberen hoofd- en bijzaken gescheiden te houden.
Interpreteren, bij voorkeur op vragende wijze, om de patiënt de gelegenheid te geven een verkeerde interpretatie te corrigeren.

Tijdens de speciële anamnese, die ook een onderdeel van de exploratie vormt, zijn gesloten vragen meer geëigend, omdat de arts dan specifieke informatie wil hebben over de klachten om één of meer specifieke diagnosen te kunnen bevestigen of uitsluiten.

## Voorbeelden voor doorvragen van klachten volgens SCEGS analyse

Somatische dimensie: vraag naar klachtgegevens zoals de aard, lokalisatie, intensiteit, frequentie en duur van de klacht (speciële anamnese), daarnaast dient lichamelijk onderzoek en eventueel laboratorium- of röntgenonderzoek plaats te vinden.

Cognities:

- Wat is er volgens u aan de hand, waar denkt u zelf aan?
- Hebt u zelf 'n idee waar uw klacht vandaan komt? Wat dan?
- Denkt u dat u iets (een ziekte) onder de leden hebt? Waar denkt u dan aan?
- Denkt u wel eens 'als 't dat maar niet is'? Wat dan?
- Denkt u dat u vatbaar bent voor bepaalde ziektes? Waar denkt u dan aan?
- Maakt u zich zorgen over uw klacht? Wat denkt u dan?
- Bent u ongerust over uw klacht? Waarover maakt u zich dan precies ongerust?
- Als u dan die klachten krijgt, wat vindt u daar dan van, wat gaat er dan door u heen?
- Kunt u uw klachten (in positieve of in negatieve zin) beïnvloeden (door bepaald gedrag of door bepaalde gedachten)? Om welke gedragingen of gedachten gaat het dan?
- Denkt u dat bepaald gedrag (b.v. bewegen) de klachten (of veronderstelde ziekte) verergert? Welke gedragingen zijn dat dan?
- Hebt u zelf greep op uw klachten? Hoe?
- Hoe zullen uw klachten (of veronderstelde ziekte) zich volgens u ontwikkelen?
- Wat verwacht u van mij?
- Wat zou ik voor u kunnen doen?'

Emoties:

- Wat doet de klacht met u (wordt u er b.v. angstig, onzeker, wanhopig, moedeloos, neerslachtig, boos of opstandig van)?
- Nu u die klachten heeft, hoe voelt u zich daaronder?
- Wordt het u wel eens teveel, nu u die klachten heeft?
- Brengen die klachten u wel eens uit het evenwicht/uit uw doen?'

## Voorbeelden voor doorvragen van klachten volgens SCEGS analyse

Gedrag:

- Wat doet u als u klachten hebt? Helpt dat of juist niet?
- Als u die klachten hebt, hoe reageert u daar dan op?
- In welke activiteiten, en in welke mate, wordt u belemmerd door uw klachten?
- Wat doet u om uw klachten te verminderen? In welke mate lukt u dit?
- Zijn er activiteiten die u achterwege laat/vermijdt sinds u (meer) klachten hebt? Waarom, denkt u dat die activiteiten schadelijk zijn? Welke activiteiten zijn dat dan?
- Hebt u ook daadwerkelijk ervaren dat die activiteiten uw klachten verergeren?
- Wat doet u niet, of niet meer, als u klachten heeft? Sinds wanneer is dat zo?

Sociaal:

- Wie weet er van dat u deze klachten heeft?
- Praat u over uw klachten? Met wie? En wat zegt u dan? Helpt u dat?
- Welke gevolgen hebben uw klachten in sociaal opzicht?
- Zijn uw sociale activiteiten toegenomen of afgenomen door/sinds uw klachten?
- Wat merkt uw omgeving van uw klachten? Hoe merken ze dat? Hoe laat u ze het merken? Wie merkt/merken het vooral?
- Hoe reageert de omgeving op uw klachten (bemoedigend, behulpzaam, beschermend, adviserend, waarschuwend, bezorgd, overbezorgd, omzichtig, onverschillig, negatief)?
- Wat denkt uw omgeving over uw klachten? Wie reageert/reageren zo?
- Waar denkt uw partner dat uw klachten vandaan komen?
- Hoe reageerden de mensen in uw omgeving erop toen u vertelde wat het bezoek aan de dokter had opgeleverd? En hoe staat u daar nu zelf tegenover?
- Hoe gaat het op het werk en thuis, hoe functioneert u daar? Hoe denken anderen daarover?
- Kunt u werken met de klachten? Hoe functioneert u op het werk? Hoe denken anderen daarover?

# 5. Uitleg geven

## 5.1 Informeren

Duidelijke informatie verstrekken over de klachten, over wat dokters weten over de oorsprong en over het beloop ervan, is een belangrijk onderdeel van de fase na de exploratie.
In deze fase kan de dokter medisch onjuiste cognities van de patiënt corrigeren door het aanbieden en bespreken van bijvoorbeeld alternatieve verklaringen voor de klachten.
De arts start deze fase met een samenvatting van de bevindingen uit anamnese en lichamelijk onderzoek en geeft weer welke verklaring hij voor de klachten heeft op basis van anamnese en onderzoek. Bij het geven van informatie dient de huisarts niet alleen aan te sluiten bij de informatiebehoefte van de patiënt, maar ook informatie te geven over de elementen uit de exploratieve fase, zoals wanneer er sprake blijkt te zijn van een verschil tussen wat de arts en de patiënt ervan vindt of weet.

### Geruststellen

Pas als de arts na de exploratieve fase voldoende weet over de ongerustheid, maar ook over de klachten, kan hij overgaan tot geruststellen. Soms kan een patiënt goed aangeven wat ervoor nodig is om hem gerust te stellen. Geruststellen houdt in: goede uitleg geven over de klachten en onderbouwen waarom de ongerustheid van de patiënt in dit geval wel voorstelbaar, maar gelukkig niet terecht is. Het houdt ook in, dat de arts expliciet aangeeft welke argumenten pleiten tegen de aandoening waaraan de patiënt denkt, of tegen het beloop van de klachten of de gevolgen waarover de patiënt zich zorgen maakt.
Als de uitslagen van aanvullende onderzoeken bekend zijn, vertel dan wat de patiënt **wel** heeft en gebruik daar zo mogelijk positieve bewoordingen voor. Uit onderzoek is duidelijk gebleken dat patiënten met een onverklaarde klacht vaker beter worden als de dokter positieve bewoordingen gebruikt, duidelijk maakt dat hij weet wat er aan de hand is en als de dokter vertelt dat de prognose gunstig is. Vertel ook wat een patiënt **niet** heeft, maar alleen als een bepaalde angst ter sprake is gekomen. Zeg dus niet ‘u heeft geen kanker’ als dit niet in het consult ter sprake kwam.
Bij het geruststellen van patiënten met lichamelijk onverklaarde klachten is het in het algemeen van belang te vertellen dat:

1. lichamelijke sensaties of symptomen veel voorkomen bij mensen
2. deze vaak niet het gevolg zijn van een ziekte of een psychosociaal probleem en
3. het ontbreken van een lichamelijke verklaring niet automatisch impliceert dat de oorzaak van de klachten psychisch of psychosociaal is.

### Bijstellen van verwachtingen

Voorwaarde voor het kunnen bijstellen van te hoge verwachtingen is, dat de patiënt zijn verwachting zo duidelijk mogelijk kenbaar maakt. De arts kan vervolgens navragen waarop die verwachting gebaseerd is. Beschikt de patiënt over specifieke informatie over behandelingsmogelijkheden, of is het een diffuse verwachting (‘er kan zoveel tegenwoordig, dus graag een paardenmiddel tegen ...’). Baseert de patiënt zijn verwachting op eigen vroegere ervaringen of op ervaringen en verhalen van anderen? Pas als dit duidelijk is, kan de arts de verwachting bijstellen door aan te geven in welk opzicht de situatie van de patiënt verschilt van die van andere mensen, of in hoeverre de huidige situatie verschilt van vroegere situaties. De arts kan diffuse verwachtingen bijstellen door expliciet de beperkingen van de geneeskunde bij veel klachten, en in het bijzonder dit soort klachten te benoemen.

## Bijstellen van cognities

Ook cognities kunnen pas bijgesteld worden als ze expliciet gemaakt zijn. Waar baseert de patiënt ze op: kennis, ervaring, verhalen van anderen? De specialist kan samen met de patiënt de plausibiliteit ervan nagaan. Zijn er alternatieve verklaringen mogelijk? Door uit te leggen waarom een bepaalde gedachtegang niet klopt of niet kan kloppen, kan de patiënt verleid worden deze los te laten.

## Bespreken van vicieuze cirkels

Vaak spelen bij het in stand houden van klachten vicieuze cirkels een rol. Uitleggen hoe een dergelijke cirkel in deze specifieke situatie bij deze patiënt eruit kan zien, is een belangrijk onderdeel van deze fase. Als voorbeeld kan de vicieuze cirkel tussen ongerustheid en klachten dienen. Als mensen zich ongerust maken over hun klachten, is hun aandacht gefocust op de klachten. Elke verandering daarin wordt waargenomen en kan aanleiding zijn tot aanwakkeren van de ongerustheid. Door de toegenomen ongerustheid zijn mensen zich nog weer meer bewust van allerlei lichamelijke sensaties hetgeen een uitbreiding van het klachtenpatroon kan veroorzaken. Dit biedt weer nieuwe brandstof voor de ongerustheid.

## Richtlijnen voor informatie geven

Uit onderzoek is bekend dat maar een klein deel van de gegeven informatie blijft hangen bij de patiënt.
De kans dat informatie beklijft neemt toe, als aan een paar voorwaarden wordt voldaan.

- De informatie dient in een voor de patiënt begrijpelijke vorm gegeven te worden, dat wil zeggen, dat het taalgebruik op de patiënt moet worden afgestemd.
- De informatie dient specifiek op de situatie van de patiënt toegesneden te zijn, dus zoveel mogelijk aansluiten bij wat de patiënt in de eerste fase verteld heeft.
- De informatie dient gedoseerd gegeven te worden met tussendoor af en toe gelegenheid om de patiënt te laten reageren.
- Waar mogelijk dient de informatie niet alleen mondeling, maar ook schriftelijk gegeven te worden.

## Effectief geruststellen

Geruststellen is iets dat iedere dokter dagelijks doet en dat heel vanzelfsprekend lijkt. Ook in de literatuur over gespreksvoering is het onderwerp vaak beperkt tot de instructie:"U stelt de patiënt gerust". Voor een slecht nieuws gesprek bestaan concrete richtlijnen, maar hoe voert een arts een goed nieuws gesprek?
De Engelse psychiater Bass geeft de volgende aanwijzingen voor effectief geruststellen:[45]
Aan geruststellen moet een aantal zaken vooraf zijn gegaan.

- Laat merken dat u de bezorgdheid van de patiënt gehoord hebt.
- Doe lichamelijk onderzoek.
- Vertel uw conclusies, zodat de patiënt weet waarop je geruststelling gebaseerd is.
- Vertel de patiënt dat u ervaring hebt met dit soort problemen; dat maakt het voor de patiënt gemakkelijker om met uw oordeel mee te gaan.
- Zwak de ongerustheid van de patiënt niet af ('ik begrijp dat u zich een beetje bezorgd maakt'). Beter is juist de bezorgdheid versterkt weer te geven ('ik heb de indruk dat u erg bang bent dat dit weer een hartaanval wordt'). De patiënt voelt zich meer (h)erkend. Dit vereist meestal wel enige oefening.

## Maatwerk

Voor sommige patiënten is geruststelling gericht op een concrete angst voldoende om weer verder te kunnen; niet de intensiteit van de klacht maar de angst voor iets ernstigs was blijkbaar de aanleiding voor het bezoek aan de dokter. Sommige patiënten willen wel iets meer, bijvoorbeeld precies weten hoe het dan kan dat er een klacht is zonder ziekte. Kortom, een goede uitleg is maatwerk. De arts kan bij de uitleg gebruik maken van bijvoorbeeld:

1. *fysiologische en psychologische mechanismen*
2. *metaforen*
3. *vicieuze cirkels en neerwaartse spiralen*

## Fysiologische en psychologische mechanismen

Waarschijnlijk hebben veel patiënten graag dat de dokter een verklaring geeft voor de klacht die dichtbij het lijf ligt. Hiervoor kan de arts gebruik maken van een aantal (psycho)fysiologische mechanismen:

- selectieve aandacht
- sensitisatie
- HPA-as: hypothalamic-pituitary-adrenal axis (hypothalamus-hypofyse-bijnier as)

Een voorbeeld van hoe effectief geruststellen en uitleggen verwoord kunnen worden als er sprake is van **selectieve aandacht:** *U hebt geen ziekte, daar ben ik zeker van – en u ook naar ik afleid uit wat u zei – maar wel klachten, dat is een situatie die vaak voorkomt. Soms kan het met aandacht te maken hebben: je hersenen zijn als het ware gefocust op een bepaalde plek in je lijf en die blijf je dan voelen; soms heeft het te maken met dingen die zich in uw leven afspelen en die veel energie vergen.* Deze uitleg kan effectief zijn bij hartkloppingen. Patiënten met hartkloppingen zijn vaak selectief waakzaam voor signalen uit de borststreek en merken dus veel meer op van wat zich daar afspeelt. Bij het proces van selectieve aandacht kan de metafoor van de filter worden ingezet:
*er spelen zich vele processen af in het lijf en het is maar goed dat u die niet allemaal voelt, anders zou u zich niet meer bezig kunnen houden met wat er om u heen gebeurt; er is dus een soort filter die voorkomt dat u alle sensaties uit uw lijf voelt; die filter zorgt ervoor dat alleen de belangrijke signalen doorkomen; bij sommige mensen staat de filter open voor signalen uit het hart omdat ze pas iets hebben meegemaakt op dat gebied, bijvoorbeeld dat een familielid hartkloppingen had en daarna in het ziekenhuis belandde met een ernstige hartkwaal.*

**Sensitisatie** is de geneigdheid tot een versterkte reactie op (pijnlijke) stimuli, als gevolg van eerdere ervaringen met die stimuli. Dit kan er toe leiden dat niet-pijnlijke prikkels als pijnlijk worden ervaren. Dit is een fysiologisch mechanisme, dat mogelijk een rol speelt bij het prikkelbare darmsyndroom en bij (andere) chronische vormen van pijn. Het geeft patiënten veel steun te horen dat dit een fysiologische reactie is, omdat velen het gevoel hebben als aansteller te worden gezien.

Bijvoorbeeld; *u hebt pijn over een veel groter gebied dan in het begin van uw ziekte; dat is een normale manier van reageren van uw zenuwstelsel; het zenuwstelsel van iedereen zorgt ervoor dat bij langdurige pijn op een plek, de omgeving van die plek gevoeliger wordt; dat kan soms zo erg zijn dat prikkels die gewoonlijk niet pijnlijk zijn (aanraking) dat wel worden.*

## Metaforen

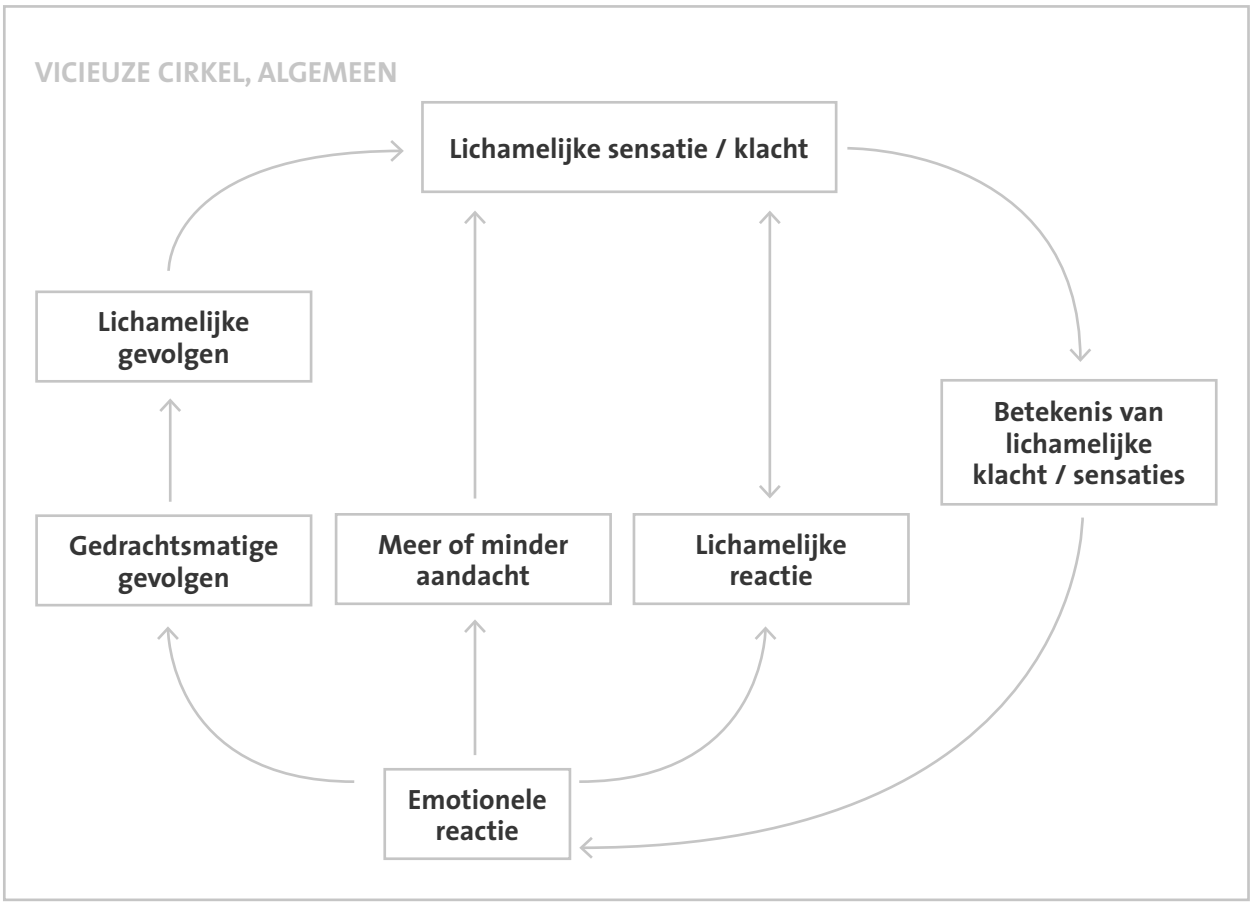
Soms helpt het mensen een metafoor voor te houden. Eenvoudige metaforen zitten bijvoorbeeld in spreekwoorden als 'je buik ervan vol hebben', 'teveel aan je hoofd hebben' en 'teveel op je nek hebben'. Een volgend voorbeeld is dat de rugzak te vol zit. Of: *als een lift te vol zit, gaat er een lampje branden; het heeft geen zin om dat lampje te gaan onderzoeken, want dat is maar een signaal; je moet op zoek naar de oorzaak van dat brandende lampje.* Artsen hebben doorgaans een vast repertoire aan metaforen; in de training is het leerzaam die met elkaar uit te wisselen.

## Bestanddelen van vicieuze cirkels bij onverklaarde klachten

Een zeer bruikbare manier om de patiënt te informeren over zijn klachten en daarmee samenhangende beperkingen bestaat uit het presenteren van de verzamelde informatie in een of meerdere vicieuze cirkels. Vicieuze cirkels zijn bijzonder geschikt om in stand houdende factoren te verduidelijken. Een vicieuze cirkel om lichamelijk onverklaarde klachten en de daaraan gerelateerde aspecten uit te leggen bestaat gewoonlijk uit de volgende onderdelen:

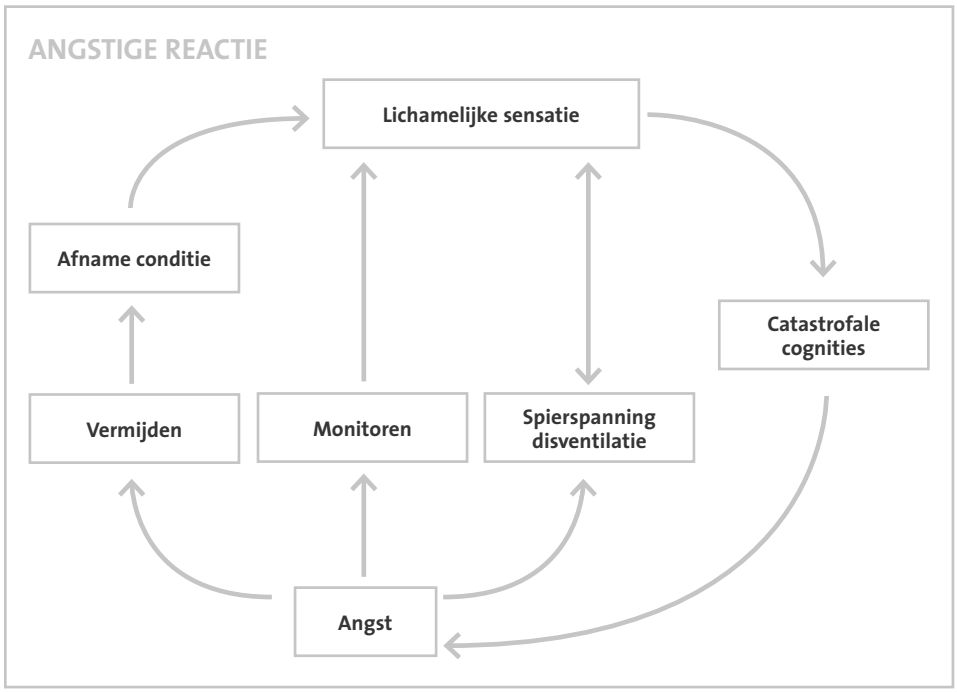
- lichamelijke klacht
- betekenis van de klacht (ideeën over de oorzaak),
- cognitieve en emotionele gevolgen (angst, woede, verdriet, maar ook beperkingen in aandacht, concentratie en geheugen),
- gedragsmatige gevolgen (b.v. afname van fysieke activiteit en toename van mentale activiteit, verzetten tegen beperkingen, forceren en doorgaan ondanks de klachten, terugtrekken uit sociale situaties, lichaamssignalen monitoren of juist negeren, gebruik van hulpmiddelen, gebruik van teveel of te weinig medicatie),
- lichamelijke gevolgen (autonome arousal, spierspanning, disventilatie, conditieverlies, verstoord dag-nacht ritme, slaapproblemen en vermoeidheid, toe- of afname van eetlust en gewicht, verminderde libido, seksualiteit),
- sociale gevolgen (b.v. verlies van werk en andere sociale functies, verlies van inkomen, verlies van sociale contacten, relatie- en gezinsconflicten, wijze waarop vrienden en familie reageren).

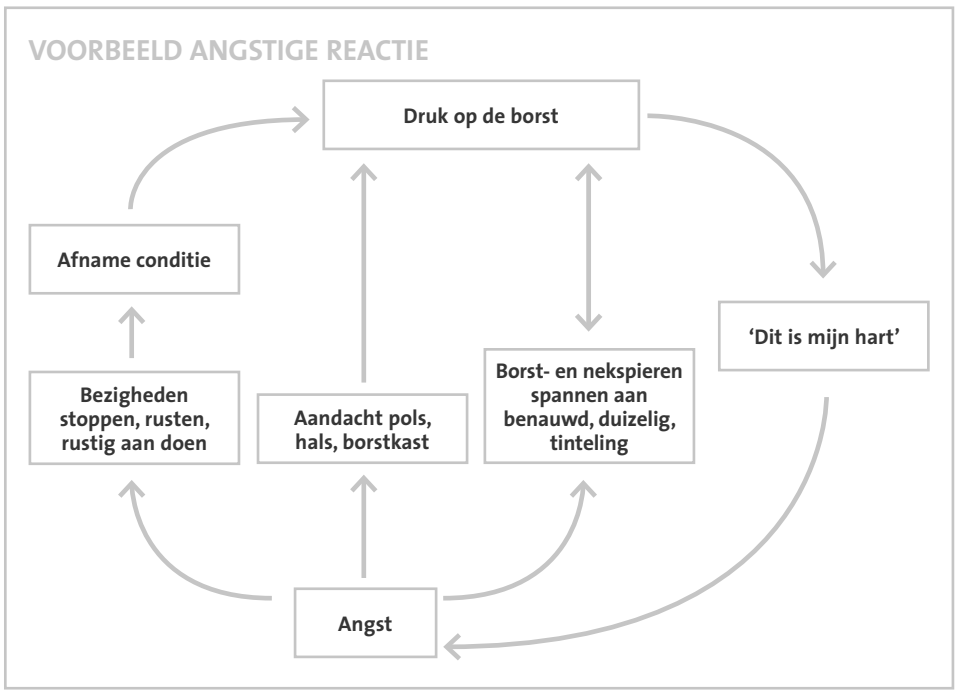
**Het algemene schema voor vicieuze cirkels ziet er als volgt uit:**

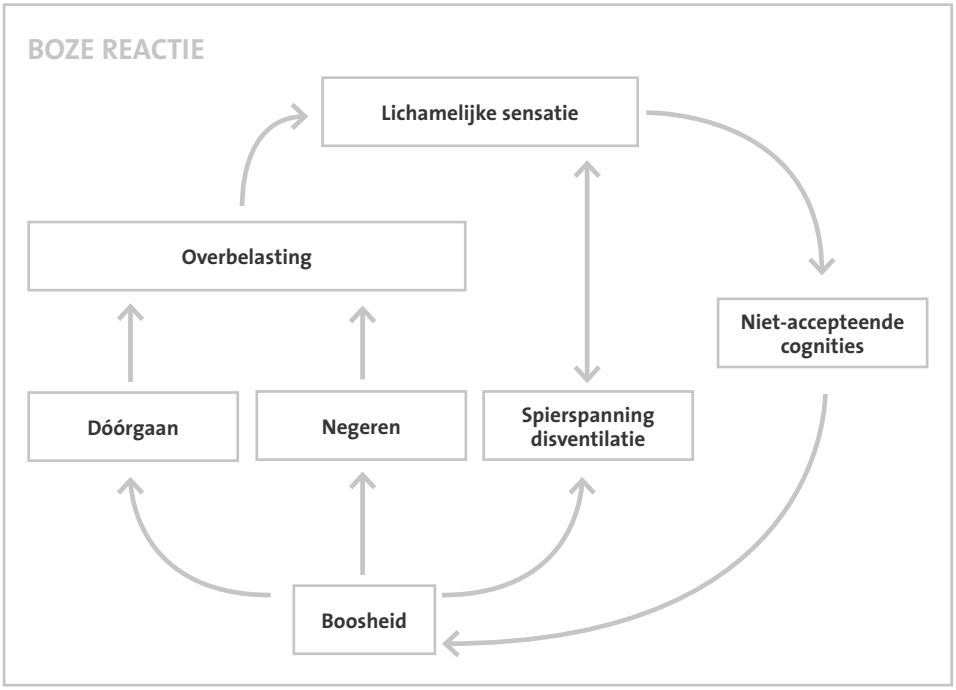


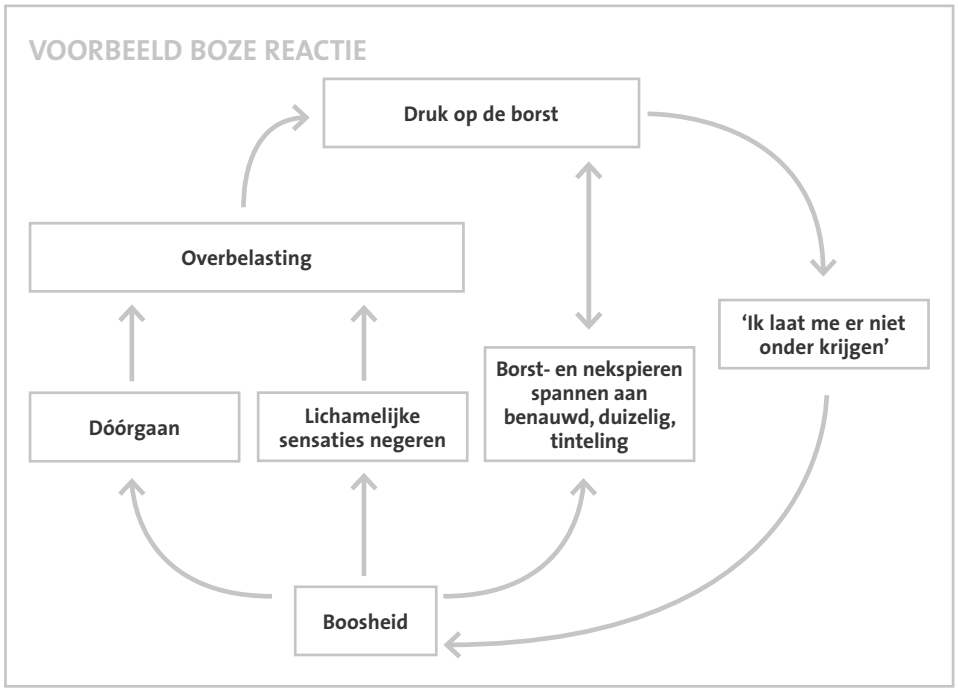

Bij het signaleren van een lichamelijke sensatie wordt daar een betekenis aan gegeven, de sensatie roept bepaalde gedachten op. Die gedachten roepen op hun beurt gevoelsreacties op; de gedachten en emoties leiden, via fysiologische mechanismen en gedragsveranderingen, tot een versterking van de lichamelijke sensaties.

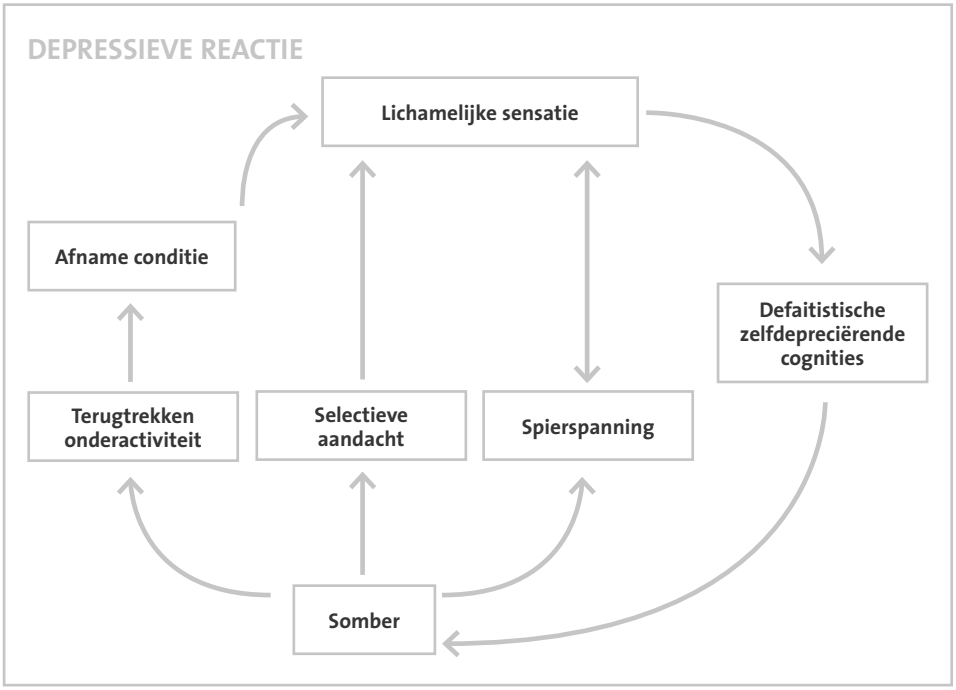
Veelvoorkomende reacties van patiënten op lichamelijke klachten zijn angst, boosheid en depressie. Deze verschillende reacties geven ook verschillende vicieuze cirkels, die hieronder staan afgebeeld.

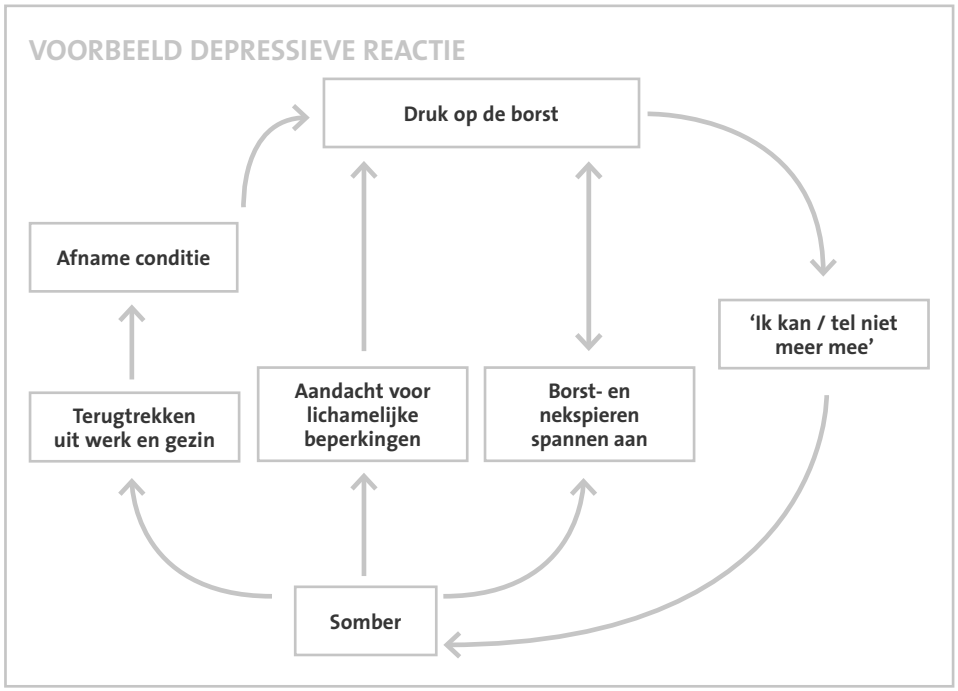



BOZE REACTIE
Lichamelijke sensatie
Overbelasting
Niet-accepteende cognities
Dóórgaan
Negeren
Spierspanning disventilatie
Boosheid


VOORBEELD BOZE REACTIE
Druk op de borst
Overbelasting
'Ik laat me er niet onder krijgen'
Borst- en nekspieren spannen aan benauwd, duizelig, tinteling
Dóórgaan
Lichamelijke sensaties negeren
Boosheid


DEPRESSIEVE REACTIE
Lichamelijke sensatie
Afname conditie
Defaitistische zelfdepreciërende cognities
Terugtrekken onderactiviteit
Selectieve aandacht
Spierspanning
Somber


VOORBEELD DEPRESSIEVE REACTIE
Druk op de borst
Afname conditie
'Ik kan / tel niet meer mee'
Terugtrekken uit werk en gezin
Aandacht voor lichamelijke beperkingen
Borst- en nekspieren spannen aan
Somber

# 6. Vervolgbeleid

In deze fase komt de andere 'poot' van de cognitief gedragstherapeutische technieken, namelijk het gedrag en het omgaan met de klachten, aan bod.
Voordat er plannen en concrete afspraken gemaakt worden, dienen arts en patiënt het eens te zijn over het behandeldoel. Uiteraard moet dat een realistisch en gewenst doel zijn. In de meeste gevallen komt het erop neer dat niet het verdwijnen van de klachten het behandeldoel is, maar het verminderen van de hinder die de patiënt ervan ondervindt en het inperken van de gevolgen ervan. Als uit de exploratie blijkt dat de patiënt in meer of mindere mate vermijdingsgedrag vertoont, is het zaak concrete plannen en afspraken te maken over hoe met dit vermijdingsgedrag om te gaan. Ditzelfde geldt voor het omgaan met ongewenste reacties uit de omgeving en het omgaan met de klachten in de werksituatie et cetera. Mensen veranderen hun gedrag niet gemakkelijk. Ze moeten niet alleen overtuigd zijn van de noodzaak van gedragsverandering, ze moeten ook het idee hebben dat gedragsverandering mogelijk is. Gedragsverandering is bij uitstek een terrein waarop de self-fulfilling prophecy's hun werk doen. Als je ervan overtuigd bent dat het stoppen met roken niet zal lukken, dan gaat het ook niet lukken.
Eén van de valkuilen bij gedragsveranderingen is, dat mensen vaak een bepaalde, ideale eindtoestand voor ogen hebben waarvan ze denken dat die voor hen onbereikbaar is. De conclusie die ze daaraan verbinden is, dat ze dus niet kunnen veranderen en dat elke poging gedoemd is te mislukken. In zo'n geval kan het helpen om de eindtoestand niet als eerste doel te zien, maar een aantal tussenstappen in te lassen die wel haalbaar zijn. Kleine stapjes maken is een belangrijke voorwaarde voor het laten slagen van gedragsverandering.
Een andere voorwaarde is, dat afspraken over gedragsverandering zo concreet mogelijk moeten zijn.
De doelstelling: 'Met ingang van volgende week gaat u elke dag 10 minuten (met de hond) wandelen' is concreet en maakt een redelijke kans. De afspraak: 'probeert u de komende tijd eens wat vaker met de hond te wandelen', is veel vager en laat de patiënt allerlei ontsnappingsmogelijkheden.
Veelal zal het de huisarts zijn die na de terugrapportage van de specialist de uitwerking van het vervolgbeleid en advies met de patiënt zal opnemen.

## Richtlijnen voor adviezen over gedragsverandering

Bij het optimaliseren van voorlichting die moet leiden tot gedragsverandering wordt gebruik gemaakt van een model dat zes stappen onderscheidt: openstaan, begrijpen, kunnen, willen, doen en blijven doen.

**Openstaan.** Een patiënt die nog steeds erg ongerust is over zijn klachten staat niet open voor allerlei adviezen. Dat geldt ook voor een patiënt die de verwachting koestert dat zijn klachten wel over zullen gaan door externe middelen (medicijnen, verwijzing naar specialist of fysiotherapeut) in te zetten. Een patiënt die ervan overtuigd is dat zijn klachten erger zullen worden als hij bepaalde activiteiten op zich neemt, zal die activiteiten niet hervatten. Als tijdens de informatiefase blijkt dat het niet lukt om iemand gerust te stellen of onjuiste cognities of verwachtingen bij te stellen, dan heeft het geen zin om met de laatste fase verder te gaan. Dan is het tijd om één van de technieken die in hoofdstuk 5 beschreven staan toe te passen.
**Begrijpen.** Een patiënt die de informatie niet begrijpt of niet snapt wat hij zou moeten doen, zal ook niet verder komen. In dat geval zal meer tijd genomen moeten worden om de benodigde informatie in begrijpelijke vorm met de patiënt te bespreken.

**Kunnen.** Hoe goed een advies of informatie ook is, de patiënt moet ook in staat zijn of zich in staat voelen om uit te voeren wat van hem gevraagd wordt. Het gaat hier niet alleen om het fysieke vermogen van de patiënt, maar ook om andere praktische belemmeringen voor het ten uitvoer brengen van de activiteit. Welke barrières ziet hij in dat geval?
**Willen.** Als de patiënt wel begrijpt wat ervan hem verwacht wordt en geen grote barrières ziet, maar niet wil meewerken, dan zal er ook niet veel gebeuren. In dat geval moet duidelijk worden waarom de patiënt niet wil: ziet hij geen heil in het advies, acht hij zichzelf niet in staat tot het uitvoeren van het advies?
**Doen.** Alleen een patiënt die openstaat voor een advies, die het advies begrijpt en het ook kan en wil opvolgen, zal het vervolgens waarschijnlijk gaan doen.

Sluit het consult af met een korte evaluatie en geef eventueel schriftelijke informatie mee.
Een korte evaluatie houdt in:

- De belangrijkste gegevens uit de exploratieve fase nog even noemen, bijvoorbeeld: 'U zei aan het begin dat u zich afvroeg of er een hernia was, bent op dat punt nu gerustgesteld? Geef ook hier de cognities of emoties in dezelfde termen weer als de patiënt gebruikt heeft.
- De besproken informatie nog een keer kort doorlopen, bijvoorbeeld: 'We hebben besproken dat uw rugklachten wel hinderlijk, maar niet ernstig zijn en dat er geen reden is om niet te gaan sporten. Integendeel, waarschijnlijk zal sporten een positieve invloed hebben, zeker op de wat langere duur, klopt dat of heb ik nog iets vergeten?' Introduceer daarbij geen nieuwe termen, maar gebruik dezelfde woorden als in het voorafgaande deel van het consult.
- De gemaakte afspraken nog even kort noemen, bijvoorbeeld: 'U gaat met ingang van volgende week weer één keer per week hardlopen en bouwt dit langzaam op.'
- Als er schriftelijke informatie voorhanden is, streep dan daarin de voor de patiënt relevante passages aan.

In de terugrapportage naar de huisarts vermeldt u zowel de hulpvragen van de verwijzer en patiënt als de antwoorden die u na onderzoek hier op hun vragen hebt kunnen geven alsmede een duidelijke uitleg van wat u hebt gedaan en wat uw bevindingen zijn.

# Eindnoten


[1] Van der Feltz-Cornelis CM, Van der Horst HE. Handboek Somatisatie. Lichamelijk onverklaarbare klachten in de eerste en tweede lijn. Utrecht: De Tijdstroom, 2003.

[2] Escobar JI. Transcultural aspects of dissociative and somatoform disorders. Psychiatric clinics of North America 1995;18:555-69.

[3] Gureje O, Simon GE, Ustun TB, Goldberg DP. Somatization in cross-cultural perspective: a World Health Organization study in primary care. Am J Psychiatry 1997;154:989-95.

[4] Huygen FJA, Van den Hoogen HJM, Van de Logt AT, Smits AJA. Nerveus-functionele klachten in de huisartsenpraktijk. I Een epidemiologisch onderzoek. Ned Tijdschr Geneeskd 1984;128:1321-7.

[5] Kroenke K, Spitzer RL, Williams JBW, Linzer M, Hahn SR, DeGruy FV et al. Physical symptoms in primary care. Predictors of psychiatric disorders and functional impairment. Arch Fam Med 1994;3:774-9.

[6] Katon WJ, Walker EA. Medically unexplained symptoms in primary care. J Clin Psychiatry 1998:59 Suppl 20:15-21.

[7] Van Hemert A, Hengeveld M, Bolk J, Rooijmans H, Vanderbroucke J. Psychological disorders in relation to medical illness among patients of a general medical outpatient clinic. Psychol Med 1993;23:167-73.

[8] Hamilton J, Campos R, Creed F. Anxiety, depression and management of medically unexplained symptoms in medical clinics. J R Coll Physicians 1996;30:18-21.

[9] Grol RPTM (red.) Huisarts en somatische fixatie. Utrecht/Antwerpen: Bohn Scheltema & Holkema, 1983.

[10] Cox MF. De vroegtijdige herkenning van somatisatie in de huisartsenpraktijk [dissertatie]. Leiden: 1992.

[11] Lipowski ZJ. Somatization: the concept and its clinical application. Am J Psychiatry 1988;145:1358-68.

[12] Barskey AJ, Borus JF. Functional somatic syndromes. Ann Intern Med 1999; 130:910-21.

[13] Goldberg DP, Bridges K. Somatic presentation of psychiatric illness in primary care setting. J Psychosom Res 1988; 32: 137-44.

[14] Diagnostic and statistical manual of mental disorders [Fourth edition, text revision], Washington DC, 2000

[15] Portegijs PJM, Kraan HF, Knottnerus JA, Stoffers HEJH. Wat heet somatisatie? 1. Een zoektocht in de internationale literatuur naar een definitie voor huisartsgeneeskundig onderzoek. Huisarts Wet 1992;35:18-25,34.

[16] Rosen G, Kleinman A, Katon W. Somatization in family practice: a biopsychosocial approach. J Fam Pract 1982;14:493-502.

[17] Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? Lancet 1999;354:936-9.

[18] Sharpe MA, Carson A. 'Unexplained' somatic symptoms, functional syndromes, and somatization: do we need a paradigm shift? Annals Intern Med 2001;134:926-30.

[19] Robbins JM, Kirmayer LJ. Cognitive and social factors in somatization. In: Kirmayer LJ, Robbins JM. Current concepts of somatization: Research and clinical perspectives. Washington DC: American psychiatric Press, 1991.

[20] Engel GL. The biopsychosocial model and the education of health professionals. Ann NY Acad Sci 1978;310;169-181.

[21] Lazarus RS, Folkman S. Stress appraisal and coping. New York: Springer 1994.

[22] Van Spaendonck KPM, Bleijenberg G. SCEGS communicatietraining. In: Praktisch Klinisch Onderwijs PKO-2, 1999; 90-5.

[23] Van Spaendonck KPM, Van de Lisdonk EH. Het biopsychosociale ziektemodel: een onderwijskundige operationalisatie. In: Pols J, Ten Cate ThJ, Polleman MC, Smal JA, redactie. Gezond onderwijs 4. Houten: Bohn, Stafleu, Van Loghum, 1995.

[24] Van der Horst HE. Irritable bowel syndrome in general practice. How effective is patient education and counselling? [Dissertatie] Amsterdam: Vrije Universiteit Amsterdam, 1997.

[25] Blankenstein AH. Somatising patients in general practice: reattribution, a promising approach. [Dissertatie] Amsterdam: Vrije Universiteit Amsterdam, 2001.

[26] Emmelkamp PMG, Bouman TK, Scholing HA. Angst, fobieën en dwang. Diagnostiek en behandeling. Houten: Bohn, Stafleu en van Loghum, 1995.

[27] Reinecke MA, Ryan NE, DuBois DL. Cognitive-behavioral Therapy of depression and depressive symptoms during adolescence: a review and meta-analysis. J Am Acad Child Adolesc Psychiatry 1998;37:26-34.

[28] Neomagus GJH, Terluin B, Aulbers LPJ, et al. NHG-Standaard Angststoornissen. Huisarts Wet 1997;40(4):167-75.

[29] Segal ZV, Whitney DK, Lam RW. Clinical guidelines for the treatment of depressive disorders: III Psychotherapy. Can J Psychiatry 2001; suppl: 29-37.

[30] Marwijk HWJ, Grundmeijer HGLM, Breuren NN. et al. NHG-Standaard Depressie. Huisarts Wet 1994;37(11):482-90.

[31] Morley S, Eccleston C, Williams A. Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. Pain 1999;(80):1-13.

[32] Barskey AJ, Borus JF. Functional somatic syndromes. Ann Intern Med 1999; 130:910-21.

[33] Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? Lancet 1999;354:936-9.

[34] Van Tulder MW. Chronische lage rugpijn: oefentherapie, multidisciplinaire programma's etc. Ned Tijdschr Geneesk 2000;144:1489-94.

[35] Van Tulder MW, Ostelo RWJG, Vlaeyen JWS, et al. Behavioral treatment for low chronic back pain. A systematic review within the framework of the Cochrane Back review Group. Spine 2001;26:270-81.

[36] Hopman-Rock M, Westhof MH. The effects of a health educational and exercise program for older adults with osteoarthritis of the hip or knee. J Rheumatol 2000; 27: in press.

[37] Van Baar ME. Effectiveness of exercise in osteoarthritis of hip and knee [Diss.]. Utrecht: Universiteit Utrecht, 1998

[38] Waddell G, Feder G, Lewis M. Systematic reviews of bed rest and advice to stay active for acute low back pain. Br J Gen Pract 1997;47:647-52.

[39] Bijl D, Dirven-Meyer PC, Opstelten W, et al. NHG-Standaard Niet-traumatische knieproblemen bij volwassenen. Huisarts Wet 1998;41:344-50.

[40] Van der Horst HE, Meijer JS, Muris JWM. NHG-Standaard Prikkelbaredarmsyndroom. Huisarts Wet 2001;44(2):58-65

[41] Faas A, Chavannes AW, Koes BW, et al. NHG-Standaard Lage rugpijn. Huisarts wet 1996;39(1):18-31.

[42] Assendelft WJJ, Rikken SAJJ, Mel M, et al. NHG-Standaard Epicondylitis. Huisarts Wet 1997; 40(1): 21-6

[43] Van Spaendonck KPM, Van de Lisdonk EH. Het biopsychosociale ziektemodel: een onderwijskundige operationalisatie. In: Pols J, Ten Cate ThJ, Polleman MC, Smal JA, redactie. Gezond onderwijs 4. Houten: Bohn, Stafleu, Van Loghum, 1995.

[44] Van Spaendonck KPM, Bleijenberg G. SCEGS communicatietraining. In: Praktisch Klinisch Onderwijs PKO-2, 1999; 90-5.

[45] Bass C, Benjamin S. The management of Chronic Somatisation. Br J Psychiatry 1993;162:472-80.

# 7. Aanvullende artikelen

## Bijlage 1


1. Nimnuan C, Hotopf M, Wessely S. Medically unexplained symptoms: an epidemiological study in seven specialties. J Psychosom Res 2001; 51:361-7.

2. Weiland, A., et al., Encounters between medical specialists and patients with medically unexplained physical symptoms; influences of communication on patient outcomes and use of health care. A literature overview. Perspect Med Educ, 2012. 1:192–206.

3. Rolfe A, et al., Reassurance after diagnostic testing with a low pretest probability of serious disease: Systematic review and meta-analysis. JAMA Internal Medicine, 2013. 173(6): p. 407-416.

4. Hall-Patch L, Brown R, House A, Howlett S, Kemp S, Lawton G, et al. Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures. Epilepsia 2010; 51(1): 70-8.

5. Petrie KJ, Müller JT et al. Effect of providing information about normal test results on patients' reassurance: randomized controlled trial. BMJ 2007;334;352.

6. Stone J, Carson A, Sharpe M. Functional symptoms in neurology: management. J Neurol Neurosurg Psychiatry 2005;76 (suppl I): i13-i21.

7. Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry 2005; 76 (suppl I): i2-i12.

1. Nimnuan C, Hotopf M, Wessely S. Medically unexplained symptoms: an epidemiological study in seven specialties. J Psychosom Res 2001; 51:361-7.

ELSEVIER

Journal of Psychosomatic Research 51 (2001) 361–367

Journal of Psychosomatic Research

# Medically unexplained symptoms
# An epidemiological study in seven specialities

Chaichana Nimnuan, Matthew Hotopf, Simon Wessely*

*Academic Department of Psychological Medicine, Guy's King's and St. Thomas' School of Medicine and Institute of Psychiatry, 103 Denmark Hill, De Crespigny Park, London SE5 8AF, UK*

Received 11 October 2000; accepted 5 March 2001

**Abstract**

**Objectives:** This study aimed to estimate the prevalence and risk factors for medically unexplained symptoms across a variety of specialities. **Methods:** A cross-sectional survey was conducted at two general hospitals in southeast London between 1995 and 1997. Eight hundred and ninety consecutive new patients from seven outpatient clinics were included. Demographic and clinical characteristic variables were assessed. **Results:** Five hundred eighty-two (65%) of the subjects surveyed returned completed questionnaires. A final diagnosis was available in 550 (62%). Two hundred twenty-eight (52%) fulfilled criteria for medically unexplained symptoms. The highest prevalence was in the gynaecology clinic (66%). After adjustment for confounders, medically unexplained symptoms were associated with being female, younger, and currently employed. Psychiatric morbidity per se was not associated with the presence of medically unexplained symptoms, but was more likely in those complaining of multiple symptoms. Those with medically unexplained symptoms were less disabled, but more likely to use alternative treatment in comparison with those whose symptoms were medically explained. Patients with medically unexplained symptoms were more likely to attribute their illness to physical causes as opposed to lifestyle factors. **Conclusions:** Medically unexplained symptoms are common across general/internal medicine and represent the most common diagnosis in some specialities. Medical behavior, training, and management need to take this into account.


*Keywords:* Medically unexplained symptoms; Somatization; Somatoform disorders; Prevalence; Illness behavior

## Introduction

Medically unexplained symptoms are a common problem across general medicine. They can be presentations of recognised psychiatric disorders such as anxiety or depression; a part of operationally defined unexplained syndromes such as chronic fatigue syndrome, irritable bowel syndrome, or fibromyalgia; or simply exist as symptoms in the absence of a defined organic diagnosis. Medically unexplained symptoms are an important problem in general medicine not only because of their prevalence but also on account of the high associated consumption of health service resources.

Medically unexplained symptoms are reported to be more common among women, younger age groups, and those from lower socioeconomic backgrounds [1–7], and are associated with the presence of psychiatric disorders [8,9]. Those without conventional medical explanation for their symptoms are about twice as likely to fulfill criteria for psychiatric disorders [10]. Another study of specialist care showed the number of lifetime somatic symptoms was significantly and positively related to the increase in the number of current and past episodes of anxiety and depression [11]. Kisely et al. [12] also found that the presence of somatic symptoms, whether medically explained or unexplained, was associated with psychiatric morbidity.

Many questions about medically unexplained symptoms remain unanswered. Most studies have taken place in one or only a few clinics; the number of variables under study have been limited; and researchers often tend to concentrate on single specific symptoms or syndromes as

* Corresponding author. Tel.: +020-7848-0778; fax: +020-7848-5129.
*E-mail address*: sphascw@iop.kck.ac.uk (S. Wessely).

0022-3999/01/$ – see front matter © 2001 Elsevier Science Inc. All rights reserved.
PII: S0022-3999(01)00223-9

opposed to looking at medically unexplained symptoms as a whole. Most research has focussed on variables such as demographic factors and psychiatric morbidity. Other variables such as illness cognitions and the social consequences of the illness have tended to be ignored.

We conducted a cross-sectional study of medically unexplained symptoms in the general hospital, which included the principal medical specialities and used the same assessment across all settings. In this paper, we report the prevalence and associations of medically unexplained symptoms in general hospital outpatients.

## Methods

### *Sample*

Consecutive new patients residing in southeast London and referred by their general practitioners to outpatient clinics at King's College and Dulwich Hospitals between 1995 and 1997 were recruited. The clinics were gastroenterology, gynaecology, neurology, rheumatology, chest, cardiology, and dentistry. Subjects were eligible for inclusion if they were aged between 16 and 65 years. Subjects who could not read or speak English; and those with psychotic illnesses or organic brain syndromes were excluded.

### *Sample size*

Sample size was estimated using Statcalc program in Epi Info for a descriptive population study. The confidence level required at the end of the study was set at 95%. The maximum allowable difference between the estimate and the true prevalence was set at 10%. The rate of medically unexplained symptoms from our pilot study at the gastroenterology clinic at King's College and Dulwich Hospitals was estimated at 58% (see below). With an allowable difference between the estimate and the true prevalence set at 10%, the value of sample size then was 94. An expected response rate was set at 70%. The required sample size was therefore 940 for all clinics.

### *Case definition*

For this study, medically unexplained symptoms were defined as any current principal somatic complaint reported by patients for which no definite medical diagnosis could be found by physical examination and appropriate investigation. To make this judgement, we used investigation results and physicians' opinions. The physician's opinion was determined by the final diagnosis stated in the clinical case notes. If the physicians gave a diagnosis of "functional," or continued to defer the diagnosis because of no detected abnormality, we considered these as indicating that the symptoms were medically unexplained. We have shown elsewhere that this method has acceptable interrater reliabilities with kappa values ranging from 0.76 to 0.88 [13].

### *Data collection*

Patients attending the above clinics were given a questionnaire with a return-stamped addressed envelope. Two postal reminders and one telephone were used to increase response rate. Case notes were reviewed to ascertain the final diagnosis approximately 3 months after the initial visit.

### *Measures*

Information on the following variables was collected in the questionnaire.

#### *Demographic data*

Age, gender, marital status, educational level (recorded as number of years of full-time education), ethnicity, work status (defined by paid and unpaid work, students, and housewives were designated in the working group), and occupation.

#### *Symptom review questionnaire (SRQ)*

This was developed for the current investigation. It consists of 11 main symptoms, which correspond to 13 recognised Functional Somatic Syndromes, with 25 additional symptoms, including somatic symptoms, sleep, and psychological complaints. A total of 27 individual somatic symptoms were inquired about. In this report, the number of somatic complaints regardless of their nature (medically explained or unexplained) was used as an explanatory variable.

#### *Illness cognition*

This three-page self-report questionnaire covered the patients' own ideas on the nature of their illness, how that information was acquired, and their illness attribution. Attribution was classified into three main categories: psychological factors (stress, depression, personality, and overwork); pattern of behaviors or habits (smoking, and drinking); and physical factors (accident or injury, infectious causes, toxins, and allergy). We applied a factor analytic technique to support the classification. The result showed three factors that accounted for 52% of variance. Although the factors extracted seemed to correspond with the categories proposed, some responses were removed and changed. "Accident or injury" was dropped because of low correlation value with all others and "overwork" was moved to the psychological dimension which proved a better fit. These three factors were then used as three explanatory variables in the analysis. Additional questions were asked about use of alternative medicines and receipt of state benefits.

#### *Psychiatric morbidity*

We used the Hospital Anxiety and Depression Scale (HADS) to detect anxiety and depression. It is a 14-item

self-assessment scale designed to be used in the medical settings [14]. The subscale of anxiety and depression were used rather than combining both into a total score. Responses to each question were scored from 0 to 3, giving a maximum score of 21 for each subscale. We applied a cut-off of 10/11 for caseness as originally proposed [14].

*Functional impairment*

Functional impairment was assessed by the Brief Disability Questionnaire (BDQ) [15] in the recoded version proposed by Ormel et al. [16], which consisted of four level categories; none, mild, moderate, and severe disability.

Other measures to be reported elsewhere covered patient satisfaction, medical perceptions, and the use of investigations.

*Analysis*

The prevalence of subjects with medically unexplained symptoms were calculated according to clinic and gender. Odds ratio and 95% confidence intervals were used as the main measurement of the association between dependent and independent variables. Univariate analyses were used to examine the association of the outcome variables with each variable of interest in turn. The chi-square test was used. Logistic regression modeling, adjusting for the effect of many variables simultaneously was used for multivariate analysis. We grouped dependent variables into three main categories: (1) demographic and clinic variables; (2) number of symptoms and psychiatric morbidity; and (3) illness cognitions. We then performed logistic regression modeling in steps, starting with demographic and clinical variables and then adding the two other categories in sequence. All modeling was performed by STATA software package (Stata, College Station, TX). Chi-square values were based on likelihood ratio statistics.

## Results

*Baseline characteristics*

During the period of the study, 890 new patients attended the seven clinics. A total of 582 valid responses were obtained (65%). There was a significant association between clinics and response rate ($P$=.01). The dental clinic had the highest response rate (75%) while gastroenterology had the lowest (55%). Nonresponders did not differ from responders in terms of ethnicity. However, responders were more likely to be female (62% responders vs. 53% nonresponders, $P$=.02) and older (mean = 43.2, S.D. = 12.6 responders vs. mean = 39.5, S.D. = 11.8 nonresponders, $P$ < .01).

Table 1 shows the characteristics of samples by clinic. Of 582 respondents, 32 case notes were missing, leaving 550 subjects to be included in analysis. We found that the clinics differed in a number of demographic variables previously

Table 1
Baseline characteristics of sample by clinic ($N$ = 550)

| | Chest, $n$ = 59 | Cardio, $n$ = 92 | GI, $n$ = 52 | Rheum, $n$ = 91 | Neurol, $n$ = 103 | Dental, $n$ = 71 | Gynae, $n$ = 82 | $P$ value |
|---|---|---|---|---|---|---|---|---|
| *Age, %* | | | | | | | | |
| 16–25 | 5 | 5 | 12 | 4 | 13 | 11 | 10 | < .001 |
| 26–35 | 17 | 14 | 23 | 17 | 32 | 23 | 43 | (Kruskal–Wallis) |
| 36–45 | 22 | 20 | 15 | 26 | 20 | 11 | 24 | |
| 46–55 | 22 | 27 | 29 | 35 | 24 | 31 | 18 | |
| 56–65 | 34 | 34 | 21 | 18 | 11 | 24 | 5 | |
| *Sex* | | | | | | | | |
| Percent female | 54 | 53 | 62 | 68 | 63 | 78 | NA | .02 |
| *Marital status* | | | | | | | | |
| Percent not married | 37 | 47 | 58 | 52 | 54 | 36 | 53 | .07 |
| *Work status* | | | | | | | | |
| Percent without work | 42 | 33 | 35 | 39 | 28 | 24 | 19 | .04 |
| *Ethnicity* | | | | | | | | |
| Percent white | 73 | 65 | 80 | 68 | 78 | 75 | 52 | .004 |
| *Years of leaving full-time education* | | | | | | | | |
| Percent years > 16 | 56 | 51 | 57 | 37 | 46 | 50 | 67 | .01 |
| *Social class* | | | | | | | | |
| Percent professional | 47 | 43 | 51 | 28 | 40 | 49 | 44 | .65 |
| Percent skilled | 38 | 41 | 35 | 54 | 42 | 37 | 40 | |
| Percent partly skilled/unskilled | 16 | 16 | 14 | 18 | 18 | 14 | 16 | |

Table 2
Prevalence of medically unexplained symptoms by clinic and gender ($N=550$)

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | Subjects with MUS (total) | Percent cases of MUS (95% CI) | Subjects with MUS (total) | Percent cases of MUS (95% CI) | Subjects with MUS (total) | Percent cases of MUS (95% CI) |
| Dental | 8 (16) | 50 (25–75) | 18 (55) | 33 (21–45) | 26 (71) | 37 (25–48) |
| Chest | 7 (27) | 26 (9–43) | 17 (32) | 53 (35–70) | 24 (59) | 41 (28–53) |
| Rheumatology | 9 (29) | 31 (14–48) | 32 (62) | 52 (39–64) | 41 (91) | 45 (35–55) |
| Cardiology | 18 (43) | 42 (27–57) | 31 (49) | 63 (50–77) | 49 (92) | 53 (43–63) |
| Gastroenterology | 10 (20) | 50 (28–72) | 20 (32) | 63 (46–80) | 30 (52) | 58 (44–71) |
| Neurology | 21 (38) | 55 (39–71) | 43 (65) | 66 (55–78) | 64 (103) | 62 (52–72) |
| Gynecology | – | – | 54 (82) | 66 (56–76) | 54 (82) | 66 (56–76) |
| Total | 73 (173) | 42 (35–50) | 215 (377) | 57 (52–62) | 288 (550) | 52 (48–57) |

identified as associated with medically unexplained symptoms. Although there were no interclinic differences in marital status and social class, there were differences in gender, age, ethnicity, and work status. For example, gynecology patients were significantly younger than cardiac clinic attendees, and more likely to belong to ethnic minorities. For these reasons, clinics are considered as a potential confounder in the analyses.

*Prevalence and risk factors of medically unexplained symptoms (univariate analysis)*

Approximately half (52%) of new attenders to the above clinics had at least one medically unexplained symptom (Table 2). The gynecology clinic had the highest prevalence while the dental clinic had the lowest. The chi-square test results showed a significant association between referral clinics and the prevalence of medically unexplained symptoms (Table 3). Using the dental clinic as the reference (because this had the lowest prevalence of medically unexplained symptoms), cardiology and gastroenterology had approximately 100% increased risk; neurology and gynaecology had approximately 200% increased risk. In all clinics, the prevalence of medically unexplained symptoms was significantly higher in females. Patients with medically unexplained symptoms were more likely to be younger, to be with work, and to have higher educational attainment.

Table 4 shows the clinical characteristics of patients with medically unexplained symptoms. Although there was no overall association between unexplained symptoms and total somatic symptoms, patients in the top quartile for somatic complaints (18 or more) were marginally more likely to have medically unexplained symptoms (OR = 1.3, 95% CI = 0.9, 2.0). Contrary to expectation, psychological symptoms on the HADS were not associated with an increased risk of having medically unexplained symptoms. The mean HADS score for patients with unexplained symptoms was 13.4 as opposed to 12.9 for the group with explained symptoms ($P=.5$).

A physical attribution was associated with an increased risk of having medically unexplained symptoms, but not psychological attributions. Subjects who believed their illness to be the result of lifestyle factors were approx-

Table 3
Univariate associations between medically unexplained somatic symptoms and explanatory variables ($N=550$)

| Explanatory variables | No. of subjects[a] (% case with medically unexplained symptoms) | OR (95% CI), $P$ value |
|---|---|---|
| *Demographic variables* | | |
| Age | | |
| 16–25 | 47 (72) | 4.6 (2.2–9.7) |
| 26–35 | 134 (57) | 2.3 (1.4–3.9) |
| 36–45 | 112 (60) | 2.6 (1.5–4.5) |
| 46–55 | 147 (48) | 1.6 (1.0–2.7) |
| 56–65 | 110 (36) | reference, $P<.001$[b] |
| Gender[c] | | |
| Male | 173 (42) | |
| Female | 295 (55) | 1.7 (1.1–2.4), $P=.01$ |
| Marital status | | |
| Married | 281 (50) | |
| Nonmarried | 263 (55) | 1.2 (0.9–1.7), $P=.29$ |
| Ethnicity | | |
| Nonwhite | 166 (52) | |
| White | 379 (52) | 1.0 (0.7–1.4), $P=.97$ |
| Work status | | |
| With work | 371 (58) | |
| Without work | 167 (39) | 0.5 (0.3–0.7), $P<.001$ |
| Age of leaving full-time education | | |
| 16 or less | 249 (47) | |
| 17 or more | 258 (57) | 1.5 (1.1–2.2), $P=.02$ |
| Social class | | |
| Semiskilled/unskilled | 69 (45) | reference |
| Skilled | 178 (55) | 1.5 (0.9–2.6) |
| Professional | 180 (57) | 1.6 (0.9–2.9), $P=.11$[b] |
| Clinic | | |
| Dental | 71 (37) | reference |
| Chest | 59 (41) | 1.2 (0.6–2.4) |
| Rheumatology | 91 (45) | 1.4 (0.8–2.7) |
| Cardiology | 92 (53) | 2.0 (1.1–3.7) |
| Gastrology | 52 (58) | 2.4 (1.1–4.9) |
| Neurology | 103 (62) | 2.8 (1.5–5.3) |
| Gynecology | 82 (66) | 3.3 (1.7–6.5), $P=.001$ |

[a] Total number of subjects for each variable varies because of missing data.
[b] Test for trend.
[c] Exclude gynaecology.

imately 40% less likely to have medically unexplained symptoms compared to those who did not ($P$=.02). Having obtained information from health professionals decreased the risk of having medically unexplained symptoms.

Receiving alternative treatment was associated with a 30% increased odds of having medically unexplained symptoms ($P$=.13) while receiving benefits was associated with a 30% decrease ($P$=.06). Medically unexplained symptoms were most common in the group with no disability, compared to groups with increasing levels of disability ($P$=.003).

Table 4
Clinical characteristics of patients with medically unexplained symptoms

| Explanatory variables | No. of subjects[a] (% case with medically unexplained symptoms) | OR (95% CI), $P$ value |
|---|---|---|
| *Number of symptoms and psychiatric morbidity* | | |
| Total number of symptom complaints (quartiles) | | |
| 1–9 | 150 (51) | reference |
| 10–13 | 137 (50) | 0.9 (0.6–1.5) |
| 14–17 | 119 (50) | 0.9 (0.6–1.5) |
| 18–27 | 144 (58) | 1.3 (0.8– 2.1), $P$=.25[b] |
| Sleep problems | | |
| None | 155 (50) | reference |
| Mild | 137 (56) | 1.3 (0.8–2.0) |
| Moderate | 136 (51) | 1.0 (0.6–1.6) |
| Severe | 122 (53) | 1.1 (0.7–1.8), $P$=.93[b] |
| Anxiety (HAD-A) | | |
| Noncase (<11) | 388 (51) | |
| Case (≥11) | 144 (56) | 1.3 (0.9–1.8), $P$=.26 |
| Depression (HAD-D) | | |
| Noncase (<11) | 472 (53) | |
| Case (≥11) | 60 (47) | 0.8 (0.5–1.3), $P$=.34 |
| *Illness cognitions* | | |
| Lifestyle attributions | | |
| Unlikely | 384 (56) | |
| Likely | 128 (44) | 0.6 (0.4–0.9), $P$=.02 |
| Physical attributions | | |
| Unlikely | 375 (50) | |
| Likely | 135 (60) | 1.5 (1.0–2.3), $P$=.04 |
| Psychological attributions | | |
| Unlikely | 367 (51) | |
| Likely | 142 (56) | 1.2 (0.8–1.8), $P$=.30 |
| Source of information about illness before visiting the clinic | | |
| Others | 159 (57) | |
| Health professionals | 321 (48) | 0.7 (0.5–1.0), $P$=.08 |
| *Consequences of the illness* | | |
| Alternative treatment | | |
| None | 294 (49) | |
| Received | 215 (56) | 1.3 (0.9–1.9), $P$=.13 |
| Disability (BDQ score) | | |
| None | 193 (63) | reference |
| Mild | 69 (44) | 0.5 (0.3–0.8) |
| Moderate | 136 (46) | 0.5 (0.3–0.8) |
| Severe | 125 (48) | 0.6 (0.4–0.9), $P$=.004[b] |
| Benefits | | |
| None | 294 (55) | |
| Received | 208 (47) | 0.7 (0.5–1.0), $P$=.06 |

[a] Total number of subjects for each variable varies because of missing data.
[b] Test for trend.

Table 5
Adjusted odds ratio for the association between medically unexplained symptoms and explanatory variables

| Explanatory variables | Adjusted OR (95% CI) |
|---|---|
| *Age*[a] | |
| 16–25 | 4.4 (1.5–13.3) |
| 26–35 | 1.5 (0.8–3.0) |
| 36–45 | 1.9 (0.9–3.9) |
| 46–55 | 1.2 (0.6–2.3) |
| 56–65 | reference, $P$=.04 |
| *Sex*[a] | |
| Male | |
| Female | 2.0 (1.3–3.2), $P$=.003 |
| *Work status*[a] | |
| With work | |
| Without work | 0.6 (0.4–1.0), $P$=.06 |
| *Clinic*[a] | |
| Dental | reference |
| Chest | 1.8 (0.8–4.1) |
| Rheumatology | 1.9 (0.9–4.0) |
| Gynaecology | 2.5 (1.1–5.5) |
| Cardiology | 2.6 (1.2–5.6) |
| Gastroenterology | 3.4 (1.4–7.8) |
| Neurology | 3.4 (1.6–7.2), $P$=.03 |
| *Total number of symptom complaints*[b] | |
| ≤17 | |
| >17 | 1.4 (0.9–2.2), $P$=.10 |
| *Illness cognitions* | |
| Lifestyle attributions[c] | |
| Unlikely | |
| Likely | 0.6 (0.4–1.0), $P$=.05 |
| Physical attributions[c] | |
| Unlikely | |
| Likely | 1.9 (1.2–3.0), $P$=.009 |

[a] Model 1 variables include age, sex, work status, educational level, social class, and clinic ($n$=409).
[b] Model 2 variables include age, sex, work status, clinic, and total number of symptoms ($n$=521).
[c] Model 3 variables include age, sex, work status, clinic, total number of symptoms, lifestyle attributions, and physical attributions ($n$=449).

## *Logistic regression model*

Table 5 shows the association between the outcome and relevant variables after logistic regression modeling. The first model included demographic factors and the clinic in which patients were seen. Being female, younger, and presenting to certain clinics were still independently associated with medically unexplained symptoms.

Because of missing data for some variables, further modeling only included age, gender, work status, and clinic as demographic variables. The second model assessed the association between total somatic symptoms and having

unexplained symptoms, and found that there was still a weak association. The third model controlled for model 2 variables and added illness cognitions. This found an association between unexplained symptoms and making physical attributions, and a tendency for those with explained symptoms to report more lifestyle attributions.

## Comment

Medically unexplained symptoms were defined as current somatic complaints reported by patients, for which conventional biomedical explanation could not be found on routine examination or investigations, rated 3 months after the initial appointment. Previous studies have rated symptoms as medically explained/unexplained on either patient self-report or the clinician's impression on the initial visit [1,17,18]. In this study, medically unexplained symptoms were judged as present on the final decision made 3 months later, after review of all the available information. We consider this to be an improvement on previous work, which may also explain why we found a different pattern of associations to previous studies. Furthermore, most previous studies have been limited to one or two clinics [4,19,20]. In this study, we have sampled six different medical specialities plus dentistry.

Our results show that between one-third and two-thirds of patients attending general medical clinics do not receive a biomedical explanation for their distress. Previous studies have suggested this in individual clinics — for example, only 16% of one series of new outpatients attendees to a US internal medicine clinic was a definite biomedical cause identified for symptoms [21]. Van Hemert et al. [10] conducted a survey in a Dutch medical outpatient clinic showed that 52% of new referrals remained medically unexplained. We have now extended this using a uniform methodology across a large number of medical specialities. We conclude that it is now time to acknowledge that the management of medically unexplained symptoms is one of the important tasks facing the specialist in internal medicine — indeed, in some clinics, it constitutes the majority of the work.

As expected, we confirmed that medically unexplained symptoms are more common in females and younger age groups [6,7,22]. However, perhaps the most relevant clinical finding is that the chance of finding a biomedical cause does not increase with the number of somatic complaints as many think, but rather the reverse. Our results also support the idea that patients with medically unexplained symptoms tend to attribute their illness to physical causes [23]. The findings also show that patients who attribute their illnesses to lifestyle factors are less likely to have medically unexplained somatic symptoms.

More surprisingly, we failed to confirm previous reports that those with medically unexplained symptoms have higher level of disability, psychiatric morbidity, and state benefits, and are less likely to be in work [2,10,17,18,24,25]. We found the reverse for most of these variables. We also found that the psychiatric morbidity was similar (approximately 50%) in both medically explained and medically unexplained categories. We did not use a psychiatric interview, and it is possible that an interview would have revealed differences in rates of current or lifetime diagnoses, however, the HADS is a sensitive questionnaire that has been widely used in these populations. A possible explanation for the differences in the results may be due to the population studied. Patients with medically unexplained symptoms may request referral for vague or unexplained symptoms, while patients with medically explained symptoms only seek referral or continuing evaluation and treatment for symptoms due to serious medical disorders. Because our study is based in a tertiary care hospital, patients with medical diagnoses (compared to those with unexplained symptoms) may have more severe illnesses than those encountered in primary care settings. Approximately 63% of the sample reported at least some disability and this rate is higher than primary care samples — for example, the World Health Organisations International Study on Psychological Problems in General Health Care found rates of disability in the Manchester center of 45% [26]. Levels of psychological distress were also high in our sample. As commented by McDaniel et al. [27], the prevalence of psychiatric morbidity is increased with the increase severity of medical illness. This explanation, however, needs to be investigated further.

This study has limitations. Firstly, the required sample size was not obtained due to the low response rate. Secondly, although we considered using the diagnosis stated in case notes 3 months after the initial visit as an improvement in defining medically unexplained symptoms, the final diagnosis is still subject to change afterwards. This may cause overdiagnoses of medically unexplained symptoms which later may be reversed. Finally, patients may endorse a number of symptoms provided in the questionnaires where some of them are not clinically relevant. This may underestimate the strength of association between the number of symptoms complaints and medically unexplained symptoms due to (undifferentiated) misclassification bias. The results therefore need to be interpreted in the light of these limitations.

## Acknowledgments

Dr. Nimnuan is supported by Thai government as a part of a PhD program. We are particularly grateful to the following consultants who allow access to their clinics: Dr. W. Gardner, Dr. I. Forgacs, Dr. M. Blott, Dr. D. Scott, Prof. N. Johnson, Dr. C. Pankhurst, Dr. T. Britton, Dr. D. Jewitt, Prof. J. Moxham. We thank all patients and medical staff who took part.

## References


[1] Kroenke K, Price RK. Symptoms in the community: prevalence, classification, and psychiatric comorbidity. Arch Intern Med 1993; 153:2474–80.

[2] Mumford DB, Devereux TA, Maddy PJ, Johnston JV. Factors leading to the reporting of "functional" somatic symptoms by general practice attenders. Br J Gen Pract 1991;41:454–8.

[3] Melville D. Descriptive clinical research and medically unexplained physical symptoms. J Psychosom Res 1987;31:359–65.

[4] Holmes KM, Salter RH, Cole TP, Girdwood RG. A profile of district hospital gastroenterology. J R Coll Physicians London 1987;21:111–4.

[5] Canino IA, Rubio-Stipec M, Canino G, Escobar JI. Functional somatic symptoms: a cross ethnic comparison. Am J Orthopsychiatry 1992;62:605–12.

[6] Robbins J, Kirmayer L. Cognitive and social factors in somatization. In: Kirmayer L, Robbins J, editors. Current concepts in somatization: research and clinical perspectives. Washington, DC: American Psychiatric Press, 1991. pp. 88–101.

[7] Smith GR, Monson RA, Ray DC. Patients with multiple unexplained symptoms: their characteristics, functional health and health care utilization. Arch Intern Med 1986;146:69–72.

[8] Katon W, Lin E, von Korff M, Russo J, Lipscomb P, Bush T. Somatization: a spectrum of severity. Am J Psychiatry 1991;148:34–40.

[9] Hotopf M, Mayou R, Wadsworth MEJ, Wessely S. Temporal relationships between physical symptoms and psychiatric disorder. Results from a national birth cohort. Br J Psychiatry 1998;173:255–61.

[10] Van Hemert AM, Hengeveld MW, Bolk JH, Rooijmans HGM, Vandenbroucke JP. Psychiatric disorders in relation to medical illness among patients of a general medical out-patient clinic. Psychol Med 1993;23:167–73.

[11] Russo J, Katon W, Sullivan M, Clark M, Buchwald D. Severity of somatization and its relationship to psychiatric disorder and personality. Psychosomatics 1994;35:546–56.

[12] Kisely S, Goldberg D, Simon G. A comparison between somatic symptoms with and without clear organic cause: results of an international study. Psychol Med 1997;27:1011–9.

[13] Reid S, Crayford T, Nimnuan C, Richards S, Hotopf M. Recognition of medically unexplained symptoms — do doctors agree? J Psychosom Res 2000;47:483–5.

[14] Zigmond AS, Snaith RP. The Hospital Anxiety and Depression Scale. Acta Psychiatr Scand 1983;67:361–70.

[15] von Korff M, Ustun B, Ormel J, Kaplan I, Simon GE. Self-report disability in an international primary care study of psychological illness. J Clin Epidemiol 1996;49:297–303.

[16] Ormel J, von Korff M, Ustun B, Pini S, Korten A, Oldehinkel T. Common mental disorders and disability across cultures. JAMA, J Am Med Assoc 1994;272:1741–8.

[17] Escobar JI, Golding JM, Hough RL, Karno M, Burman MA, Wells KB. Somatisation in the community: relationship to disability and use of services. Am J Public Health 1987;77:837–40.

[18] Kroenke K, Spitzer RL, de Gruy FV, Hahn SR, Linzer M, Williams JBW, Brody D, Davies N. Multisomatoform disorder. Arch Gen Psychiatry 1997;54:352–8.

[19] Hamilton J, Campos R, Creed F. Anxiety, depression and management of medically unexplained symptoms in medical clinics. J R Coll Physicians London 1996;30:18–20.

[20] Harvey RF, Salih SY, Read AE. Organic and functional disorders. Lancet 1983;1:632–4.

[21] Kroenke K, Mangelsdorff AD. Common symptoms in ambulatory care: incidence, evaluation, therapy, and outcome. Am J Med 1989; 86:262–6.

[22] Stewart D. The changing face of somatisation. Psychosomatics 1990;31:153–8.

[23] Wessely S, Powell R. Fatigue syndromes: a comparison of chronic "postviral" fatigue with neuromuscular and affective disorders. J Neurol Neurosurg Psychiatry 1989;52:940–8.

[24] Walker EA, Keegan D, Gardner G, Sullivan M, Katon WJ, Bernstein D. Psychosocial factors in fibromyalgia compared with rheumatoid arthritis: I. Psychiatric diagnoses and functional disability. Psychosom Med 1997;59:565–71.

[25] Kouyanou K, Pither C, Rabe-Heskith S, Wessely S. A comparative study of iatrogenesis, medication abuse, and psychiatric morbidity in chronic pain patients with and without medically unexplained symptoms. Pain 1998;76:417–26.

[26] Kisely S, Gater R, Goldberg D. Results from the Manchester centre. In: Ustun B, Sartorius N, editors. Mental illness in general health care: an international study. Chichester: Wiley, 1995. pp. 175–92.

[27] McDaniel JS, Musselman DL, Porter MR, Reed DA, Nemeroff CB. Depression in patients with cancer: diagnosis, biology and treatment. Arch Gen Psychiatry 1995;52:89–99.

2. **Anne Weiland, Rianne E. Van de Kraats, Annette H. Blankenstein, Jan L. C. M. Van Saase, Henk T. Van der Molen, Wichor M. Bramer, Alexandra M. Van Dulmen, Lidia R. Arends**
   **Encounters between medical specialists and patients with medically unexplained physical symptoms; influences of communication on patient outcomes and use of health care: a literature overview.**
   **Perspect Med Educ (2012) 1:192–206**

Perspect Med Educ (2012) 1:192–206
DOI 10.1007/s40037-012-0025-0

REVIEW ARTICLE

# Encounters between medical specialists and patients with medically unexplained physical symptoms; influences of communication on patient outcomes and use of health care: a literature overview

**Anne Weiland · Rianne E. Van de Kraats · Annette H. Blankenstein · Jan L. C. M. Van Saase · Henk T. Van der Molen · Wichor M. Bramer · Alexandra M. Van Dulmen · Lidia R. Arends**

Published online: 27 September 2012


## Abstract
Medically unexplained physical symptoms (MUPS) burden patients and health services due to large quantities of consultations and medical interventions. The aim of this study is to determine which elements of communication in non-psychiatric specialist MUPS care influence health outcomes. Systematic search in PubMed,

A. Weiland (✉)
Department of Internal Medicine, Erasmus MC, University Medical Center, P.O. Box 2040, 3000 CA Rotterdam, the Netherlands
email: info@anneweiland.nl

A. Weiland
Faculty of Social Sciences, Institute of Psychology, Erasmus University Rotterdam, Rotterdam, the Netherlands

R. E. Van de Kraats
Department of Psychiatry, University Medical Center Utrecht, Utrecht, the Netherlands

A. H. Blankenstein
Department of General Practice & Elderly Care Medicine, VU University Medical Center, Amsterdam, the Netherlands

J. L. C. M. Van Saase
Department of Internal Medicine, Erasmus MC, University Medical Center, Rotterdam, the Netherlands

H. T. Van der Molen
Faculty of Social Sciences, Institute of Psychology, Erasmus University Rotterdam, Rotterdam, the Netherlands

W. M. Bramer
Medical Library, Erasmus MC, University Medical Center, Rotterdam, the Netherlands

A. M. Van Dulmen
Netherlands Institute for Health Services Research (NIVEL), Utrecht, the Netherlands

PsycINFO and Embase. Data extraction comprising study design, patient characteristics, number of patients, communication strategies, outcome measures and results. Elements of doctor-patient communication were framed according to symptoms, health anxiety, satisfaction, daily functioning and use of health care. Eight included studies. Two studies described the effect of communication on patient outcome in physical symptoms, three studies on health anxiety and patient satisfaction and one study on daily functioning. Two studies contained research on use of health care. Qualitative synthesis of findings was conducted. Communication matters in non-psychiatric MUPS specialist care. Perceiving patients' expectations correctly enables specialists to influence patients' cognitions, to reduce patients' anxiety and improve patients' satisfaction. Patients report less symptoms and health anxiety when symptoms are properly explained. Positive interaction and feedback reduces use of health care and improves coping. Development of communication skills focused on MUPS patients should be part of postgraduate education for medical specialists.

**Keywords** Medically unexplained physical symptoms · Physician-patient relations · Communication · Medical specialists · Use of Health Care

## Introduction

Communication, defined as the intentional verbal and non-verbal actions of a health professional, is generally understood to be an important component of patient care [1]. A systematic review of randomized clinical trials and descriptive studies about physician-patient communication indeed revealed a positive influence of effective communication on health outcomes [2]. When physicians have no medical explanation for persisting physical symptoms (e.g. chronic fatigue syndrome, irritable bowel syndrome (IBS), chronic pain syndrome, fibromyalgia syndrome, chronic pelvic pain, pseudo non-epileptic seizures) many patients feel that they are not being taken seriously, whereas doctors often feel unable to come to an agreement with their patients on problem definition [3]. Dissatisfaction and pressure on the

A. M. Van Dulmen
Department of Primary and Community Care, Radboud University Nijmegen Medical Centre, Nijmegen, the Netherlands

A. M. Van Dulmen
Department of Health Science, Buskerud University College, Drammen, Norway

L. R. Arends
Faculty of Social Sciences, Institute of Psychology and Institute of Pedagogy, Erasmus University, Rotterdam, the Netherlands

L. R. Arends
Department of Biostatistics, Erasmus MC, University Medical Center, Rotterdam, the Netherlands

doctor-patient relationship hamper their communication. The health outcome of patients with MUPS in primary care can be influenced positively by patient-centred communication, effective reassurance, reliable patient information and a clear and positive explanation about the nature of the symptoms [4–8].

Patient-centred communication in general is incorporated in Dutch undergraduate medical education. MUPS-focused communication skills training is available in postgraduate education for GPs and trainees [9] but not for medical specialists and residents.

Since at least 40 % of physical symptoms presented in outpatient clinics of gynaecology, neurology or rheumatology remain medically unexplained [10–12], medical specialists could benefit from MUPS-focused training programmes. MUPS burden patients and health services due to large quantities of consultations and medical interventions [13]. Comorbidity, lack of clear guidelines and limited knowledge about MUPS among non-psychiatric specialists [14–16] often cause unnecessary medical interventions and unintentionally reinforce somatisation [17]. Normal test results of additional specialist investigations naturally do not reassure MUPS patients [18, 19].

In short, MUPS in specialist care is a big issue. Therefore, we want to explore what is known about effective physician-patient communication in MUPS specialist care. Are there MUPS-focused communication strategies for specialists? Does communication matter in MUPS specialist care?

### Objective

To study the questions above, our objective is: 'Which elements of doctor-patient communication by non-psychiatric specialists in patients with MUPS influence symptoms, health anxiety, satisfaction, daily functioning and use of health care?' These specific outcome measures were used in different types of health care research [20–24]. MUPS specialist care, being far more costly than general care, could benefit from improving these outcomes.

## Methods

### Data sources and search strategy

We conducted systematic searches in the electronic databases PubMed, Embase and PsycINFO in April 2011. Medically unexplained physical symptoms was searched in four different ways. The word 'unexplained' and its synonym was combined with 'subjective symptoms' and its synonyms, with behaviours often occurring in MUPS patients and for general complaints (such as headache) combined with factors that make it unexplained (such as chronic). This search for MUPS was combined with a search for non-psychiatric specialist or secondary care and their synonyms and with a search for interaction as a combination of synonyms for the word professional near the word patient. Table 1 shows the complete search string in Embase.

**Table 1** Search for www.embase.com

| | |
|---|---|
| #1 | (unexplain* OR (un NEXT/1 explain*) OR ('not' NEXT/3 explain*)):de,ab,ti |
| #2 | (nonspecific* OR (non NEXT/1 specific*) OR ('not' NEXT/3 specific*)):de,ab,ti |
| #3 | ((subjective OR Somatoform OR functional) NEXT/5 (symptom* OR disorder* OR complaint*)): de,ab,ti |
| #4 | ((frequent NEXT/1 attend*) OR (high NEXT/1 utili*) OR hypochondri*):de,ab,ti |
| #5 | ((Headache OR 'chest pain' OR 'neck pain' OR 'pelvic pain' OR 'benign pain' OR 'back pain' OR trauma OR 'chemical sensitivity' OR gastrointest* OR dyspepsia OR seizure* OR Fatigue OR dizziness OR hysteri* OR premenstrual OR 'irritable bowel' OR fibromyalgia) NEAR/3 (psycholog* OR psychogen* OR Psychosom* OR Psychophysiol* OR functional* OR chronic OR syndrome OR non-cardiac OR noncardiac OR Tension OR cumulative OR multiple)):de,ab,ti |
| #6 | #1 OR #2 OR #3 OR #4 OR #5 |
| #7 | (specialis* OR specialization OR physician* OR (vocational NEXT/1 trainee*) OR intern OR interns OR resident* OR 'secondary care' OR hospital*):de,ab,ti |
| #8 | ((professional* OR doctor* OR physician* OR provider*) NEAR/3 patient):de,ab,ti |
| #9 | #6 AND #7 AND #8 |

### Study inclusion and selection

Studies were eligible for selection if they were published in peer-reviewed journals in English, German, French or Dutch; involved an adult human population; had a publication year between January 1984, when PubMed started, and April 2011; had an empirical study design; and contained an outcome at patient level in terms of symptoms, health anxiety, satisfaction, daily functioning or the use of health care. After removing the duplicates, two authors (AW, RK) independently screened titles and abstracts to select eligible studies; selection was checked by two co-authors (AB, LA), who each revised the first selection. Full text papers were obtained of the selected studies. AW and RK independently critically appraised the full-text papers and excluded studies that did not meet the inclusion criteria. Disagreement was solved by discussion between authors (AW, RK, AB, LA).

### Data extraction and analysis

For all included studies, data extraction was undertaken comprising study design, patient characteristics, number of patients, communication aspects, and outcomes, as shown in Table 2. Meta-analysis was not feasible due to the small number of studies and variety in study design and outcome measures; therefore a qualitative synthesis of findings was conducted.

## Results

### Selection of studies

The combined search resulted in 1981 articles. After screening titles and abstracts, 74 articles met the inclusion criteria and were retrieved for further assessment. Two

**Table 2** Overview of included studies

| Author, year[Ref #] | Study design | Study group | Number of patients | Outcome | Intervention/study subject | Effect |
|---|---|---|---|---|---|---|
| Bieber 2008 [30] | RCT | Fibromyalgia syndrome patients | 83 | Patient satisfaction | A shared decision-making training programme for specialists combined with an information leaflet for patients versus information leaflet only | No difference in patient satisfaction was found in the shared decision making group and the information only group |
| Bieber 2006 [31] | RCT | Fibromyalgia syndrome patients | 67 | Functioning | A shared decision-making training programme for specialists combined with an information leaflet for patients versus information leaflet only or standard care as usual | Functional capacity did not differ across the study groups. The patients of the share decision making study group improved coping with pain and being more positive |
| Collins 2009 [28] | Cohort study | Patients with functional gastrointestinal disorders | 13 | Health anxiety<br>Use of health care | Concordance between specialists' understanding of patients reported symptoms and their actual needs | Underestimating patients' expectations and symptoms maintained health anxiety and was likely to lead to more use of health care |
| Van Dulmen 1995 [27] | Cohort study | Patients with functional abdominal pain | 110 | Patient satisfaction<br>Health anxiety | Correct perceptions of patients' attributions and having the same doctor | Reduced health anxiety ($p = 0.01$) and improved satisfaction by consulting the same doctor ($p = 0.02$) |
| Hall-Patch 2010 [25] | Cohort study | Patients with pseudo neurological epileptic seizures | 50 | Course of symptoms | A patient information leaflet and a communication protocol for neurologists to explain the psychological nature of the seizures | Reduced frequency of seizures |
| Owens 1995 [32] | Cohort study | Patients with irritable bowel syndrome | 112 | Use of health care | Physician-patient relationship on use of health care | Reduced number of return visits for IBS-related symptoms |

**Table 2** continued

| Author, year[Ref #] | Study design | Study group | Number of patients | Outcome | Intervention/study subject | Effect |
|---|---|---|---|---|---|---|
| Petrie 2007 [26] | RCT | Patients with nonspecific chest pain | 92 | Health anxiety<br>Course of symptoms | Providing information about normal test results before testing | The number of patients still reporting chest pain after 1 month decreased significantly ($p < 0.001$). Addressing patients' attributions by information about normal test results prior to testing diminished health anxiety |
| Stones 2006 [29] | Cohort study | Women with chronic pelvic pain | 100 | Patient satisfaction | Doctors affect, appropriateness of information and ability to meet patients expectations | Initial consultation influenced further care experiences. Doctors affect, appropriate information and meeting patients' expectations enhanced patient satisfaction |

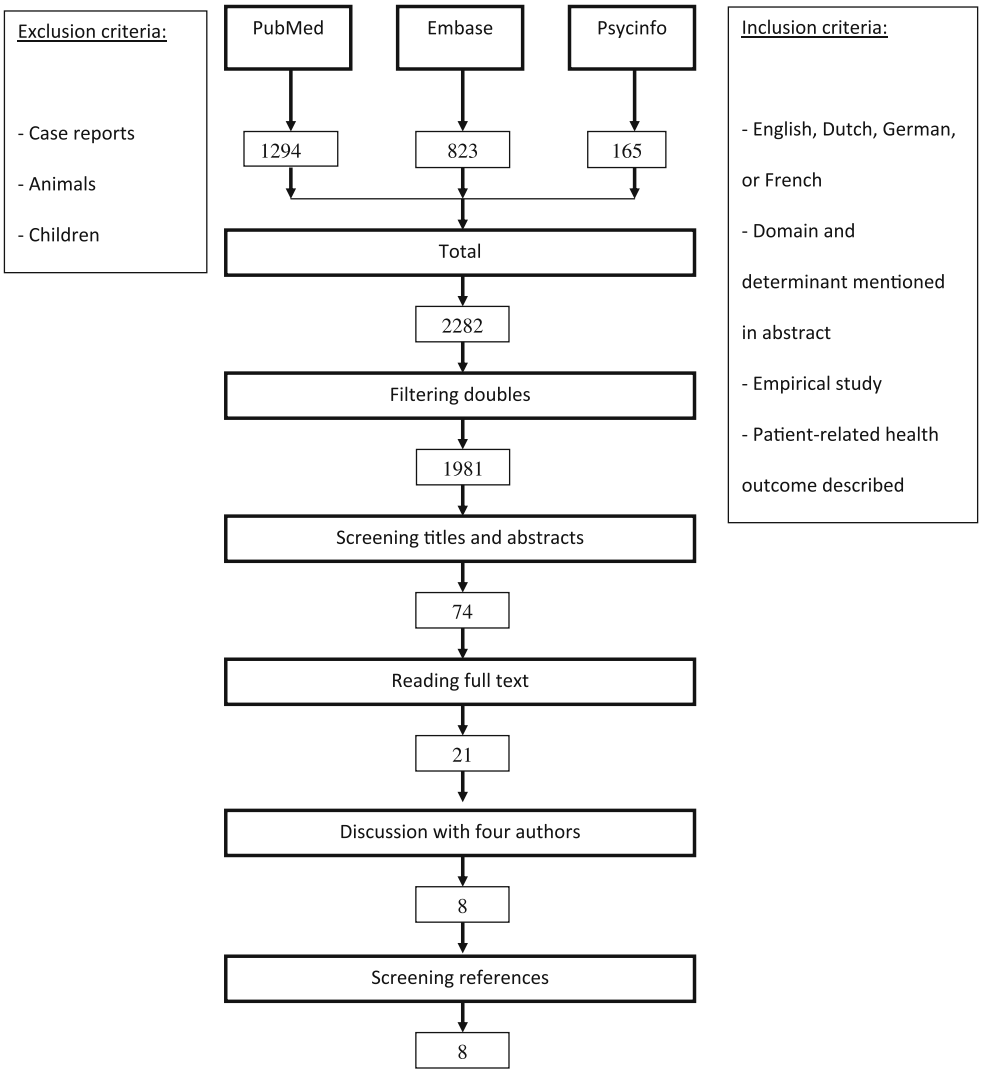



**Fig. 1** Flow chart

authors (AW, RK) reviewed these full-text articles and selected 21 articles according to inclusion and exclusion criteria. Discussion with four authors (AW, RK, AB, LA) reduced the number to eight eligible studies. Thirteen articles were excluded because they lacked outcomes fitting our study question. Of the selected articles, a thorough search of related articles, references and citing articles was performed. This yielded no extra article for inclusion. Figure 1 presents the flowchart of the systematic search.

## Synthesis of findings

The included articles discuss different types of MUPS patients, and describe different elements of communication strategies used by medical specialists that may have an impact on health outcomes and use of health care. We framed and summarised these

elements of doctor-patient communication according to the outcomes defined in our study question: symptoms, health anxiety, satisfaction, daily functioning and use of health care.

### Symptoms

In the study by Hall-Patch et al. [25] most patients with psychogenic non-epileptic seizures (PNES) were initially diagnosed as having epilepsy and had been treated with antiepileptics for several years. Participants received the diagnosis of PNES on average 5.2 years after seizure manifestation. The study was carried out to assess the acceptability and effectiveness of a patient information leaflet and a communication protocol for neurologists to explain the psychological nature of the seizures to their patients; 44 patients positively evaluated the information leaflet. The frequency of the seizures between diagnosis and follow-up after 3 months was reduced by more than 50 % in 63 % of the patients; 14 % of the patients were seizure-free after 3 months.

Petrie et al. [26] investigated whether providing information about normal findings prior to a diagnostic test improves patients' reassurance and reduces health anxiety. They studied 92 patients with chest pain who were referred for a diagnostic exercise stress test. These patients were divided into a group of 30 patients receiving a pamphlet explaining the function and meaning of normal test results; a group of 34 patients receiving the pamphlet and a brief discussion about the meaning of normal test results and a control group of 28 patients receiving standard information. The number of patients still reporting chest pain after 1 month decreased significantly in the discussion group ($p < 0.001$) and pamphlet group ($p = 0.005$) but not in the control group ($p = 0.09$). Another finding was that fewer patients in the discussion group were taking cardiac drugs after 1 month. In conclusion, explaining the nature of MUPS with an information leaflet, a core points crib sheet for specialists and a brief discussion about the meaning of normal test results prior to testing reduces symptoms in patients.

### Health anxiety

The study by Petrie et al. [26], mentioned above showed that the mean levels of reassurance in patients with chest pain after testing and feedback from the doctor were significantly higher in the discussion group [$M = 42.0$, 95 % confidence interval (CI) 39.7–44.2] than in the pamphlet group ($M = 39.2$, 95 % CI 36.1–42.3) and control group ($M = 35.8$, 95 % CI 31.6–39.9). This difference was maintained after 1 month. So, addressing patients attributions and providing patients with information about normal test results before testing can improve reassurance and thus diminish health anxiety.

Van Dulmen et al. [27] explored changes in complaint-related cognitions and anxiety of 110 patients with IBS during a series of consultations in an outpatient clinic of internal medicine. They found that anxiety ($p = 0.01$), fear of cancer ($p < 0.001$), somatic attributions ($p < 0.001$) and catastrophising cognitions ($p = 0.008$) diminished significantly between the first and last consultation of patients with IBS. Aspects of communication that accounted for the measured effects

were doctors' correct perceptions of patients' attributions and having the same doctor throughout the consultations.

Collins et al. [28] studied concordance between 13 eligible patients with functional gastrointestinal disorders (FGID) and doctors (11 gastroenterologists and 13 GPs). They investigated patients' needs and expectations at initial consultations and whether their specialists and GPs recognised these patient perceptions. Gastroenterologists underestimated patients' reported number of symptoms (82 %), pain (48 %), and interference with daily functioning (41 %). Views on the best treatment options diverged: patients preferred operation (41 %) or diet (31 %), whereas the specialists were focused on symptom control by medication (41 %) or managing worry (28 %). A persisting expectation of finding a specific cause and cure was present in these patients. Only one out of 13 patients acknowledged the diagnosis FGID at follow-up. So, underestimating patients' expectations and symptoms does not reassure patients and maintains existing health anxiety.

### Patient satisfaction

Van Dulmen et al. [27] found that patients whose anxiety diminished ($N = 59$) were more satisfied with the visit to the doctor than patients whose anxiety did not diminish ($p = 0.02$). Patients consulting the same doctor throughout the consultations were more satisfied with the consultations than patients who visited different doctors ($p = 0.05$).

The study by Stones et al. [29] aimed to identify the three dimensions of patient satisfaction (affect, cognition and expectation) through which initial consultations were subsequently recalled at follow-up in 100 gynaecology patients with chronic pelvic pain (CPP). These authors demonstrated that doctors' affect, appropriateness of information and the ability to meet patients' expectations are strong influences on experiences of care. These three elements of patient satisfaction were interrelated and influenced the experiences of care. Building a good relationship in the first hospital visit improves the understanding of the diagnosis and makes a positive coping of the patient more likely.

Bieber et al. [30, 31] assessed whether shared decision-making improves the quality of physician-patient interaction from the perspective of the patient in 85 patients with fibromyalgia syndrome. They measured patient satisfaction with the decisions and did not find significant group differences. Decisional conflicts and satisfaction with decisions were similar in the study groups.

### Daily functioning

Bieber et al. [30, 31] found that fibromyalgia syndrome patients benefit from a shared decision-making communication training programme for physicians combined with an information package for patients. During the training, doctors learned to consider their patients' individual needs and to meet their patients' expectations. These elements accounted for a better physician-patient interaction. Qualitative assessment revealed a dramatic difference: at 1-year follow-up more patients in the shared decision-making group (62 %) than in the care as usual group (28 %) mentioned that

their coping with pain had improved. Patients from the shared decision group adopted a more positive view when thinking of the future with their illness than patients from the care as usual group.

### Use of health care

Collins et al. [28] suggest that failure of patients to acknowledge their diagnosis of FGID might underpin recurrent consultations and possibly leads to unnecessary use of health care. Patients who believe that their symptoms are not adequately explained are not able to accept the diagnosis. Collins et al. also found that when patients seek specialist consultation, the reason for the visit often remains unclear to the specialist. Possible reasons found are the need of diagnosing the cause of symptoms and the initiation or the readjustment of treatment. Effective consultation with MUPS patients starts with exploring the reason why the patient visits the doctor.

Owens et al. [32] found that a strong physician-patient interaction may be related to a reduced number of return visits for patients with IBS. Comparison of the strongest and weakest interaction groups (1.8 and 4.9 hospitalizations, respectively; $p < 0.05$) indicated that positive interaction was associated with fewer hospitalizations. However, the authors found no association between strength of the physician-patient interaction and number of surgeries. Notation in medical records of the patient's psychosocial history ($p < 0.01$) about precipitating factors causing the patient to seek medical help ($p < 0.01$) and notation of discussions with the patient ($p < 0.02$) were associated with fewer follow-up visits for IBS-related symptoms.

## Discussion

### Main findings

This review demonstrates that the research on specialist communication with MUPS patients and its effect on patient outcomes and use of health care is limited. We did not restrict our search to RCTs and CCTs. Despite having broad inclusion criteria we only found 8 studies describing different outcomes and aspects of communication:

1. Perceiving patients' expectations correctly enables specialists to influence patients' cognitions, reduces patients' anxiety, and improves patient satisfaction [27].
2. Explaining the nature of MUPS with an information leaflet and a core points crib sheet for specialists reduces health anxiety and symptoms in patients [25].
3. Providing patients with information about normal test results prior to investigation helps to reassure patients [26].
4. Positive doctor–patient interaction [28, 29] and positive feedback from the doctor contributes to reduced use of health care [32] and better coping with complaints in the long term [30, 31].

Incorporating these four elements in a vocational and postgraduate MUPS-focused communication skills training for specialists could improve MUPS specialist care and support specialists in their consultations with MUPS patients.

## Comparison with the literature

We found that proper explanation and showing an effect in communication with MUPS patients in specialist care improve patient outcomes and reduce the use of health care. Specialists trained in shared decision-making [30, 31] and in communicating the diagnosis MUPS to patients [27] influenced health outcomes positively. These elements are also important in general practice and in patients with minor ailments. Blankenstein et al. [33] found that trained GPs were able to apply cognitive-behavioural techniques to patients with MUPS during normal consultation hours. At follow-up subjective health was increased, use of health care and sick-leave were decreased [26]. Fassaert et al. [5] studied positive communication strategies during 524 videotaped consultations in general practice with patients with minor ailments related to medication adherence, consultation frequency, functional health status and state anxiety. Results show that, to some extent, it seems helpful when GPs are at the same time clear and optimistic about the nature and course of minor ailments. Results of this study indicate that it is important for physicians to pay attention to the patients' mood. Thomas studied 200 patients in general practice who presented symptoms without abnormal physical signs and in whom no definite diagnosis was made. Patients who received a positive consultation from their GP for their symptoms were more likely to improve than those who received no explanation [8]. Sometimes MUPS patients are referred frequently to secondary care even after having received multiple specialist opinions that their symptoms were medically unexplained [34]. Referring MUPS patients to hospital clinics repeatedly is not the best way to address their needs [35]. These patients are unlikely to benefit from repeated referrals to specialist services that are designed to find or exclude disease rather than to deal with symptoms [36]. Positive communication between specialists and GPs is required to reduce unnecessary medical interventions, use of health care and aggravation of symptoms, and improves care for MUPS patients by sharing knowledge and stepped care [37].

## Strengths and limitations of this review

This review is the first paper to give an overview on the knowledge of doctor-patient MUPS-focused communication in specialist care. Although the selected studies contain a limited variety of MUPS, patient characteristics and aetiological mechanisms appear to be quite similar for different MUPS [38]. Therefore our results can probably be transferred to MUPS patients in general. From all selected studies, only three described explicit communication programmes for specialists [25, 26, 31].This indicates the low priority in specialist care for MUPS-focused communication. Enhancement of knowledge and communication skills might improve specialist care for MUPS patients [39, 40]. Methodological and clinical variety of the studies and small number of (quantitative) studies made pooling of results of the different studies not useful.

## Conclusion

This review shows that communication matters in specialist care. Perceiving patients' expectations correctly enables specialists to influence patients' cognitions, reduce patients' anxiety and improve patients' satisfaction. Providing patients with information helps them to feel reassured. Patients report less symptoms and health anxiety when they get a proper explanation of their symptoms. Positive doctor-patient interaction and positive feedback from the doctor reduces the use of health care and improves coping with complaints on the long term. These elements should be integrated in postgraduate education for specialists.

Recommendations for research and post-graduate education

First, we recommend further research on communication with MUPS patients in non-psychiatric specialist care and related health outcomes. Second, we recommend research on postgraduate education in specialist care for MUPS patients to enhance communication skills for specialists that contribute to the quality of specialist care for MUPS patients.

## Essentials

- Explaining the nature of MUPS with an information leaflet and a core points crib sheet for specialists reduces health anxiety and symptoms in patients.
- Perceiving patients' expectations correctly enables specialists to influence patients' cognitions and reduces patients' anxiety and improves satisfaction.
- Providing patients with information about normal test results prior to investigation helps to reassure patients.
- Positive doctor-patient interaction and positive feedback from the doctor contributes to reduced use of health care and better coping in the long term with complaints.

**Acknowledgments** This study has been done without any grants.



## References

1. Bensing JM, Sluijs EM. Evaluation of an interview training course for general practitioners. Soc Sci Med. 1985;20(7):737–44.
2. Teutsch C. Patient-doctor communication. Med Clin North Am. 2003;87(5):1115–45.
3. Salmon P. Conflict, collusion or collaboration in consultations about medically unexplained symptoms: the need for a curriculum of medical explanation. Patient Educ Couns. 2007;67(3):246–54.
4. Dowrick CF, Ring A, Humphris GM, Salmon P. Normalisation of unexplained symptoms by general practitioners: a functional typology. Br J Gen Pract. 2004;54(500):165–70.

5. Fassaert T, van Dulmen S, Schellevis F, van der Jagt L, Bensing J. Raising positive expectations helps patients with minor ailments: a cross-sectional study. BMC Fam Pract. 2008;9:38.
6. Kappen T, van Dulmen S. General practitioners' responses to the initial presentation of medically unexplained symptoms: a quantitative analysis. Biopsychosoc Med. 2008;2:22.
7. Salmon P, Peters S, Stanley I. Patients' perceptions of medical explanations for somatisation disorders: qualitative analysis. Br Med J. 1999;318(7180):372–6.
8. Thomas KB. General practice consultations: is there any point in being positive? Br Med J (Clin Res Ed). 1987;294(6581):1200–2.
9. Blankenstein AH, van der Horst HE, Essers G, Floor E. Training Course Policy towards MUPS patients in general practice; use of CBT for the GP. 2003.
10. Maiden NL, Hurst NP, Lochhead A, Carson AJ, Sharpe M. Medically unexplained symptoms in patients referred to a specialist rheumatology service: prevalence and associations. Rheumatology (Oxford). 2003;42(1):108–12.
11. Nimnuan C, Hotopf M, Wessely S. Medically unexplained symptoms: an epidemiological study in seven specialities. J Psychosom Res. 2001;51(1):361–7.
12. Reid S, Wessely S, Crayford T, Hotopf M. Medically unexplained symptoms in frequent attenders of secondary health care: retrospective cohort study. Br Med J. 2001;322(7289):767.
13. Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. Arch Gen Psychiatry. 2005;62(8):903–10.
14. Carson AJ, Ringbauer B, Stone J, McKenzie L, Warlow C, Sharpe M. Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. J Neurol Neurosurg Psychiatry. 2000;68(2):207–10.
15. Hamilton J, Campos R, Creed F. Anxiety, depression and management of medically unexplained symptoms in medical clinics. J R Coll Physicians Lond. 1996;30(1):18–20.
16. Kroenke K, Mangelsdorff AD. Common symptoms in ambulatory care: incidence, evaluation, therapy, and outcome. Am J Med. 1989;86(3):262–6.
17. Stephenson DT, Price JR. Medically unexplained physical symptoms in emergency medicine. Emerg Med J. 2006;23(8):595–600.
18. Mayou RA, Bass CM, Bryant BM. Management of non-cardiac chest pain: from research to clinical practice. Heart. 1999;81(4):387–92.
19. McDonald IG, Daly J, Jelinek VM, Panetta F, Gutman JM. Opening Pandora's box: the unpredictability of reassurance by a normal test result. Br Med J. 1996;313(7053):329–32.
20. Blankenstein AH. Somatising patients in general practice. Reattribution, a promising approach. http://hdl.handle.net/1871/11723. (Thesis). 2001.
21. de Beurs EBA, van Balkom AJ, Deeg DJ, van Dyck R, van Tilburg W. Consequences of anxiety in older persons: its effect on disability, well-being and use of health services. Psychol Med. 1999; 29(3):583–93.
22. Gotay CC, Korn EL, McCabe MS, Moore TD, Cheson BD. Quality-of-life assessment in cancer treatment protocols: research issues in protocol development. J Natl Cancer Inst. 1992;84(8):575–9.
23. Escobar JI, Gara MA, Diaz-Martinez AM, Interian A, Warman M, Allen LA, Woolfolk RL, Jahn E, Rodgers D. Effectiveness of a time-limited cognitive behavior therapy type intervention among primary care patients with medically unexplained symptoms. Ann Fam Med. 2007;5(4):328–35.
24. DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. Arch Intern Med. 2000;160(14):2101–7.
25. Hall-Patch L, Brown R, House A, Howlett S, Kemp S, Lawton G, et al. Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures. Epilepsia. 2010;51(1):70–8.
26. Petrie KJ, Muller JT, Schirmbeck F, Donkin L, Broadbent E, Ellis CJ, et al. Effect of providing information about normal test results on patients' reassurance: randomised controlled trial. Br Med J. 2007;334(7589):352.
27. Van Dulmen AM, Fennis JF, Mokkink HG, Van der Velden HG, Bleijenberg G. Doctor-dependent changes in complaint-related cognitions and anxiety during medical consultations in functional abdominal complaints. Psychol Med. 1995;25(5):1011–8.
28. Collins J, Farrall E, Turnbull DA, Hetzel DJ, Holtmann G, Andrews JM. Do we know what patients want? The doctor-patient communication gap in functional gastrointestinal disorders. Clin Gastroenterol Hepatol. 2009;7(11):1252–4. 4 e1-2.

29. Stones RW, Lawrence WT, Selfe SA. Lasting impressions: influence of the initial hospital consultation for chronic pelvic pain on dimensions of patient satisfaction at follow-up. J Psychosom Res. 2006;60(2):163–7.
30. Bieber C, Muller KG, Blumenstiel K, Hochlehnert A, Wilke S, Hartmann M, et al. A shared decision-making communication training program for physicians treating fibromyalgia patients: effects of a randomized controlled trial. J Psychosom Res. 2008;64(1):13–20.
31. Bieber C, Muller KG, Blumenstiel K, Schneider A, Richter A, Wilke S, et al. Long-term effects of a shared decision-making intervention on physician-patient interaction and outcome in fibromyalgia. A qualitative and quantitative 1 year follow-up of a randomized controlled trial. Patient Educ Couns. 2006;63(3):357–66.
32. Owens DM, Nelson DK, Talley NJ. The irritable bowel syndrome: long-term prognosis and the physician-patient interaction. Ann Intern Med. 1995;122(2):107–12.
33. Blankenstein AH, van der Horst HE, Schilte AF, de Vries D, Zaat JO, Andre Knottnerus J, et al. Development and feasibility of a modified reattribution model for somatising patients, applied by their own general practitioners. Patient Educ Couns. 2002;47(3):229–35.
34. McGorm K, Burton C, Weller D, Murray G, Sharpe M. Patients repeatedly referred to secondary care with symptoms unexplained by organic disease: prevalence, characteristics and referral pattern. Fam Pract. 2010;27(5):479–86.
35. Salmon P, Ring A, Dowrick CF, Humphris GM. What do general practice patients want when they present medically unexplained symptoms, and why do their doctors feel pressurized? J Psychosom Res. 2005;59(4):255–60. discussion 61-2.
36. Dominice Dao M, Kirmayer L, Groleau D, Young A. Mingling mind and body: patients' complex networks of meaning in the absence of medical diagnosis. J Psychosom Res. 2006;61:410.
37. National Steering Committee Guideline Development in the Mental Health Care. Dutch Multidisciplinary Guideline on MUPS and somatoform disorders. Trimbos-Instituut, Netherlands Institute of Mental Health and Addiction; 2010; Available from: http://www.ggzrichtlijnen.nl.
38. Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? Lancet. 1999;354(9182):936–9.
39. Donovan JL, Blake DR. Qualitative study of interpretation of reassurance among patients attending rheumatology clinics: 'just a touch of arthritis, doctor?'. Br Med J. 2000;320(7234):541–4.
40. Kenny DT. Constructions of chronic pain in doctor-patient relationships: bridging the communication chasm. Patient Educ Couns. 2004;52(3):297–305.

## Author Biographies

**Anne Weiland** is specialist in adult education and has a broad experience in the education of medical professionals. As behavioural scientist and practitioner, she has developed and performed consultation skills training programmes for GPs and medical specialists on MUPS. She wrote this article and contributed to all chapters of this review.

**Rianne E. van de Kraats** is resident psychiatry and dedicated to patients with MUPS. As second author she contributed to all chapters of this review.

**Annette H. Blankenstein** is head of the GP education programme and a member of the editorial committee for the Dutch Multi Disciplinary Guideline on MUPS. She developed and supervised GP training programmes on MUPS patients. She was involved from study question to discussion in this review.

**Jan L. C. M. Van Saase** is head of the internal medicine education programme and endowed professor with a special interest in competence-oriented education of specialists. As dedicated clinician he supervised this review and contributed with his feedback to the results and discussion.

**Henk T. van der Molen** is specialist in the development, execution and evaluation of communication skill training programmes. From that experience he was involved in giving feedback on this literature review several times.

**Wichor M. Bramer** is information specialist, designed the search string in cooperation with the first authors and supervised the search strategy.

**Alexandra M. van Dulmen** is specialist in communication in health care research and MUPS. Her PhD thesis was on 'Exploring cognitions in IBS: implications for the role of the doctor'. Her critical remarks and advice on this literature review were her contribution.

**Lidia R. Arends** has statistical expertise in the development of new techniques for multivariate meta-analysis. She is involved in the Generation R Study and was one of the first researchers to participate in cost-effectiveness analyses of mental health care. For this review she participated in selection of studies, data extraction and analysis.

3. Alexandra Rolfe, MBChB; Christopher Burton, MD
Less is more, Reassurance After Diagnostic Testing
With a Low Pretest Probability of Serious Disease
Systematic Review and Meta-analysis.
JAMA Intern Med. 2013;173(6):407-416.

REVIEW ARTICLE

LESS IS MORE

# Reassurance After Diagnostic Testing With a Low Pretest Probability of Serious Disease

## *Systematic Review and Meta-analysis*

*Alexandra Rolfe, MBChB; Christopher Burton, MD*

**Importance:** Diagnostic tests are often ordered by physicians in patients with a low pretest probability of disease to rule out conditions and reassure the patient.

**Objective:** To study the effect of diagnostic tests on worry about illness, anxiety, symptom persistence, and subsequent use of health care resources in patients with a low pretest probability of serious illness.

**Evidence Acquisition:** Systematic review and meta-analysis. We searched MEDLINE, the Cochrane Central Register of Controlled Trials, EMBASE, PsychINFO, CINAHL, and ProQuest Dissertations electronic databases through December 31, 2011, for eligible randomized controlled trials. We independently identified studies for inclusion and extracted the data. Disagreements were resolved by discussion. We performed meta-analysis if heterogeneity was low or moderate ($I^2 < 50\%$).

**Results:** Fourteen randomized controlled trials that included 3828 patients met the inclusion criteria and were analyzed with outcomes categorized as short term (≤3 months) or long term (>3 months). Three trials showed no overall effect of diagnostic tests on illness worry (odds ratio, 0.87 [95% CI, 0.55-1.39]), and 2 showed no effect on nonspecific anxiety (standardized mean difference, 0.06 [−0.16 to 0.28]). Ten trials showed no overall long-term effect on symptom persistence (odds ratio, 0.99 [95% CI, 0.85-1.15]). Eleven trials measured subsequent primary care visits. We observed a high level of heterogeneity among trials ($I^2$=80%). Meta-analysis after exclusion of outliers suggested a small reduction in visits after investigation (odds ratio, 0.77 [95% CI, 0.62-0.96]).

**Conclusions and Relevance:** Diagnostic tests for symptoms with a low risk of serious illness do little to reassure patients, decrease their anxiety, or resolve their symptoms, although the tests may reduce further primary care visits. Further research is needed to maximize reassurance from medically necessary tests and to develop safe strategies for managing patients without testing when an abnormal result is unlikely.

*JAMA Intern Med. 2013;173(6):407-416.*
*Published online February 25, 2013.*
*doi:10.1001/jamainternmed.2013.2762*

MANY PATIENTS PRESENT in primary care with symptoms that are not caused by serious illness. Approximately one-sixth of primary care visits and more than one-third of referrals from primary to secondary care occur for symptoms for which no organic pathology is apparent.[1-4] Such symptoms with a low probability of disease pose a problem for clinicians and health services in terms of whether or how far to perform diagnostic tests.[5] Although clinical guidelines promote the rational use of diagnostic tests,[6] clinicians often order these tests for patients who do not meet these criteria. Physicians commonly express the belief that patients want these diagnostic tests and that the tests reassure patients[3,7]; consequently, they may propose such tests more often than patients actually seek them.[8,9]

*CME available online at jamanetworkcme.com and questions on page 405*

*See also page 418*

*See Invited Commentary at end of article*

Psychological models of reassurance suggest the following 2 components: short-term emotional relief and longer-term cognitive assurance.[10] The cognitive component of reassurance is necessary for long-term benefit through alterations in symptom appraisal.[11,12] This change in turn leads to reduced anxiety, less awareness of symptoms, less seeking of medical help, and a change in the belief that the symptoms may represent serious disease.[10]

**Author Affiliations:** Centre for Population Health Sciences, University of Edinburgh, Edinburgh, Scotland. Dr Burton is now affiliated with the Centre of Academic Primary Care, University of Aberdeen, Aberdeen, Scotland.

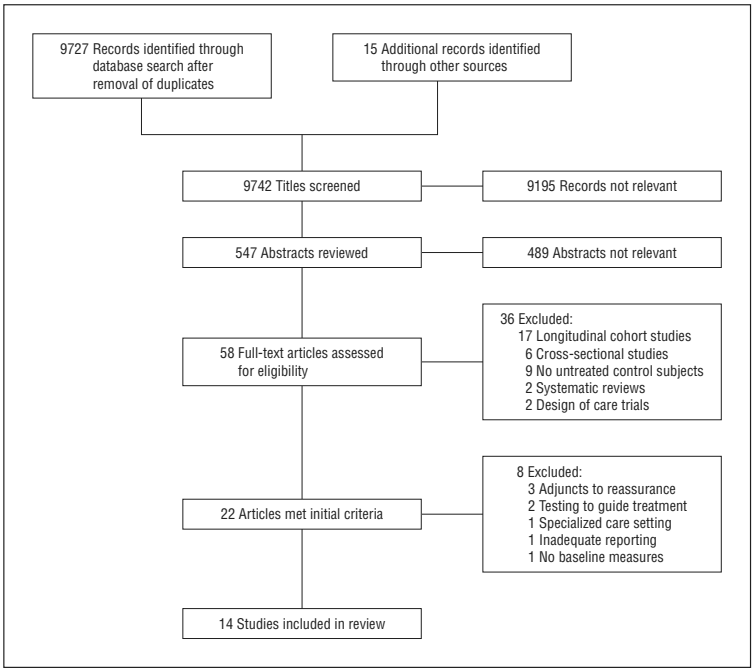



**Figure 1.** Flowchart indicating selection of studies for inclusion. Reasons for exclusion of trials exceed the numbers of excluded trials owing to more than 1 reason applied to some trials.

We performed a systematic review to measure the effect of diagnostic tests on reassurance in patients with a low probability of serious disease. Because a previous systematic review identified very few trials that directly measured reassurance,[13] we expanded the concept of reassurance to include the following 4 separate components and consequences: the specific concern that symptoms might represent serious illness (illness concern), nonspecific anxiety, persistence of the symptoms, and subsequent primary care visits.

## METHODS

We performed a systematic review and meta-analysis of published trials following meta-analysis guidelines[14] and using the preferred reporting items for systematic review and meta-analysis guidelines (the PRISMA statement).[15] The protocol for the review is held by the authors. Because this study did not involve individual patients, ethical approval was not required.

### SEARCH STRATEGY

We performed a systematic search of the OVID MEDLINE, Cochrane Central Register of Controlled Trials, EMBASE, PsychINFO, CINAHL, and ProQuest Dissertations electronic databases for publications from inception through December 31, 2011. The search was designed to be sensitive rather than specific and identified randomized controlled trials that included the following search terms: *reassur*, anxi*, quality of life,* or *satisfaction* (1 of these); *investigat*, test*, imag*, x-ray, radiography, endoscopy, colonoscopy,* or *scan* (1 of these); and *negativ*, normal,* or *benign* (≥1 of these) (eAppendix; http://www.jamainternalmed.com). We included original articles and checked the reference lists of relevant systematic reviews for trials that met the eligibility criteria. Trials published in languages other than English were eligible for inclusion. We also obtained references cited in other identified publications and considered them for inclusion.

### REVIEW OF ELIGIBILITY CRITERIA

We considered trials for inclusion if they met the following 5 criteria:

1. The study design was a randomized controlled trial (including cluster trials).
2. Participants included adults (aged ≥18 years) with symptoms indicating a low probability of serious disease.
3. Interventions consisted of initial diagnostic testing in primary or secondary care for symptoms with a low probability of serious disease based on clinical features. Tests included imaging (radiography, computed tomography, magnetic resonance imaging, and ultrasonography), endoscopy, and cardiac testing. For trials with more than 2 intervention arms, we included the intervention most closely representing testing alone.
4. Comparisons included initial nontesting. Control groups received usual care or empirical treatment. Trials that permitted subsequent diagnostic testing if symptoms persisted were eligible.
5. Outcomes included illness concern, generalized or nonspecific anxiety, change in the original symptoms, and subsequent use of health care resources in primary care defined as subsequent visits to a physician. We defined outcomes as short term (≤3 months) or long term (>3 months); if multiple points after 3 months were available, we used those closest to 1 year.

Trials were excluded if they were not published in a peer-reviewed journal or were undertaken in tertiary care (eg, a spinal surgery center)[16] where the prevalence of serious conditions was expected to be high. In addition, we excluded trials for which outcomes were not reported at baseline and completion.

### TRIAL SELECTION AND ANALYSIS

The methods for trial selection and assessment of risk of bias (using the Cochrane risk of bias tool[17]) are described in detail in the eMethods. Eligible data were converted to odds ratios and displayed as forest plots. If statistical heterogeneity was low to moderate ($I^2 < 50\%$), we performed random-effects meta-analysis using methods from the Cochrane Handbook[18]; details of this procedure are available in the eMethods. We converted odds ratios for the use of health care resources to a number needed to investigate.

## RESULTS

**Figure 1** shows the yield of relevant literature identified from the search strategy and the application of the review eligibility criteria.

**Table 1. Characteristics of RCTs**

| Source[a] | Setting | No. of Patients/ Controls | Mean Age, y | Male Sex, % | Symptom | Dx Test vs Control | Outcomes | Follow-up | Additional Information[b] |
|---|---|---|---|---|---|---|---|---|---|
| Asante et al,[28] 1998; Asante et al,[33] 1999 (1996) | UK Secondary care | 78/76 | 32 | 56 | Dyspepsia | Endoscopy vs no endoscopy | Symptoms Use of health care | 6 mo | Patients aged <45 y and seronegative for *Helicobacter pylori* 14 Controls underwent endoscopy No cancer detected in 92 endoscopies |
| Bytzer et al,[19] 1994 (1989) | Denmark Primary care | 208/206 | 44 | 43 | Dyspepsia | Endoscopy vs empirical treatment | Symptoms Use of health care | 12 mo | Dyspepsia for several years, 10% debilitating symptoms, 33% vomiting, 50% night pain 136 Controls underwent endoscopy 4 Cancers detected in 344 endoscopies |
| Cuddihy et al,[20] 2005 (1998) | US Primary care | 13/11 | 52 | 29 | Dyspepsia | Endoscopy vs empirical treatment | Use of health care | 1.5-6 mo | Additional Dx testing uncommon No cancer detected in endoscopies |
| Delaney et al,[21] 2000 (1995) | UK Primary care | 256/186 | 62 | 49 | Dyspepsia | Endoscopy vs usual care | Symptoms Use of health care | 12-18 mo | Patients aged >50 y with new or recently recurrent dyspepsia 75 Controls underwent endoscopy 8 Cancers detected in 363 endoscopies |
| Delaney et al,[22] 2001 | UK Primary care | 285/193 | 37 | 57 | Dyspepsia | *H pylori* testing with selective endoscopy vs usual care | Symptoms Use of health care | 12-18 mo | Patients aged <50 y with dyspepsia >4 wk 127 Patients and 48 controls underwent endoscopy No cancers detected in endoscopies |
| Djais and Kalim,[29] 2005 (2003) | Indonesia Secondary care | 51/50 | 40 | 55 | Back pain | Radiography vs usual care | Symptoms | 3 wk | Low back pain <3 mo No serious disease detected |
| Duggan et al,[23] 2009 (1995) | UK Primary care | 187/178 | 45% > 50 | 55 | Dyspepsia | Endoscopy vs empirical treatment | Symptoms Use of health care | 2-12 mo | 70 Controls underwent endoscopy No cancer detected |

*(continued)*

## STUDY CHARACTERISTICS

We found 14 trials comparing testing with nontesting; these trials included 3828 patients. All the trials were published in English. Nine trials took place in a general practice/family medicine setting,[19-27] and 5, in a general or specialist internal medicine practice setting.[28-32] Eight of the trials involved diagnostic testing for dyspepsia (endoscopy or radiography)[19-23,27,28,30]; 3 involved radiography for back pain.[25,26,29] One each involved blood tests and electrocardiography for chest pain,[32] imaging for headache,[31] and continuous event recorders for palpitations.[24] With the exception of 2 trials,[19,27] studies involved recent rather than persistent symptoms. Several studies specified a minimum duration to exclude trivial conditions for which diagnostic testing may not be clinically necessary. Trials comparing testing with nontesting reported outcome data on illness concern,[25,31,32] nonspecific or general anxiety,[24,26] symptoms,[19,21-23,25-30,32] and subsequent primary care visits.[19-23,25-28,31,32] Ten trials reported short-term data[20,23-27,29-32] and 13, long-term data.[19-28,30-32] Long-term follow-up varied from 4 to 18 months. Substantial variation existed between trials in the measurement tools used. Trial characteristics are summarized in **Table 1** and main outcomes in **Table 2**.

## RISK OF BIAS

**Figure 2** summarizes the risk of bias for all included trials. All trials were randomized at the level of the individual patient. No study had clearly inadequate randomization procedures or allocation concealment, although details were unclear in 6 trials for each of these criteria.[19,24,27,29,30,32] One study used a modified Zelen preconsent randomization to minimize the risk of patients feeling more anxious about being declined a diagnostic test.[31] Most studies were of moderate quality; outcomes from those with the highest quality[23,27,31,35] did not differ noticeably from the other outcomes.

**Table 1. Characteristics of RCTs (continued)**

| Source[a] | Setting | No. of Patients/ Controls | Mean Age, y | Male Sex, % | Symptom | Dx Test vs Control | Outcomes | Follow-up | Additional Information[b] |
|---|---|---|---|---|---|---|---|---|---|
| Giannini et al,[30] 2008 | Italy Secondary care | 303/309 | 44 | 57 | Dyspepsia (GERD only) | Endoscopy vs empirical treatment | Symptoms | 1-6 mo | ≥3 mo typical GERD symptoms<br>42 Controls underwent endoscopy<br>No cancer detected |
| Hoefman et al,[24] 2007; Hoefman et al,[34] 2005 (1999) | The Netherlands Primary care | 127/117 | 50 | 26 | Palpitations | Continuous event recorder vs usual care | Anxiety | 1.5-6 mo | New episodes or lightheadedness<br>Relevant cardiac Dx in 22% of both groups |
| Howard et al,[31] 2005 (1999) | UK Secondary care | 76/74 | 38 | 78 | Chronic daily headache | Cranial MRI vs not offered scan | Illness concern<br>Use of health care | 3-12 mo | Of 33 controls with HADS, 11 underwent MRI<br>No serious disease detected |
| Kendrick et al,[25,35] 2001 (1995) | UK Primary care | 210/211 | 39 | 41 | Back pain | Radiography vs usual care | Illness concern<br>Symptoms<br>Use of health care | 1.5-2 mo | Recruited at first visit for back pain |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 (1996) | UK Primary care | 73/80 | 44 | 50 | New-onset back pain | Radiography vs no radiography | Anxiety<br>Symptoms<br>Use of health care | 1.5-12 mo | Recruited at first visit for back pain |
| Laheij et al,[27] 1998 (1995) | The Netherlands Primary care | 42/42 | 43 | 51 | Dyspepsia | Endoscopy vs empirical treatment | Use of health care<br>Symptoms | 2.5-12 mo | 13 Controls underwent endoscopy<br>3 Cancers detected in 51 endoscopies |
| Sox et al,[32] 1981 (1978) | US Secondary care | 93/93 | Not stated | Not stated | Chest pain | ECG and blood tests vs no tests | Illness concern<br>Symptoms<br>Use of health care | 3 wk to 4 mo | Open-access clinic, recent chest pain and low probability of cardiac disease<br>5 Controls underwent ECG |

Abbreviations: Dx, diagnosis; ECG, electrocardiography; GERD, gastroesophageal reflux disease; HADS, Hospital Anxiety and Depression Scale; MRI, magnetic resonance imaging; RCT, randomized controlled trial; UK, United Kingdom; US, United States.

[a] The year of first recruitment is reported in parentheses. If only the year of publication is given, the dates of recruitment were not reported.

[b] Includes patient characteristics, diagnostic testing rate in controls, and serious illness detection rates where available.

No trials blinded the patients or their clinicians to the intervention, and only 1 trial reported adequate blinding of the outcome assessor at follow-up.[31] Most (but not all) patients randomized to diagnostic tests received them. Most trials included the option for patients allocated to no testing to receive subsequent diagnostic testing. The rates of later testing in patients randomized to nontesting varied, from 10% to 66% for endoscopy to 1.5% to 13% for other diagnostic tests. Three trials did not provide clear information about subsequent testing.[20,24,29]

Attrition rates varied between trials and with the duration of follow-up. Short-term outcome data were available for 75% to 100% of randomized patients; longer-term data were available for 71% to 100%. Few trials used statistical techniques to adjust for the effects of loss to follow-up.[22,24,31]

## ILLNESS CONCERN

Three trials examined illness concern in relation to magnetic resonance imaging for patients with headache,[31] lumbar spine radiography for patients with back pain,[25] and blood tests and electrocardiography in patients with chest pain.[32] Heterogeneity between studies was low ($I^2$ = 0%). Investigation was associated with no significant reduction in illness concern in the short (odds ratio, 0.90 [95% CI, 0.51-1.59]) or in the longer term (0.87 [0.55-1.39]), as shown in **Figure 3**.

## ANXIETY

Only 2 trials examined nonspecific or generalized anxiety: one in relation to lumbar radiography for patients with back pain[26] and the other in relation to the use of a continuous cardiac event recorder for patients with palpitations.[24] Heterogeneity between studies was low ($I^2$ = 0%). We observed a statistically insignificant increase in anxiety at longer-term follow-up in patients who underwent investigation, as shown in **Figure 4**.

## SYMPTOMS

Eleven trials reported the original symptoms after 1 or more follow-up periods.[19,21-23,25-30,32] Heterogeneity between studies was high in the short term ($I^2$ = 67%) and low in the longer term ($I^2$ = 0%). Meta-analysis (**Figure 5**) indicated no effect of diagnostic testing on symptoms in the longer term (odds ratio, 0.99 [95% CI, 0.85-1.15]).

**Table 2. Individual Trial Outcome Measures and Summary Results**

| Source | Trial Type | Measure | Diagnostic Test Group Value | Diagnostic Test Group Total No. of Patients | Control Group Value | Control Group Total No. of Patients | SMD (95% CI) | OR (95% CI)[a] |
|---|---|---|---|---|---|---|---|---|
| **Illness Concern >3 mo** | | | | | | | | |
| Howard et al,[31] 2005 | C | VAS, mean (SD) | −21 (49) | 54 | −17 (49) | 42 | −0.08 (−0.48 to 0.32) | 0.86 (0.42 to 1.79) |
| Kendrick et al,[25,35] 2001 | D | Statement agreement, No. of patients | 17 | 40 | 17 | 44 | | 117 (0.49 to 2.81) |
| Sox et al,[32] 1981 | D | Statement agreement, No. of patients | 12 | 74 | 16 | 72 | | 0.68 (0.30 to 1.56) |
| **Illness Concern ≤3 mo** | | | | | | | | |
| Kendrick et al,[25,35] 2001 | D | Statement agreement, No. of patients | 39 | 104 | 26 | 58 | | 0.74 (0.38 to 1.42) |
| Sox et al,[32] 1981 | D | Statement agreement, No. of patients | 17 | 84 | 17 | 87 | | 1.04 (0.49 to 2.21) |
| **Anxiety >3 mo** | | | | | | | | |
| Hoefman et al,[24] 2007; Hoefman et al,[34] 2005 | C | STAI score, mean (SD)[b] | −1.1 (9.94) | 103 | −4.0 (10.1) | 91 | 0.29 (0.01 to 0.57) | |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 | C | HADS score, mean (SD) | −1.1 (5.5) | 50 | −1.5 (5.5) | 58 | 0.07 (−0.31 to 0.45) | |
| **Anxiety ≤3 mo** | | | | | | | | |
| Hoefman et al,[24] 2007; Hoefman et al,[34] 2005 | C | STAI score, mean (SD)[b] | −1.6 (8.3) | 108 | −2.6 (8.3) | 82 | 0.12 (−0.17 to 0.41) | |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 | C | HADS score, mean (SD) | −0.6 (4.2) | 59 | −0.5 (4.2) | 67 | −0.02 (−0.37 to 0.33) | |
| **Symptoms >3 mo** | | | | | | | | |
| Bytzer et al,[19] 1994[c] | C | 3-Point Likert scale, mean (SD) | −20 (22.6) | 187 | −20 (23.2) | 186 | 0.01 (−0.19 to 0.21) | *1.02 (0.71 to 1.46)* |
| Delaney et al,[21] 2000 | C | BDSS, mean (SD) | −4.7 (5.0) | 190 | −3.5 (5.0) | 135 | −0.24 (−0.46 to −0.02) | *1.36 (0.91 to 2.03)* |
| Delaney et al,[22] 2001 | C | BDSS, mean (SD) | −3.8 (4.8) | 183 | −3.5 (4.5) | 107 | −0.06 (−0.30 to 0.17) | *0.90 (0.59 to 1.37)* |
| Giannini et al,[30] 2008[c] | C | 4-Point Likert scale, mean (SD) | −7 (7.6) | 209 | −7.7 (7.6) | 222 | 0.09 (−0.10 to 0.28) | *1.24 (0.88 to 1.75)* |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 | C | SF-36 bodily pain score, mean (SD) | −5.7 (5.57) | 50 | −6.6 (5.31) | 58 | 0.16 (−0.21 to 0.54) | 1.11 (0.56 to 2.22) |
| Asante et al,[28] 1998; Asante et al,[33] 1999 | D | Still has symptom, No. of patients | 34 | 60 | 30 | 57 | | 118 (0.57 to 2.44) |
| Bytzer et al,[19] 1994 | D | Still has symptom, No. of patients | 50 | 187 | 50 | 186 | | 0.99 (0.63 to 1.57) |
| Duggan et al,[23] 2009 | D | Still has symptom, No. of patients | 64 | 143 | 69 | 137 | | 0.80 (0.50 to 1.28) |
| Giannini et al,[30] 2008 | D | Still has symptom, No. of patients | 55 | 262 | 46 | 268 | | 1.28 (0.83 to 1.98) |
| Kendrick et al,[25,35] 2001 | D | Still has symptom, No. of patients | 126 | 195 | 133 | 199 | | 0.91 (0.60 to 1.37) |
| Laheij et al,[27] 1998 | D | No. of days without treatment | 96 | 255 | 100 | 266 | | 1.00 (0.70 to 1.43) |
| Sox et al,[32] 1981 | D | Still has symptom, No. of patients | 14 | 74 | 14 | 72 | | 0.97 (0.42 to 2.20) |
| **Symptoms ≤3 mo** | | | | | | | | |
| Djais and Kalim,[29] 2005[c] | C | VAS, mean (SD) | −43 (5.44) | 59 | −4 (5.92) | 67 | −0.05 (−0.04 to 0.30) | *1.82 (0.80 to 4.12)* |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 | C | SF-36 bodily pain score, No. of events | −2 (2.56) | 38 | −3 (2.08) | 38 | 0.42 (−0.03 to 0.88) | *0.91 (0.48 to 1.72)* |
| Djais and Kalim,[29] 2005 | D | | 10 | 36 | 7 | 33 | | 1.43 (0.47 to 4.33) |
| Duggan et al,[23] 2009 | D | Still has symptom, No. of patients | 40 | 154 | 61 | 137 | | 0.44 (0.27 to 0.72) |
| Giannini et al,[30] 2008 | D | Still has symptom, No. of patients | 30 | 395 | 31 | 298 | | 0.98 (0.57 to 1.66) |
| Kendrick et al,[25,35] 2001 | D | Still has symptom, No. of patients | 148 | 199 | 132 | 203 | | 1.56 (1.02 to 2.40) |
| Sox et al,[32] 1981 | D | Still has symptom, No. of patients | 19 | 84 | 23 | 87 | | 0.81 (0.40 to 1.64) |

*(continued)*

**Table 2. Individual Trial Outcome Measures and Summary Results (continued)**

| Source | Trial Type | Measure | Diagnostic Test Group Value | Diagnostic Test Group Total No. of Patients | Control Group Value | Control Group Total No. of Patients | SMD (95% CI) | OR (95% CI)[a] |
|---|---|---|---|---|---|---|---|---|
| | | | **Use of Health Care Resources** | | | | | |
| Asante et al,[28] 1998; Asante et al,[33] 1999 | C | Case notes review, mean (SD) | −0.8 (3.5) | 78 | −1.2 (4.4) | 76 | 0.10 (−0.22 to 0.42) | *1.20 (0.67 to 2.14)* |
| Cuddihy et al,[20] 2005 | C | Case notes review, mean (SD) | 238 (139) | 13 | 133 (79) | 11 | 0.88 (0.03 to 1.72) | *4.93 (1.07 to 22.85)* |
| Delaney et al,[21] 2000 | C | Case notes review, mean (SD) | 3.46 (2.61) | 254 | 3.95 (3.36) | 184 | −0.17 (−0.36 to 0.02) | *0.73 (0.52 to 1.04)* |
| Delaney et al,[22] 2001 | C | Case notes review, mean (SD) | 3.26 (2.73) | 284 | 3.30 (2.67) | 191 | −001 (−0.20 to 0.17) | *0.98 (0.70 to 1.37)* |
| Howard et al,[31] 2005 | C | Case notes review, mean (SD) | 124.8 (86.4) | 76 | 148.9 (132.4) | 74 | −0.22 (−0.54 to 0.11) | *1.35 (0.45 to 4.08)* |
| Kerry et al,[26] 2002; Kerry et al,[36] 2000 | C | Case notes review, mean (SD) | 1.0 (1.6) | 50 | 1.6 (2.1) | 58 | −0.32 (−0.70 to 0.06) | *0.56 (0.28 to 1.11)* |
| Laheij et al,[27] 1998 | C | Case notes review, mean (SD) | 3.0 (2.4) | 38 | 3.9 (2.5) | 42 | −0.36 (−0.81 to 0.08) | *0.52 (0.23 to 1.17)* |
| Bytzer et al,[19] 1994 | D | Self-reported, No. of events | 47 | 187 | 114 | 186 | | 0.21 (0.14 to 0.33) |
| Duggan et al,[23] 2009 | D | Case notes review, No. of events | 80 | 186 | 108 | 177 | | 0.48 (0.32 to 0.73) |
| Kendrick et al,[25,35] 2001 | D | Self-reported, No. of events | 42 | 195 | 47 | 199 | | 0.89 (0.55 to 1.42) |
| Sox et al,[32] 1981 | D | Questionnaire, No. of events | 10 | 84 | 13 | 87 | | 0.77 (0.32 to 1.86) |

Abbreviations: BDSS, Birmingham Dyspepsia Symptoms Score; C, continuous measure; D, dichotomous; HADS, Hospital Anxiety and Depression Scale; OR, odds ratio; SF-36, 36-Item Short Form Health Survey; SMD, standardized mean difference; STAI, State-Trait Anxiety Inventory; VAS, visual analog scale.

[a] Values in italics refer to estimated ORs converted from SMD for use in meta-analysis combining continuous and dichotomous variables.

[b] Only the state anxiety component was used in the analysis.

[c] Continuous data are shown for completeness; the comparison uses the OR from dichotomous data, which was also reported.

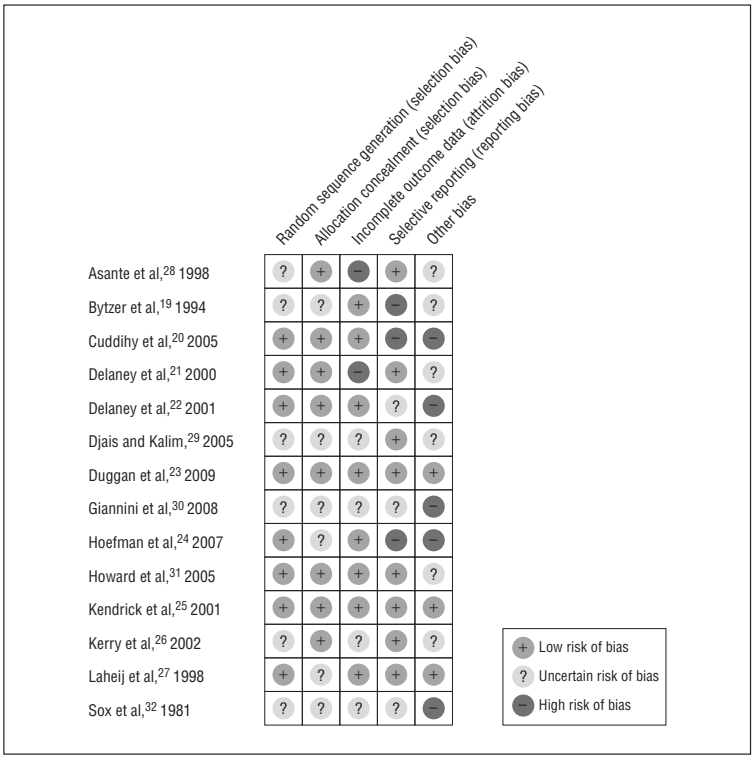



**Figure 2.** Risk of bias summary.

## SUBSEQUENT PRIMARY CARE VISITS

Eleven trials examined primary care visit rates after the intervention.[19-23,25-28,31,32] Eight trials used case note reviews[20-23,26-28,31] and 3 used patient self-report.[19,25,32] Individual and overall effects are shown in **Figure 6**, which indicates marked heterogeneity between trials ($I^2$ = 80%). Most of this heterogeneity arose from 2 trials: one with a very small number of participants[20] and the other an older trial that predated *Helicobacter pylori* eradication for peptic ulcer disease.[19] When these 2 trials were excluded, heterogeneity was reduced ($I^2$ = 33%), and the overall effect was a reduction in subsequent visit rates of 0.77 (0.62-0.96). The rates of repeated visits for control patients were 60% in dyspepsia trials, with a number needed to investigate of 16 (95% CI, 8-100), and 20% in back pain trials, with a number needed to investigate of 26 (95% CI, 15-155).

## MAIN FINDINGS

This systematic review indicates that patients' illness concern, health anxiety, and symptoms are not reduced by diagnostic testing in the short or the long term. Subsequent use of health care resources may be reduced by diagnostic testing, although the number of patients needed to investigate and avoid 1 subsequent visit varied from 16 to 26 depending on the symptom. In the context of widespread belief that diagnostic testing reassures patients, these findings suggest that physicians overestimate the value of testing when the probability of serious disease is low.

## STRENGTHS AND LIMITATIONS

Although previous systematic reviews have used a narrow definition of reassurance[13] or have been limited to 1 specific clinical problem,[37,38] we included a broader assessment of reassurance by including the reduction of illness concern and the expected consequences of reassurance; we also included a wide range of clinical problems. This approach risks comparing trials that are too dissimilar and for which meta-analysis may be inappropriate; however, we took the view that models of symptom appraisal and reassurance are consistent across contexts[10-12] and that all trials centered on the decision to perform diagnostic testing or not. Substantial heterogeneity was seen only for the use of health care resources and, because the number of trials was small, we did not perform a formal subgroup comparison. Outcome measures varied from the well validated (such as the 36-Item Short Form Health Survey) to the ad hoc; the poorly validated measures might have been insufficiently sensitive to change.

The studies in this review were conducted in different places and times during which the practice of medicine and the expectations of patients changed. The study that showed the greatest influence of diagnostic testing on reassurance[19] was for peptic ulcer disease in the 1980s, when surgical treatment was common and before the recognition of *H pylori*. Most of the eligible studies were conducted in European health care systems, where access to diagnostic testing may be more constrained than in US health care. We examined reassurance for patients only; we did not examine the reassurance (including the reassurance that they were less likely to be sued) that diagnostic tests provided for physicians. A health economic analysis was beyond the scope of this review; however, because the cost of a primary care consultation is less than the cost of most diagnostic tests and because several tests were required to avert 1 consultation, the balance would not favor testing.

We did not examine differential effects of anxiety at baseline on subsequent reassurance. One study reported a prespecified comparison between more and less anxious patients and found that illness concern was reduced more by diagnostic testing in patients with high anxiety levels, but these data were not available from other trials.[31] Although our analysis highlights the limited value of diagnostic testing in terms of reassurance, it does not address the wider role of investigations in identifying disease or allowing the physician to rule out a particular differential diagnosis. Although the prevalence of serious disease such as cancer in the eligible trials varied from less than 0.5% to 3%, our findings do not address what pretest probability of disease constitutes an appropriate threshold for investigation to obtain a diagnosis. In addition, our find-

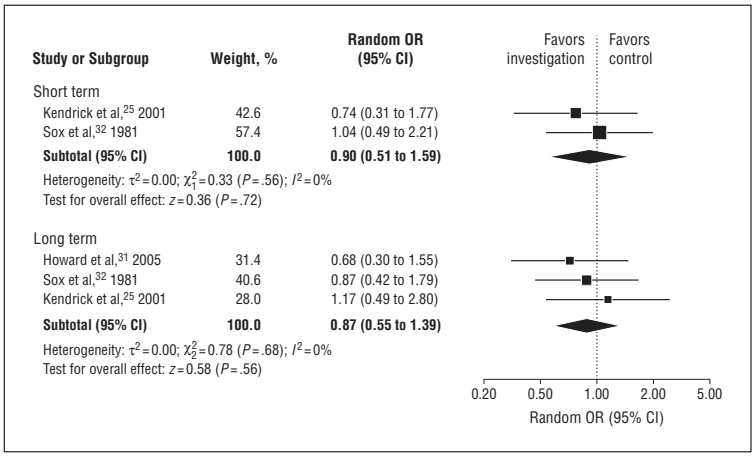



**Figure 3.** Effect of diagnostic testing on reduction of illness concern. The size of the data marker corresponds to the relative weight assigned in the pooled analysis using random-effects models. OR indicates odds ratio.

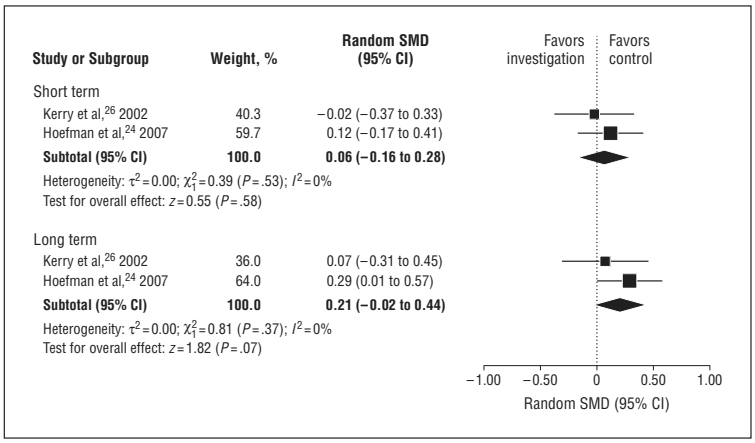



**Figure 4.** Effect of diagnostic testing on anxiety. The size of the data marker corresponds to the relative weight assigned in the pooled analysis using random-effects models. SMD indicates standardized mean difference.

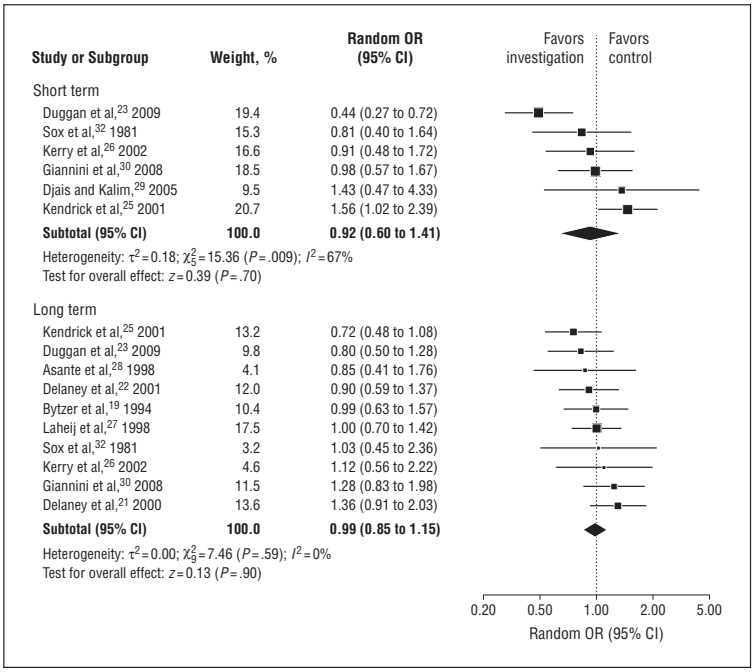



**Figure 5.** Effect of diagnostic testing on presenting symptoms. The size of the data marker corresponds to the relative weight assigned in the pooled analysis using random-effects models. OR indicates odds ratio.

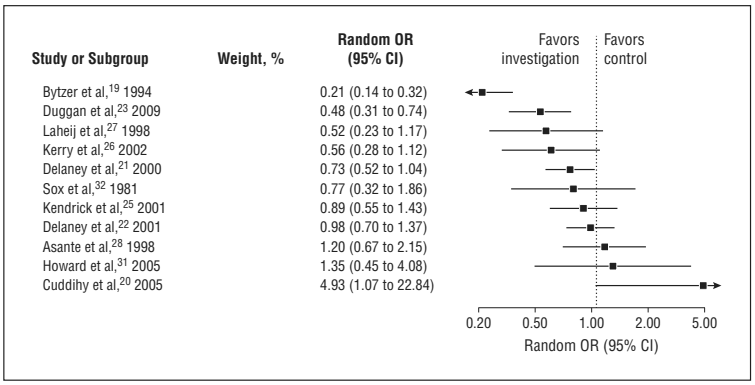



**Figure 6.** Effect of diagnostic testing on primary care visits. OR indicates odds ratio.

ings do not contradict guidelines for rational requesting of tests that balance benefits against harms.

We did not find studies of complex or chronic symptoms. However, persistent symptoms with negative test results are associated with frustration and dissatisfaction.[39]

## INTERPRETATION

We found the use of diagnostic testing did little to reassure patients, and this finding is inconsistent with beliefs expressed by physicians.[3,7] One explanation is that the reassurance obtained by patients from negative diagnostic test results is transient. Observational studies suggest that illness concerns reappear within hours of receiving a normal (negative) test result,[40,41] whereas the trials in our review measured effects after weeks or months. The mechanism of transient reassurance appears to be predominantly emotional—a fleeting sense of relief—in contrast to a more sustained cognitive reassurance.[10] One trial included in the review attempted to place a value on reassurance and found that patients were willing to pay for the reassurance of normal findings on spine radiography, although no discernible effect was observed on measures of concern.[25] Thus, patients and physicians may value the immediate relief of reassurance, although the benefits are not sustained. We found a small reduction in subsequent primary care visits after diagnostic testing, but this reduction required several patients to undergo testing to prevent 1 visit.

## RECOMMENDATIONS FOR RESEARCH AND PRACTICE

Because the number of trials in this review is relatively small, further trials may demonstrate an effect of diagnostic testing on reassurance among patients with symptoms indicating a low probability of disease. However, the small effect sizes are in keeping with the postulated psychological and behavioral mechanisms underlying symptom appraisal[11] and reassurance.[10,12] Thus, concentration of future research on the following 2 questions may be more important: (1) how to maximize the reassurance value of diagnostic tests and (2) whether reassurance should be targeted to particular patients. Three trials have reported that brief interventions to increase the acceptability of negative test results lead to improved reassurance,[42-44] and theoretical work supports this finding.[45] Targeting interventions (including reassurance and cognitive-based rehabilitation) to patients at higher risk of persistent symptoms is effective in patients with low back pain,[46] and diagnostic testing accompanied by enhanced explanation can be an appropriate strategy. Meanwhile, physicians and health care organizations should be aware of the limitations of the transient reassurance provided by negative diagnostic test results and should limit tests to those that influence clinical management. In summary, commonly used diagnostic tests have little effect on several aspects of reassurance in patients whose symptoms indicate a low pretest probability of serious illness.

**Accepted for Publication:** October 28, 2012.
**Published Online:** February 25, 2013. doi:10.1001/jamainternmed.2013.2762

**Correspondence:** Christopher Burton, MD, Centre of Academic Primary Care, University of Aberdeen, Polwarth Building, Foresterhill, Aberdeen AB25 2ZD, Scotland (c.burton@abdn.ac.uk).

**Author Contributions:** Drs Rolfe and Burton are joint guarantors of this study. *Study concept and design:* Rolfe and Burton. *Acquisition of data:* Rolfe and Burton. *Analysis and interpretation of data:* Rolfe and Burton. *Drafting of the manuscript:* Rolfe and Burton. *Critical revision of the manuscript for important intellectual content:* Burton. *Statistical analysis:* Rolfe and Burton. *Administrative, technical, and material support:* Rolfe.

**Conflict of Interest Disclosures:** None reported.

**Funding/Support:** Dr Burton was supported by a Research Career Award from the Chief Scientist Office of the Scottish Government.

**Previous Presentation:** This study was presented at the Society for Academic Primary Care Annual Scientific Meeting; July 6, 2011; Bristol, England.

**Online-Only Material:** The eAppendix and eMethods are available at http://www.jamainternalmed.com.

**Additional Contributions:** Fay Crawford, PhD, and Kurt Kroenke, MD, provided comments on earlier versions of this manuscript.

## REFERENCES


1. Burton C. Beyond somatisation: a review of the understanding and treatment of medically unexplained physical symptoms (MUPS). *Br J Gen Pract.* 2003;53(488):231-239.
2. Hatcher S, Arroll B. Assessment and management of medically unexplained symptoms. *BMJ.* 2008;336(7653):1124-1128.
3. Van der Weijden T, Van Velson M, Dinant GJ, Van Hasselt CM, Grol R. Unexplained complaints in general practice: prevalence, patients' expectations and professionals' test-ordering behaviour. *Med Decis Making.* 2003;23:226-231.
4. Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Arch Gen Psychiatry.* 2005;62(8):903-910.
5. Salmon P, Humphris GM, Ring A, Davies JC, Dowrick CF. Primary care consultations about medically unexplained symptoms: patient presentations and doctor responses that influence the probability of somatic intervention. *Psychosom Med.* 2007;69(6):571-577.
6. Royal College of Radiologists. *Making the Best Use of Clinical Radiology Services (MBUR).* 6th ed. London, England: Royal College of Radiologists; 2007.
7. Ring A, Dowrick C, Humphris G, Salmon P. Do patients with unexplained physical symptoms pressurise general practitioners for somatic treatment? a qualitative study. *BMJ.* 2004;328(7447):157. doi:10.1136/bmj.38057.622639.EE.
8. Salmon P, Ring A, Dowrick CF, Humphris GM. What do general practice patients want when they present medically unexplained symptoms, and why do their doctors feel pressurized? *J Psychosom Res.* 2005;59(4):255-262.
9. Little P, Dorward M, Warner G, Stephens K, Senior J, Moore M. Importance of patient pressure and perceived pressure and perceived medical need for investigations, referral, and prescribing in primary care: nested observational study. *BMJ.* 2004;328(7437):444-446.
10. Coia P, Morley S. Medical reassurance and patients' responses. *J Psychosom Res.* 1998;45(5):377-386.
11. Leventhal H, Meyer D, Nerenz D. *The Commonsense Representation of Illness Danger.* Oxford, England: Pergamon; 1980.
12. Rief W, Broadbent E. Explaining medically unexplained symptoms-models and mechanisms. *Clin Psychol Rev.* 2007;27(7):821-841.
13. van Ravesteijn H, van Dijk I, Darmon D, et al. The reassuring value of diagnostic tests: a systematic review. *Patient Educ Couns.* 2012;86(1):3-8.
14. Centre for Reviews and Dissemination. *Systematic Review: CRD's Guidance for Undertaking Reviews in Health Care.* York, England: Centre for Reviews and Dissemination, University of York; 2009.
15. Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: explanation and elaboration. *BMJ.* 2009;339:b2700. doi:10.1136/bmj.b2700.
16. Gilbert FJ, Grant AM, Gillan MGC, et al. Low back pain: influence of early MR imaging or CT on treatment and outcome: multicentre randomized trial. *Radiology.* 2004;231(2):343-351.
17. *Review Manager (RevMan), Version 5.1* [computer program]. København, Denmark: Nordic Cochrane Centre; 2011.
18. Higgins J, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions, Version 5.1.0.* The Cochrane Collaboration website. http://www.cochrane-handbook.org. Updated March 2011. Accessed January 18, 2013.
19. Bytzer P, Hansen JM, Schaffalitzky de Muckadell OB. Empirical $H_2$-blocker therapy or prompt endoscopy in management of dyspepsia. *Lancet.* 1994;343(8901):811-816.
20. Cuddihy MT, Locke GR III, Wahner-Roedler D, et al. Dyspepsia management in primary care: a management trial. *Int J Clin Pract.* 2005;59(2):194-201.
21. Delaney BC, Wilson S, Roalfe A, et al. Cost effectiveness of initial endoscopy for dyspepsia in patients over age 50 years: a randomised controlled trial in primary care. *Lancet.* 2000;356(9246):1965-1969.
22. Delaney BC, Wilson S, Roalfe A, et al. Randomised controlled trial of *Helicobacter pylori* testing and endoscopy for dyspepsia in primary care. *BMJ.* 2001;322(7291):898-901.
23. Duggan AE, Elliot CA, Miller P, Hawkey CJ, Logan RF. A clinical trial: a randomized trial of early endoscopy, *Helicobacter pylori* testing and empirical therapy for the management of dyspepsia in primary care. *Aliment Pharmacol Ther.* 2009;29(1):55-68.
24. Hoefman E, Boer KR, van Weert HCPM, Reitsma JB, Koster RW, Bindels PJP. Continuous event recorders did not affect anxiety or quality of life in patients with palpitations. *J Clin Epidemiol.* 2007;60(10):1060-1066.
25. Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. *BMJ.* 2001;322(7283):400-405.
26. Kerry S, Hilton S, Dundas D, Rink E, Oakeshott P. Radiography for low back pain: a randomised controlled trial and observational study in primary care. *Br J Gen Pract.* 2002;52(479):469-474.
27. Laheij RJF, Severens JL, Van de Lisdonk EH, Verbeek ALM, Jansen JBMJ. Randomized controlled trial of omeprazole or endoscopy in patients with persistent dyspepsia: a cost-effectiveness analysis. *Aliment Pharmacol Ther.* 1998;12(12):1249-1256.
28. Asante MA, Mendall M, Patel P, Ballam L, Northfield TC. A randomized trial of endoscopy vs no endoscopy in the management of seronegative *Helicobacter pylori* dyspepsia. *Eur J Gastroenterol Hepatol.* 1998;10(12):983-989.
29. Djais N, Kalim H. The role of lumbar spine radiography in the outcomes of patients with simple acute low back pain. *APLAR J Rheumatol.* 2005;8(1):45-50.
30. Giannini EG, Zentilin P, Dulbecco P, Vigneri S, Scarlata P, Saverino V. Management strategy for patients with gastroesophageal reflux disease: a comparison between empirical treatment with esomeprazole and endoscopy-orientated treatment. *Am J Gastroenterol.* 2008;103(2):267-275.
31. Howard L, Wessely S, Leese M, et al. Are investigations anxiolytic or anxiogenic? a randomised controlled trial of neuroimaging to provide reassurance in chronic daily headache. *J Neurol Neurosurg Psychiatry.* 2005;76(11):1558-1564.
32. Sox HC Jr, Margulies I, Sox CH. Psychologically mediated effects of diagnostic tests. *Ann Intern Med.* 1981;95(6):680-685.
33. Asante MA, Lord J, Mendall M, Northfield T. Endoscopy for *Helicobacter pylori* sero-negative young dyspeptic patients: an economic evaluation based on a randomized trial. *Eur J Gastroenterol Hepatol.* 1999;11(8):851-856.
34. Hoefman E, van Weert HC, Reitsma JB, Koster RW, Bindels PJ. Diagnostic yield of patient-activated loop recorders for detecting heart rhythm abnormalities in general practice: a randomised clinical trial. *Fam Pract.* 2005;22(5):478-484.
35. Kendrick D, Fielding K, Bentley E, Miller P, Kerslake R, Pringle M. The role of radiography in primary care patients with low back pain of at least 6 weeks duration: a randomised (unblinded) controlled trial. *Health Technol Assess.* 2001;5(30):1-69.
36. Kerry S, Hilton S, Patel S, Dundas D, Rink E, Lord J. Routine referral for radiography of patients presenting with low back pain: is patients' outcome influenced by GPs' referral for plain radiography? *Health Technol Assess.* 2000;4(20):i-iv, 1-119.
37. Chou R, Fu R, Carrino JA, Deyo RA. Imaging strategies for low-back pain: systematic review and meta-analysis. *Lancet.* 2009;373(9662):463-472.
38. Delaney B, Ford AC, Forman D, Moayeddi P, Qume M. Initial management strategies for dyspepsia. *Cochrane Database Syst Rev.* 2005;19(4):CD001961.
39. Speckens AEM, Spinhoven P, Van Hemert AM, Bolk JH, Bolk JH. The Reassurance Questionnaire (RQ): psychometric properties of a self-report questionnaire to assess reassurability. *Psychol Med.* 2000;30(4):841-847.
40. Lucock MP, Morley S, White C, Peake MD. Responses of consecutive patients to reassurance after gastroscopy: results of self administered questionnaire survey. *BMJ.* 1997;315(7108):572-575.
41. Wiklund I, Glise H, Jerndal P, Carlsson J, Talley

NJ. Does endoscopy have a positive impact on quality of life in dyspepsia? *Gastrointest Endosc.* 1998;47(6):449-454.
42. Arnold J, Goodacre S, Bath P, Price J. Information sheets for patients with acute chest pain: randomised controlled trial. *BMJ.* 2009;338:b541. doi:10.1136/bmj.b541.
43. Petrie KJ, Müller JT, Schirmbeck F, et al. Effect of providing information about normal test results on patients' reassurance: randomised controlled trial. *BMJ.* 2007;334(7589):352 doi:http://dx.doi.org/10.1136/bmj.39093.464190.55.
44. Serinken M, Zencir M, Karcioglu O, Sener S, Turkcuer I. Value of the timing of informing the emergency department patients on cardiac test results: a randomized controlled study. *Eur J Emerg Med.* 2009;16(2):74-79.
45. Salmon P. Conflict, collusion or collaboration in consultations about medically unexplained symptoms: the need for a curriculum of medical explanation. *Patient Educ Couns.* 2007;67(3):246-254.
46. Hill JC, Whitehurst DG, Lewis M, et al. Comparison of stratified primary care management for low back pain with current best practice (STarT Back): a randomised controlled trial. *Lancet.* 2011;378(9802):1560-1571.

INVITED COMMENTARY

# Diagnostic Testing and the Illusory Reassurance of Normal Results

Diagnostic testing is enticing to patients and clinicians. It appears more objective and less pedestrian than a simple clinical interview and physical examination. Medical certainty is seldom solidified until all the tests results are in. Patients anxiously await the telephone call or letter announcing "your tests are all normal." Indeed, the grander the technology, the more alluring. However, the testing imperative can become addictive. As noted in a 1991 cautionary essay:

> Technology pounds upon the shore, but the danger is the undertow. The effacement of sand castles we abide; the relentless tug is another matter, sucking us deeper. Like systole and diastole, there is faint pause, endless indications. Imaging fits the metaphor, wave after wave: radionuclide scanning, computerized tomography, magnetic resonance imaging, duplex sonography. The very names captivate our diagnostic instincts, and yet excess appears inevitable. . . . Endoscopy is equally irrepressible. . . . To witness a cause transcends the more banal concerns of costs and therapeutic outcome. Follow-up is inconvenient. To wait and see whether growing suspicions will justify exploration or whether signs and symptoms prove merely transitory cannot compete with immediate visualization.[1]

Like many of our treatments, however, diagnostic testing is not without its adverse effects. Increased health care costs are the most obvious: wide geographic variations in the use of expensive tests persist more than 30 years after such inexplicable variation was first exposed.[2] Still more insidious consequences lurk. One is the problem of false-positive results. The prevalence of detecting a serious condition may be as low as 0.5% to 3.0% when diagnostic tests are ordered in patients with a low probability of disease,[3] meaning that a diagnostic test with a 90% sensitivity and 90% specificity would yield 4 to 19 false-positive results for every true-positive result in patients for whom the test is ordered simply to rule out a disease for which the clinical suspicion is already low. This disproportionately high false-positive rate may then cascade into additional and sometimes invasive procedures, not to mention considerable patient anxiety that may persist months after a negative finding of a workup cancels out the initial test results. One might consider this PTSD (posttest stress disorder) an iatrogenic variant of the traditional PTSD (posttraumatic stress disorder).

False-negative results can also be a concern. For example, the high diagnostic accuracy of abdominal computed tomography for appendicitis and renal colic does not generalize to patients presenting to the emergency department with undifferentiated upper abdominal pain, where the negative predictive value is only 64%.[4] That means as many as 1 of 3 normal abdominal computed tomographic scans in this population may represent a false-negative test result, with the most commonly missed pathologic changes being inflammatory conditions of the biliary tract and upper gastrointestinal tract systems.

In addition, reflexive test ordering may marginalize the clinical examination. Preliminary data suggest that that the history typically accounts for 75% or more of the diagnostic yield when evaluating common symptoms; the physical examination, 10% to 15%; and testing, generally less than 10%.[5] Ironically, the US reimbursement system financially incentivizes these components in the reverse order. Diagnostic testing and procedures receive the highest remuneration and, even within the clinical encounter, evaluation and management coding favors from a billing standpoint the physical examination of more bodily parts (even if irrelevant to the presenting complaint) over a detailed and more diagnostically informative interview.

Despite these limitations of diagnostic testing in patients with a low probability of disease, a conventional justification is reassurance of the patient. However, the meta-analysis by Rolfe and Burton[3] suggests that even this benefit may be overestimated. The authors included only trials in which patients with a low probability of disease were randomized to receive initial diagnostic testing vs a nontesting approach. The patient sample is appropriate because most would agree that diagnostic testing is warranted to rule in or confirm a suspected disease, determine its extent or sever-

4. Hall-Patch L, Brown R, House A, Howlett S, Kemp S, Lawton G, et al. Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures. Epilepsia 2010; 51(1): 70-8.

*Epilepsia*, 51(1):70–78, 2010
doi: 10.1111/j.1528-1167.2009.02099.x

FULL-LENGTH ORIGINAL RESEARCH

# Acceptability and effectiveness of a strategy for the communication of the diagnosis of psychogenic nonepileptic seizures

***Lindsey Hall-Patch, †Richard Brown, *Allan House, ‡Stephanie Howlett, §Steven Kemp, ‡Gemma Lawton, ‡Rebecca Mayor, ¶Phil Smith, and ‡Markus Reuber for the NEST collaborators[1]**

***Academic Unit of Psychiatry and Behavioural Sciences, University of Leeds, Leeds, United Kingdom; †Division of Clinical Psychology, University of Manchester, Manchester, United Kingdom; ‡Academic Neurology Unit, University of Sheffield, Royal Hallamshire Hospital, Sheffield, United Kingdom; §Department of Neuropsychology, Leeds Teaching Hospitals NHS Trust, St James's Hospital, Leeds, United Kingdom; and ¶Department of Neurology, University Hospital of Wales, Cardiff, United Kingdom**

## Summary

**Purpose: Communicating the diagnosis of psychogenic nonepileptic seizures (PNES) is a challenging task. This study was carried out to assess the acceptability and effectiveness of a new communication procedure consisting of a patient information leaflet and a communication strategy for neurologists.**

**Methods: In a multicenter prospective study, 50 patients newly diagnosed with PNES were informed about the diagnosis by 10 different neurologists using the communication procedure. Follow-up data were gathered by telephone interview and completion of a questionnaire about symptom attributions (psychological/physical) and illness cognitions (Illness Perception Questionnaire-Revised, IPQ-R).**

**Results: Ninety-four percent of patients found the leaflet easy to understand. Ninety-four percent stated their questions were answered by the doctor; 70% got what they wanted from the consultation; only 4% reported feeling angry during the consultation. Eighty-six percent of patients acknowledged that psychological factors were at least contributing to their seizures. On the IPQ-R, "emotional" causes for the seizures were endorsed more commonly than "nonemotional" causes ($p < 0.001$). After 3 months, 14% of patients were seizure-free and 63% reported a >50% reduction in seizure frequency.**

**Discussion: We conclude that our procedure is acceptable and effectively communicates a psychological etiologic model for PNES.**

**KEY WORDS: Psychogenic nonepileptic seizures, Diagnosis, Communication, Illness perceptions, Psychological treatment.**

Psychogenic nonepileptic seizures (PNES) are episodes of altered movement, sensation, or experience resembling epileptic seizures but not associated with epileptic discharges in the brain. They can be defined positively as episodes of loss of control that occur in response to distressing situations, sensations, emotions, conflicts, or memories when alternative coping mechanisms are inadequate or have been overwhelmed (Reuber, 2008). PNES are classified as a manifestation of dissociative or conversion disorder in the current nosologies (World Health Organization, 1992; American Psychiatric Association, 1994). Although many questions about their etiology remain unanswered, and there are presently no proven interventions for PNES, most neurologists consider psychological treatment the management of choice (LaFrance, Jr et al., 2008).

PNES are not uncommon. The population prevalence of PNES has been estimated as 2–33 per 100,000 (Benbadis et al., 2000). Two recent studies have shown that patients

Accepted February 10, 2009; Early View publication April 27, 2009.
Address correspondence to Markus Reuber, Academic Neurology Unit, University of Sheffield, Royal Hallamshire Hospital, Glossop Road, Sheffield, S10 2JF. U.K. E-mail: markus.reuber@sth.nhs.uk
[1]See Appendix.

Wiley Periodicals, Inc.

with PNES make up 12–18% of patients newly presenting with seizures (Kotsopoulos et al., 2003; Angus-Leppan, 2008). The same studies suggest that, in a first seizure clinic, physicians should expect to make one diagnosis of PNES for every three or four of epilepsy. However, most patients with PNES are initially misdiagnosed as having epilepsy. In fact, the diagnosis of PNES is typically made only after patients have been treated inappropriately with antiepileptic drugs for several years (Reuber et al., 2002; de Timary et al., 2002).

Once the diagnosis has been made, neurologists face the challenge of explaining to patients that they do not have epilepsy, but that their seizures are a manifestation of psychological distress. This can be difficult as patients may be reluctant to accept that their seizures do not have a ''physical'' cause. Although they report more symptoms of psychopathology and more negative life events (Prueter et al., 2002; Galimberti et al., 2003; Reuber et al., 2003a; Binzer et al., 2004; Lawton et al., 2008), as a group, patients with PNES are paradoxically less likely to agree that stress or emotional factors could be a cause of seizures than patients with epilepsy (Stone et al., 2004).

The question of how the diagnosis should be communicated has attracted considerable interest (Harden & Ferrando, 2001; Kanner, 2003). One reason that ''good'' communication is important at this point is that outcome is better in patients who accept the diagnosis (Ettinger et al., 1999). In fact, PNES can resolve when clinicians have explained the nature of the problem (Aboukasm et al., 1998; Kanner et al., 1999). What is more, effective communication at this point has the potential to change patients' healthcare utilization behavior significantly and to reduce healthcare costs (Martin et al., 1998).

At present, however, many patients report feeling confused or angry after receiving the diagnosis of PNES (Carton et al., 2003; Green et al., 2004; Thompson et al., 2005, 2009). Only three in five patients referred by a neurologist attend an assessment interview for psychological treatment (Howlett et al., 2007). ''Unsuccessful'' communication may also increase the risk of continuing inappropriate treatment with anticonvulsants. We have previously shown that 41% of patients diagnosed with PNES (and no additional epileptic seizures) were taking antiepileptic drugs a mean of 4 years after the diagnosis of PNES (Reuber et al., 2003b).

A protocol for a more satisfactory communication of the diagnosis of PNES has been proposed (Shen et al., 1990), but its effectiveness or acceptability has never been tested. A number of individuals and organizations have produced information leaflets for patients intended to help explain the diagnosis, but these have not been evaluated in clinical studies either (for examples see http://hsc.usf.edu/COM/epilepsy/PNESbrochure.pdf or http://www.epilepsyfoundation.org/about/types/types/nonepileptic/index.cfm).

The primary aim of the present study was to evaluate the acceptability and short-term effectiveness of a communication strategy consisting of a detailed patient information leaflet and doctors' guide encouraging clinicians to emphasize the core points made in the leaflet. The communication strategy was designed to address all domains of patients' illness representations. Illness representations are an essential part of the self-regulation model, which proposes that how people behave in relation to illness depends on their own perception or representation of the illness. In this model, illness representations consist of five elements: identity (symptoms or label), cause, consequences (effects on life or lifestyle), time line (time to develop and duration), and controllability or cure (Leventhal et al., 1992). A recent review concluded that there was a clear relationship between illness cognitions, coping behavior, and illness outcomes (Hagger & Orbell, 2003), and one study demonstrated that (with some minor modification) the self-regulation model was applicable to how patients think about PNES (Green et al., 2004).

This communication strategy was conceived as a first step toward the development of an evidence-based management pathway for patients with PNES. The secondary aim of this study was to explore the recruitment potential of the collaborating clinicians for future treatment studies and to describe the characteristics of the patients entering such studies.

## Methods

### Development of the communication strategy

Based on a review of the literature, available patient information leaflets, and their own clinical practice, the collaborators identified 14 core points addressing all major domains of patients' illness representations. A patient leaflet was compiled that covered and elaborated the 14 core points over 27 pages. The leaflet refers to PNES as ''nonepileptic attacks'' because this has become the most commonly used label in the United Kingdom. However, the authors consider the label of ''nonepileptic attacks'' as synonymous with the label PNES (which has been used more frequently in recent research publications) and terms such as ''pseudoseizures,'' ''dissociative seizures,'' or ''conversion seizures.'' The leaflet is available for download at http://www.shef.ac.uk/content/1/c6/08/82/45/NEST%20Patient%20Booklet.pdf. A doctor's guide was written to accompany the leaflet, offering further explanation of the reasons for including each point, references to relevant research, and advice on how to communicate the information to patients (http://www.shef.ac.uk/content/1/c6/08/82/45/NEST%201%20Clinician%20Handbook.doc). A crib sheet was also produced, and neurologists were encouraged to use this as a checklist to try to ensure they covered all the points (see Table 1).

*Epilepsia*, 51(1):70–78, 2010
doi: 10.1111/j.1528-1167.2009.02099.x

**Table 1. Crib sheet with 14 core points of the strategy for the communication of the diagnosis of PNES**

| |
|---|
| Genuine symptoms |
| *Real attacks*—can be frightening or disabling |
| Label |
| *Give a name* for the condition |
| *Give alternative names* they may hear |
| Reassure that this is a *common and recognized condition* |
| Cause and maintaining factors |
| *Not epilepsy* |
| Predisposing factors—*difficult to find out causes* |
| Precipitating factors—*can be related to stress/emotions* |
| Perpetuating factors—vicious cycle – *worry* → *stress* → *attacks* → *worry* |
| Provide a *model* for the attacks—e.g., brain becomes overloaded and shuts down |
| Treatment |
| *Antiepileptic drugs are not effective* |
| Evidence that *psychological treatment is effective* |
| Talk to the patient about *referral to a specialist* |
| Expectations |
| *Can resolve* |
| *Can expect improvement* |

PNES, psychogenic nonepileptic seizures.

### Collaborating clinicians

Fully trained neurologists with a clinical interest in seizure disorders from centers throughout the United Kingdom were invited to take part in the study. Each neurologist was encouraged to recruit all patients who met the criteria and who presented within a 6-months period. Recruitment began in the first center in October 2006 and finished in the last in June 2008.

### Patients

Patients received the patient leaflet at the point of receiving the diagnosis of PNES. During the same consultation they were asked whether they would mind finding out more about a research study evaluating the communication of the diagnosis. If they agreed to find out more about the project, their contact details were passed on to the research psychologist (LHP) who contacted them, offered information, and obtained written informed consent via mail. Patients were included if they had received the diagnosis of PNES from the neurologist for the first time and if the diagnosis was secure (confirmed by video-electroencephalography), or based on the direct observation of a seizure by an experienced healthcare professional (e.g., neurologist, experienced EEG technician, or specialist nurse). Patients with a history of possible epilepsy (but no epileptic seizures for at least 1 year) were eligible for participation. Patients were excluded if they did not speak English sufficiently to take part in a telephone interview or to complete the self-report questionnaires used in the study.

### Evaluating the communication strategy

Patients' recollections of their encounter with the neurologist were assessed in a semistructured telephone interview, which was conducted by the research psychologist after patients had returned a form documenting their written informed consent. This interview was conducted approximately 2 weeks after their consultation with the neurologist. The interview consisted of a series of cued questions. These were followed by closed (yes/no) questions if the patient had not given the expected answer to the initial question (e.g., "What kind of treatment did your doctor suggest for your attacks?" expected answer: "Psychological treatment." follow-up question: "Did your doctor suggest psychological treatment?"). For the full telephone questionnaire see http://www.shef.ac.uk/content/1/c6/08/82/45/NEST%201%20Telephone%20interview.doc. Patients were also asked to provide a self-report measure of their seizure frequency before they spoke to their neurologist about their diagnosis and at the time of the telephone interview. Responses were categorized into "daily" (one seizure or more per day), "weekly" (at least one seizure per week, but less than one per day), "monthly" (at least one seizure per month but less than one per week), "occasionally" (less than one seizure per month), and "seizure free."

Patients were sent a questionnaire immediately after this telephone interview, which was intended to reveal their thoughts about their disorder at a point at which they may start psychological treatment in future intervention studies. It included a forced choice question with five response options previously used to sample symptom attribution to physical or psychological causes in other patient groups with medically explained or unexplained physical symptoms (see Results section for wording) (Wessely & Powell, 1989). It also contained a version of the Illness Perception Questionnaire-Revised (IPQ-R), which had been adapted for seizures in line with the instructions of its authors (Moss-Morris et al., 2002). The IPQ-R has been shown to have high internal and external validity and good test–retest reliability. It provides a quantitative assessment of patients' illness perceptions in the five major domains listed previously and has been used in a wide range of disorders and clinical scenarios (Moss-Morris et al., 2002; Moss-Morris & Chalder, 2003; Gray & Rutter, 2007; Stockford et al., 2007). In line with a previous study (Goldstein et al., 2004), the list of possible causes of seizures included in the IPQ-R was split into seven "emotional" causes (stress or worry; my own behavior; my mental attitude; family problems or worries; overwork; my emotional state; my personality) and 11 "nonemotional" causes (hereditary; a germ or virus; diet or eating habits; chance or bad luck; poor medical care in the past; pollution; aging; alcohol; smoking; accident or injury; altered immunity). Patients scored the self-perceived relevance of these potential causes on a scale from 1 (strongly

disagree) to 5 (strongly agree). The responses " I neither agree nor disagree," "I agree," and "I strongly agree" were categorized as "(potential) endorsement." The responses "I strongly disagree" and "I disagree" were categorized as "nonendorsement." The proportion of "(potentially) endorsed" and "nonendorsed" "emotional" and "non-emotional" causes was compared using chi¯square statistics.

Patients were telephoned again approximately 11 weeks after receiving the diagnosis and asked to provide a further self-report estimate of seizure frequency.

### Statutory permission

This study was approved by the Northern and Yorkshire Multicentre Research Ethics Committee, Leeds, United Kingdom. Approval was also secured separately from the Research Governance departments of all participating National Health Service Trusts.

## Results

### Neurologists

The 23 neurologists who participated in this study worked for 15 different NHS Trusts across England, Wales, and Scotland. Eighty-six percent (19 of 23) were male. The proportion of the neurologist's work time taken up by seizure disorders ranged from less than 10% to 100% (median 55%). Ten of the 23 neurologists who took part in this study recruited patients.

### Patients

A total of 56 eligible patients were identified. Five declined to participate after receiving the study information, and one could not be contacted. Fifty patients completed the telephone interview. Thirty-six patients returned the postal questionnaires (72%), and 49 patients completed the telephone follow-up (98%).

Ninety percent of patients were female. The participants received the diagnosis of PNES a median of 5.2 years after seizure manifestation (range 2 months to 27 years); their median age was 29.5 years (range 17–69 years). Twenty percent of patients (10 of 50) not only saw the neurologist at the point of diagnosis but also met an Epilepsy Specialist Nurse. Of these patients 90% (9 of 10) stated that the nurse discussed the same things they had talked about with the neurologist. Forty percent of patients (20 of 50) received a letter from their neurologist following the consultation. Of these 90% (18 of 20) stated that the letter discussed what had been covered in the consultation.

Fifty percent of the participants (27 of 50) had taken antiepileptic drugs (AEDs) prior to the diagnosis of PNES. Of these patients, 89% (24 of 27) were advised to stop their AEDs during the consultation. Two weeks after the clinic visit, 67% (18 of 27) had stopped the AED treatment, and 11% (3 of 27) were in the process of reducing the medication. Three patients had not been advised to reduce their AEDs, and three decided not to reduce the AEDs despite being advised to do so. This means that 12% of all patients (6 of 50) in this group continued to take AEDs.

### Patients' experience with the communication strategy

Ninety-four percent of patients (47 of 50) could recall being given the PNES booklet developed by the researchers. Of those, 94% (44 of 47) found it clear and easy to understand and 91% (43 of 47) particularly liked something about the booklet. Nine percent (4 of 47) disliked something, for example, parts not being personally relevant and finding parts of it confusing. Eleven percent (5 of 47) had questions that were not covered. These questions related to memory, headaches, pregnancy, the possibility of concurrent epilepsy, and the difficulty in finding the cause of the attacks.

When asked "did you get out of the consultation what you wanted?" 70% of patients said "yes," 24% "partly," and 6% "no." Ninety-four percent of patients stated that they had their questions answered, 2% said questions had been partly answered, and 4% not answered. Fourteen percent of patients found the consultation confusing, 30% partly confusing, and 56% not confusing. Table 2 gives an overview of patients' feelings during the consultation. Initially cued to describe any positive or negative emotions during their meeting with the neurologist, patients were also prompted with a list of possible reactions generated by the authors on the basis of previous studies (Carton et al., 2003; Green et al., 2004; Thompson et al., 2005, 2009).

Table 3 summarizes patients' cued and prompted reports of the factual information included in the information leaflet and crib sheet 2 weeks after receiving the diagnosis. The most important 12 points of the communication

**Table 2. Patients' feelings during the consultation in which they were given the diagnosis of PNES**

| Feelings during the consultation | | Cued recall (%) | Cued and prompted (%) |
|---|---|---|---|
| Negative (any: 86%) | Upset | 48 | 48 |
| | Anxious | 38 | 40 |
| | Confused | 24 | 26 |
| | Down/depressed | 14 | 16 |
| | Angry/frustrated | 4 | 6 |
| Positive (any: 98%) | Felt listened to | 2 | 90 |
| | Relieved | 14 | 86 |
| | Felt understood | 0 | 86 |
| | Happy | 18 | 60 |

PNES, psychogenic nonepileptic seizures.

*Epilepsia*, 51(1):70–78, 2010
doi: 10.1111/j.1528-1167.2009.02099.x

**Table 3. Patients' reports of factual information contained in the communication strategy**

| Item | Cued reports (%) | Cued and prompted reports (%) |
|---|---|---|
| Name of the condition ("nonepileptic attacks") | 78 | 98 |
| Alternative names | 32 | N/A |
| PNES are common | 54 | 70 |
| PNES are not due to epilepsy | 28 | 92 |
| Difficult to know why attacks first started | 0 | 74 |
| Stress could be a maintaining factor | 20 | 94 |
| Understanding how stress leads to attacks | 8 | 76 |
| Some understanding of psychological etiology | 72 | 72 |
| Antiepileptic drugs are not effective | 54 | 72 |
| Psychological treatment suggested | 58 | 92 |
| Attacks are likely to improve | 42 | 84 |
| Attacks are likely to stop | 50 | 82 |

PNES, psychogenic nonepileptic seizures.

procedure were reportedly covered in a median of 82% of consultations (range 70–98%).

When given the choice of five different physical or psychological attributions about their attacks previously put to patients with medically explained or unexplained fatigue (Wessely & Powell, 1989), 13.8% of patients endorsed "my problem is a purely physical one," 11.1% "my problem is mainly physical but some psychological factors are involved," 25% "both physical and psychological factors are involved in my problem," 16.7% "although there are some physical reasons for my problems, it is mainly psychological in nature," and 25% "my problem is a psychological one."

The results of the IPQ-R completed 2–4 weeks after the encounters with the neurologists are summarized in Table 4. The mean response score on the "emotional" causes for the seizures offered by the IPQ-R was 2.7, with a range of 2.1 (overwork) to 3.7 (stress/worry). The mean response score on the "nonemotional" items was 1.9, with a range of 1.4 (alcohol) to 2.8 (accident/injury). Participants "(potentially) endorsed" 52.5% "emotional" compared to 21.7% "nonemotional" causes ($\chi^2 = 55.74$, $p < 0.001$). The list of the three most important causes most commonly included stress/worry (61%), physical injury/illness (50%), emotional state (50%), mental attitude (28%), family problems (25%), and poor medical care in the past (19%).

### PNES frequency

Table 5 shows the distribution of PNES frequency before the diagnosis, 2 weeks [mean 15 days, standard deviation (SD) 21.1] and 11 weeks (mean 77.9 days, SD 28.4) after patients received the diagnosis. Three patients could not be included because they were unable to estimate their seizure frequency before diagnosis; one patient was lost to follow-up. The seizure frequency improved by at least one category (i.e., by >50%) between diagnosis and follow-up in 63% of patients (29 of 46), remained unchanged in 24% (11 of 46), and deteriorated in 14% (6 of 46).

## Discussion

Patients with PNES are a clinically heterogeneous group (Lesser, 1996; Reuber, 2008). Nevertheless the occasion when a seizure expert (usually a neurologist) informs the patient of the diagnosis of PNES is a relatively stereotyped encounter. Because of this we devised a standardized procedure consisting of a detailed patient information leaflet, a more extensive booklet for neurologists, and a crib sheet covering the core aspects of the communication. The communication strategy was conceived as the starting point of future intervention studies. This preliminary study evaluates the acceptability and effectiveness of this strategy in a real-life clinic

**Table 4. Illness perceptions after the consultation with the neurologist and receipt of the information leaflet, as revealed by the IPQ-R (Moss-Morris et al., 2002)**

| IPQ-R Scale | Explanation | Possible range | Median (n = 36) | IQR |
|---|---|---|---|---|
| Illness identity | High scores represent strongly held beliefs about the number of symptoms attributed to the illness | 0–29 | 10 | +/−7 |
| Timeline acute/chronic | High scores represent strongly held beliefs about the chronicity of the condition | 6–30 | 19 | +/−7 |
| Timeline cyclical | High scores represent strongly held beliefs about the cyclical nature of the condition | 4–20 | 15 | +/−3 |
| Consequences | High scores represent strongly held beliefs about the negative consequences of the illness | 6–30 | 22 | +/−7 |
| Personal control | High scores represent positive beliefs about the controllability of the illness | 6–30 | 18 | +/−5 |
| Treatment control | High scores represent positive beliefs about the controllability of the illness | 5–25 | 16 | +/−3 |
| Emotional representation | High scores represent negative emotional representations of the illness | 6–30 | 22 | +/−7 |
| Illness coherence | High scores represent a personal understanding of the condition | 5–25 | 11 | +/−5 |

IPQ-R, Illness Perception Questionnaire-Revised.

**Table 5. Change in PNES frequency after communication of the diagnosis**

| | Before diagnosis (n = 50) | 2 weeks after diagnosis (n = 50) | 11 weeks after diagnosis (n = 49) |
|---|---|---|---|
| Daily | 21 (42) | 8 (16) | 9 (18) |
| Weekly | 20 (40) | 23 (46) | 22 (45) |
| Monthly | 6 (12) | 12 (24) | 8 (16) |
| Occasionally | 0 (0) | 4 (8) | 3 (6) |
| None | 0 (0) | 3 (6) | 7 (14) |
| Not classified | 3 (6) | 0 (0) | 0 (0) |

Values are expressed as n (%).
PNES, psychogenic nonepileptic seizures.

setting. The high levels of reported adherence to the communication strategy suggest that the communication strategy was acceptable to the participating clinicians and that the core points were communicated in most encounters.

Our approach differs from the only previously published proposal of how to communicate the diagnosis of PNES in a number of important details (Shen et al., 1990). The communication protocol suggested by Shen et al. begins with a demonstration of the recorded PNES. However, 23–30% of patients with PNES never have a seizure during video-EEG tests (even when seizure provocation is used) (Benbadis et al., 2004; McGonigal et al., 2004). What is more, most diagnoses in the United Kingdom are given in an outpatient setting with limited access to seizure recordings. Another important difference between the procedures is that our strategy uses a medical label (''non-epileptic attacks'') and provides patients with a psychological model of the etiology of the seizures, whereas the older proposal emphasizes that it is not known what the attacks are and uses the nonspecific term ''spell.'' There is ample evidence that patients (especially patients with medically unexplained symptoms) (Madden & Sim, 2006) are keen to understand what is causing their symptoms and to receive a diagnostic label (Green et al., 2004). They are more likely to achieve mastery over their health problems or cope with ongoing symptoms if they have a better understanding of their nature (Peters et al., 1998). There is no convincing evidence that the use of labels is harmful. We felt that a comprehensible and acceptable etiologic model and a clear label were likely to reduce confusion, and increase confidence in the clinician and acceptance of the treatment or referral plan.

Furthermore, Shen et al. suggest that patients should be questioned specifically about the possibility of sexual abuse. Childhood sexual abuse is likely to be reported in a significant proportion of clinic cases of PNES (Sharpe & Faye, 2006), but it is probably centrally important in only a minority (Reuber et al., 2007). We do not think that it needs to be addressed in this particular setting. In fact, it might be unsafe to discuss this issue in a clinic without rapid follow-up or suitable support systems. Disclosure of traumatic events (without any further intervention) was found not to have a beneficial effect on outcome in a similar patient group (Schilte et al., 2001).

Perhaps the most significant difference between the communication protocol suggested by Shen et al. and the approach described here is the central role apportioned to the detailed patient information leaflet in this study. The older proposal does not involve the use of written information. Our leaflet was devised because previous studies identified ''confusion'' as the most common sentiment after receipt of the diagnosis of PNES (Carton et al., 2003; Thompson et al., 2009). The leaflet was also used as a means of ensuring that all patients in this study were equipped with the same basic information about PNES at the point of diagnosis. This would obviously be of great benefit in future intervention studies. The leaflet was very well received by the patients, only 11% of whom found that they had questions that had not been sufficiently well covered. Most of these questions would not have been applicable to the majority of readers.

Patients' reported feelings about the consultation with the neurologist were more positive than previous studies would suggest (Carton et al., 2003; Thompson et al., 2005, 2009). Although patients volunteered more negative than positive feelings—with prompting, positive feelings were reported more frequently than negative sentiments. What is more, most negative feelings (like being ''upset,'' ''anxious,'' or ''confused'') may have been attributable to the nature of the problem rather than the quality of the interaction with the neurologist. This interpretation is supported by the fact that 94% of patients stated that they had their questions answered by the neurologist, 90% of patients felt they had been ''listened to,'' and only 14% of patients found the consultation confusing. The number of patients who claimed to have been angry during the consultation (4%) was much lower than the 18% reporting anger after receiving the diagnosis in a previous study (Carton et al., 2003).

Open inquiries were followed by prompts in this study because we wanted to ensure that participants had understood the question fully and were given an opportunity to consider all possible answers. The use of prompts made it easier to compare responses of different participants. At this point it is not known whether the marked differences of responses to open or prompted questions is characteristic of this particular patient group. The difference certainly raises important questions about the validity of self-report questionnaires in this clinical area. The discrepancies observed here suggest that in future outcome studies, self-report questionnaires should be combined with other methods of observation (such as qualitative approaches, the use of objective behavioral data, or additional questioning of healthcare staff or care givers).

In marked contrast to patients who present to neurologists with unexplained fatigue of whom 94% attributed their symptoms to physical causes (Wessely & Powell, 1989), only 11% of the patients in this study thought that their seizures had a purely physical etiology after they had received the information leaflet and seen the doctor. On the IPQ-R, patients endorsed emotional causes more strongly and frequently than nonemotional causes for their symptoms. Four of the most common six causes listed as most important by the participants in this study can be classed as ''emotional'' (stress/worry, emotional state, mental attitude, family problems). These findings suggest that only a small minority of patients failed to take on board core aspects of the psychological etiologic model that the communication was meant to convey.

Patients' illness perceptions after receipt of the diagnosis of PNES were characterized by much higher IPQ-R ''Illness Identity'' and ''Timeline Cyclical'' scores than reported in previous studies of patients with rheumatoid arthritis, chronic fatigue syndrome, or chronic pain (Moss-Morris et al., 2002; Moss-Morris & Chalder, 2003). The ''Illness Identity'' score measures the number of symptoms that patients attribute to their condition; the high ''Timeline Cyclical'' score reflects the unpredictability of their condition. Perhaps these two factors explain why the ''Illness Coherence'' score in our study (reflecting patients' subjective understanding of their problem) was lower than in the conditions listed previously, although patients reported less confusion about the diagnosis here than in previous studies (Carton et al., 2003; Green et al., 2004). A low ''Illness Coherence'' score is not necessarily a negative finding. One study in patients with eating disorders found that patients are less likely to resist change and more likely to embrace treatment if they have low ''Illness Coherence'' and high ''Emotional Representation'' scores (Stockford et al., 2007).

Our finding that 6% of patients were seizure-free 2 weeks after diagnosis and 14% after 11 weeks, fits well with a range of previous studies, which show that the short term outcome of PNES after diagnosis (at least in terms of achieving seizure control) is better than naturalistic long-term outcome studies suggest (Reuber et al., 2003b). One prospective investigation found that 6 months after diagnosis, PNES had stopped in 29% of patients (Kanner et al., 1999). A second study demonstrated that 42% of patients had become seizure-free after 1 year (So et al., 2004). Two studies showed that PNES had stopped in half of the patients at 18 months (Ettinger et al., 1999), or after 2 years of follow-up (Thompson et al., 2005). In fact the ''spontaneous'' improvements in terms of seizure cessation or reduction seen here are no worse than the treatment outcomes reported in the only controlled (pilot) study of a cognitive behavioral therapy treatment for patients with PNES (Goldstein et al., 2004), indicating the urgent need for larger controlled studies of the management modality currently favored by most clinicians.

Our study has a number of limitations. First and foremost, we did not standardize the communication procedure completely. Although the leaflet and crib sheet were core parts of the communication package, some patients also spoke to an epilepsy specialist nurse and some received a personalized clinic letter from their neurologist. It would be desirable, especially for comparative studies, to standardize the communication procedure more rigorously. Next, there has to be a degree of uncertainty about the self-reported seizure frequency data. Confirmation of frequency estimates was sought from caregivers, friends, or relatives, if possible, but ideally seizure frequency data would be collected prospectively using diaries. Furthermore, we cannot say how well the communication strategy fared compared to neurologists' historical performance or other possible strategies. However, the authors have made all the communication materials and evaluation procedures used here available on the Internet and are happy for these materials to be used in comparative studies. The neurologists contributing to this study all had extensive experience with PNES patients. It is possible that this experience contributed to the effectiveness of their communication behavior and patients' perceptions of their encounters with the doctor. This study was carried out by neurologists who were established in this clinical area, to aid patient recruitment, but it would be desirable to confirm the effectiveness and acceptability of the communication strategy in the hands of physicians who face the challenge of explaining the diagnosis of PNES less frequently. The fact that the patient leaflet is a core aspect of our communication strategy should facilitate this. Finally, we cannot say how our communication procedure would have performed in a group of patients with recent-onset PNES. Although we were keen to recruit patients for this study who had had seizures for only weeks or months, the patients described here received the diagnosis a median of 5.2 years after seizure manifestation. However, one might expect that it would be easier to explain a psychological etiologic model to someone who has just developed seizures than to someone who has received medication for an incorrect diagnosis for several years.

Taking account of these limitations our results suggest that the information booklet and communication strategy we have devised for patients who are newly diagnosed with PNES is acceptable to patients, and communicates effectively a psychological model for PNES. Our findings suggest that giving a psychological explanation for PNES rarely results in patients becoming angry or being dissatisfied with their doctor. A substantial number of patients become free of seizures after the diagnosis of PNES had been communicated to them.

## Acknowledgment

This study was supported by the Ryder Briggs Trust.

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Disclosure: None of the authors has any conflict of interest to disclose.

## References

Aboukasm A, Mahr G, Gahry BR, Thomas A, Barkley GL. (1998) Retrospective analysis of the effects of psychotherapeutic interventions on outcomes of psychogenic nonepileptic seizures. *Epilepsia* 39:470–473.

American Psychiatric Association. (1994) *Diagnostic and statistical manual of mental disorders*. APA, Washington, DC.

Angus-Leppan H. (2008) Diagnosing epilepsy in neurology clinics: a prospective study. *Seizure* 17:431–436.

Benbadis SR, Allen HW, Hauser WA. (2000) An estimate of the prevalence of psychogenic non-epileptic seizures. *Seizure* 9:280–281.

Benbadis SR, O'Neill E, Tatum WO, Heriaud L. (2004) Outcome of prolonged video-EEG monitoring at a typical referral epilepsy center. *Epilepsia* 45:1150–1153.

Binzer M, Stone J, Sharpe M. (2004) Recent onset pseudoseizures - clues to aetiology. *Seizure* 13:146–155.

Carton S, Thompson PJ, Duncan JS. (2003) Non-epileptic seizures: patients' understanding and reaction to the diagnosis and impact on outcome. *Seizure* 12:287–294.

de Timary P, Fouchet P, Sylin M, Indriets JP, de Barsy T, Lefebvre A, van Rijckevorsel K. (2002) Non-epileptic seizures: delayed diagnosis in patients presenting with electroencephalographic (EEG) or clinical signs of epileptic seizures. *Seizure* 11:193–197.

Ettinger AB, Devinsky O, Weisbrot DM, Ramakrishna RK, Goyal A. (1999) A comprehensive profile of clinical, psychiatric, and psychosocial characteristics of patients with psychogenic nonepileptic seizures. *Epilepsia* 40:1292–1298.

Galimberti CA, Ratti MT, Murelli R, Marchioni E, Manni R, Tartara A. (2003) Patients with psychogenic nonepileptic seizures, alone or epilepsy associated, share a psychological profile distinct of epilepsy patients. *J Neurol* 250:338–346.

Goldstein LH, Deale A, Mitchell-O'Malley S, Toone BK, Mellers JDC. (2004) An evaluation of cognitive behavioral therapy as a treatment for dissociative seizures. *Cogn Behav Neurol* 17:41–49.

Gray SE, Rutter DR. (2007) Illness representations in young people with Chronic Fatigue Syndrome. *Psychol Health* 22:159–174.

Green A, Payne S, Barnitt R. (2004) Illness representations among people with non-epileptic seizures attending a neuropsychiatry clinic: a qualitative study based on the self-regulation model. *Seizure* 13:331–339.

Hagger MS, Orbell S. (2003) A meta-analytic review of the common-sense model of illness representations. *Psychol Health* 18:141–184.

Harden CL, Ferrando S. (2001) Controversies in epilepsy and behavior: delivering the diagnosis of psychogenic pseudoseizures, should the neurologist or psychiatrist be responsible. *Epilepsy Behav* 2:519–523.

Howlett S, Grünewald R, Khan A, Reuber M. (2007) Engagement in psychological treatment for functional neurological symptoms - barriers and solutions. *Psychother Theory Res Pract Train* 44:354–360.

Kanner AM. (2003) More controversies on the treatment of psychogenic pseudoseizures: an addendum. *Epilepsy Behav* 4:360–364.

Kanner AM, Parra J, Frey M, Stebbins G, Pierre-Louis S, Iriarte J. (1999) Psychiatric and neurologic predictors of psychogenic pseudoseizure outcome. *Neurology* 53:933–938.

Kotsopoulos IA, de Krom MC, Kessels FG, Lodder J, Troost J, Twellaar M, van MT, Knottnerus AJ. (2003) The diagnosis of epileptic and non-epileptic seizures. *Epilepsy Res* 57:59–67.

LaFrance WC Jr, Rusch MD, Machan JT. (2008) What is "treatment as usual" for nonepileptic seizures? *Epilepsy Behav* 12:388–394.

Lawton G, Baker GA, Brown RJ. (2008) Comparison of two types of dissociation in epileptic and nonepileptic seizures. *Epilepsy Behav* 13:333–336.

Lesser RP. (1996) Psychogenic seizures. *Neurology* 46:1499–1507.

Leventhal H, Diefenbach M, Leventhal EA. (1992) Illness cognitions: using common sense to understand treatment adherence and affect cognition interactions. *Cognit Ther Res* 16:143–163.

Madden S, Sim J. (2006) Creating meaning in fibromyalgia syndrome. *Soc Sci Med* 63:2962–2973.

Martin RC, Gilliam FG, Kilgore M, Faught E, Kuzniecky R. (1998) Improved health care resource utilization following video-EEG-confirmed diagnosis of nonepileptic psychogenic seizures. *Seizure* 7:385–390.

McGonigal A, Russell AJ, Mallik AK, Oto M, Duncan R. (2004) Use of short term video EEG in the diagnosis of attack disorders. *J Neurol Neurosurg Psychiatry* 75:771–772.

Moss-Morris R, Chalder T. (2003) Illness perceptions and levels of disability in patients with chronic fatigue syndrome and rheumatoid arthritis. *J Psychosom Res* 55:305–308.

Moss-Morris R, Weinman J, Petrie KJ, Horne R, Cameron LD, Buick D. (2002) The Revised Illness Perception Questionnaire (IPQ-R). *Psychol Health* 17:1–16.

Peters S, Stanley I, Rose M, Salmon P. (1998) Patients with medically unexplained symptoms: sources of patients' authority and implications for demand on medical care. *Soc Sci Med* 46:559–565.

Prueter C, Schulz-Venrath U, Rimpau W. (2002) Dissociative and associated psychopathological symptoms in patients with epilepsy, pseudoseizures and both seizure forms. *Epilepsia* 43:188–192.

Reuber M, Fernández G, Bauer J, Helmstaedter C, Elger CE. (2002) Diagnostic delay in psychogenic nonepileptic seizures. *Neurology* 58:493–495.

Reuber M, House AO, Pukrop R, Bauer J, Elger CE. (2003a) Somatization, dissociation and psychopathology in patients with psychogenic nonepileptic seizures. *Epilepsy Res* 57:159–167.

Reuber M, Pukrop R, Bauer J, Helmstaedter C, Tessendorf N, Elger CE. (2003b) Outcome in psychogenic nonepileptic seizures: 1 to 10 year follow-up in 164 patients. *Ann Neurol* 53:305–311.

Reuber M, Howlett S, Khan A, Grünewald R. (2007) Non-epileptic seizures and other functional neurological symptoms: predisposing, precipitating and perpetuating factors. *Psychosomatics* 48: 230–238.

Reuber M. (2008) Psychogenic nonepileptic seizures: answers and questions. *Epilepsy Behav* 12:622–635.

Schilte AF, Portegijs PJ, Blankenstein AH, Der Horst HE, Latour MB, van Eijk JT, Knottnerus JA. (2001) Randomised controlled trial of disclosure of emotionally important events in somatisation in primary care. *Br Med J* 323:86.

Sharpe D, Faye C. (2006) Non-epileptic seizures and child sexual abuse: a critical review of the literature. *Clin Psychol Rev* 26:1020–1040.

Shen W, Bowman ES, Markand ON. (1990) Presenting the diagnosis of pseudoseizure. *Neurology* 40:756–759.

So NK, Bennett C, Smith B. (2004) Prospective outcome after diagnosis of nonepileptic seizures. *Epilepsia* 45(Suppl. 7):266–267.

Stockford K, Turner H, Cooper M. (2007) Illness perception and its relationship to readiness to change in the eating disorders: a preliminary investigation. *Br J Clin Psychol* 46:139–154.

Stone J, Binzer M, Sharpe M. (2004) Illness beliefs and locus of control: a comparison of patients with pseudoseizures and epilepsy. *J Psychosom Res* 57:541–547.

Thompson NC, Osorio I, Hunter EE. (2005) Nonepileptic seizures: reframing the diagnosis. *Perspect Psychiatr Care* 41:71–78.

Thompson R, Isaac CL, Shakes G, Tooth C, Reuber M. (2009) What is it like to receive a diagnosis of non-epileptic seizures? *Epilepsy Behav* In press.

Wessely S, Powell R. (1989) Fatigue syndromes: a comparison of chronic "postviral" fatigue with neuromuscular and affective disorders. *J Neurol Neurosurg Psychiatry* 52:940–948.

World Health Organization. (1992) *The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines*. WHO, Geneva.

*Epilepsia*, 51(1):70–78, 2010
doi: 10.1111/j.1528-1167.2009.02099.x

## Appendix

The other Non-Epileptic Seizure Treatment (NEST) collaborators are: Dr. Naghme Adab, Coventry; Dr. Jane Adcock, Oxford; Dr. Steven Allder, Plymouth; Dr. Holger Allroggen, Coventry; Dr. Jonathan Bird, Bristol; Dr. Peter Clough; Dr. Hannah Cock, London; Dr. Paul Cooper, Manchester; Dr. Gary Dennis, Sheffield; Dr. Peter Goulding, Leeds; Dr. Richard Grünewald, Sheffield; Dr. Yvonne Hart, Oxford; Dr. Steven Howell, Sheffield; Dr. Mark Manford, Bedford; Dr. Tony Marson, Liverpool; Dr. Piers Newman, Hereford; Dr. Andrew Nicholson, Liverpool; Dr. Fiona Pattison, Bristol; Dr. Martin Sadler, Plymouth; Dr. David Smith, Liverpool; Dr. Tim von Oertzen, London; Dr. Martin Zeidler, Kirkcaldy.

5. Petrie KJ, Müller JT et al. Effect of providing information about normal test results on patients' reassurance: randomized controlled trial. BMJ 2007;334;352.

Cite this article as: BMJ, doi:10.1136/bmj.39093.464190.55 (published 26 January 2007)

BMJ

RESEARCH

# Effect of providing information about normal test results on patients' reassurance: randomised controlled trial

Keith J Petrie, professor,[1] Jan Tobias Müller, diplom psychologist,[3] Frederike Schirmbeck, diplom psychologist,[3] Liesje Donkin, student,[1] Elizabeth Broadbent, lecturer,[1] Christopher J Ellis, cardiologist,[2] Greg Gamble, statistician,[2] Winfried Rief, professor[3]

[1]Department of Psychological Medicine, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, New Zealand

[2]Department of Cardiology, Auckland City Hospital

[3]Philipps-University, Faculty of Psychology, Marburg, Germany

**Correspondence to:**
**K J Petrie**
**kj.petrie@auckland.ac.nz**

doi: 10.1136/bmj.39093.464190.55

## ABSTRACT

**Objective** To investigate whether providing information about normal findings before a diagnostic test improves patients' reassurance and reduces anxiety about symptoms.

**Design** Randomised controlled trial.

**Setting** Outpatient cardiology clinic.

**Participants** 92 patients with chest pain referred for a diagnostic exercise stress test.

**Intervention** Before undergoing testing patients were randomised to receive standard information (n=28; control group), a pamphlet explaining the function of the test and the meaning of normal test results (n=30; pamphlet group), or the pamphlet and a brief discussion about the meaning of normal test results (n=34; discussion group).

**Main outcome measures** The primary outcome was patients' reported reassurance on a 5 item scale immediately after the test and at one month. Secondary outcomes were the proportion of patients still with chest pain and still taking cardiac drugs at one month.

**Results** The mean levels of reassurance after testing and feedback from the doctor were significantly higher in the discussion group (42.0, 95% confidence interval 39.7 to 44.2) than in the pamphlet (39.2, 36.1 to 42.3) and control groups (35.8, 31.6 to 39.9). This difference was maintained at one month. The proportion of patients still reporting chest pain at one month decreased significantly in the discussion group (to 17%) and pamphlet group (to 28%) but not in the control group (to 36%). A trend was for fewer patients in the discussion group to be taking cardiac drugs at one month.

**Conclusion** Providing patients with information about normal test results before testing can improve rates of reassurance and reduce the likelihood of future reports of chest pain.

**Trial registration** Current Controlled Trials ISRCTN87589121.

## INTRODUCTION

Reassurance is one of the most common interventions in medical practice. Yet many patients with symptoms remain anxious about their condition even after investigations and reassurance from their doctor.[1 2] Such patients often continue to be disabled by their condition, use drugs inappropriately, and seek medical help from other health professionals for their symptoms.[3 4]

Doctors typically give reassurance after investigations. Although this may seem logical, evidence suggests that by the time patients undergo tests many have already developed negative ideas and beliefs about their symptoms, and thus reassurance is much less effective.[5] This may especially be the case when there are delays in completing investigations. Patients' established negative beliefs about their symptoms may limit their ability to assimilate reassuring messages that are by then inconsistent with their view of the seriousness of their condition.[6] Furthermore, increasing the amount of reassurance after investigations does not always seem to reduce concerns about symptoms.[7]

The effects that patients' pre-existing ideas have on reassurance suggest a possible pathway to improve reassurance. Providing an explanation about the meaning of normal test results before testing may weaken patients' preconceived ideas about their illness and provide a context to help patients make sense of the test result. Patients will be better prepared to receive reassurance from their doctor and the effects will be strengthened.

We investigated whether giving patients information about a diagnostic test and discussing the meaning of normal results before the test would improve rates of reassurance.

## METHODS

Eligible participants were those with chest pain referred for a diagnostic exercise stress test at Auckland City Hospital. The study took place between June and October 2004. We excluded patients aged less than 18 years and those who had a previous diagnosis of cardiac disease, had no symptoms of chest pain, or were undergoing the test as part of a presurgical examination. Participants were randomised to one of three intervention groups according to a computer generated random number sequence. Allocation was concealed in sequentially numbered sealed opaque envelopes.

### Intervention groups

*Control group*

Patients in the control group received a sheet of information on the exercise stress test from the cardiology clinic with their clinic appointment. This included advice on what to wear, the procedure, and the risks of complications.

*Pamphlet group*

The pamphlet group received a 450 word pamphlet to read before their stress exercise test (additional information is provided by the authors at www.health.auckland.ac.nz/psych-med/staff/keiths%20papers/bmj%20appendix.html). The pamphlet explained the function of the test, the meaning of normal results, and other possible reasons for chest pain that were less serious. The pamphlet was divided into seven sections, with each section headed by the following questions: What is an exercise stress test? How does the exercise stress test work? What happens during testing? What if it is too hard for me? What could the results mean? Could there be other causes for my chest pain? What if my chest pain continues but my test is normal?

*Discussion group*

Patients in the discussion group received the pamphlet and were later engaged in discussion with the research health psychologist, who asked if they had any questions about what they had read. The psychologist briefly reiterated the main points of the pamphlet–that a lot of people with chest pain worry that there might be something wrong with their heart; that if the result of the test is normal the patient's risk for coronary artery disease is as low as for anyone in the general population; and that just because the pain may not be related to the heart does not mean that it is not real pain and that it is important to keep in mind that many other causes of chest pain are less serious.

Patients completed the test according to the standard Bruce exercise stress testing protocol.[8] When results were negative, a cardiology registrar provided patients with their standard reassurance that the result was normal and did not show cardiac disease and that a report would be sent to their general practitioner.

### Baseline assessment

Patients meeting the eligibility criteria were approached in the waiting room. After providing informed consent they completed a questionnaire on personal data, pain ratings, concerns about symptoms, and self-rated health. They were asked to rate how worried they were about their health (from 0 "not at all" to 10 "extremely") and the extent to which they believed there was something seriously wrong with their heart (from 0 "not at all" to 10 "strongly believe"). The psychologist then opened the envelope with the randomisation code.

### Post-testing questionnaire

Immediately after the test the patients completed a brief questionnaire comprising the two items on concerns about their health and heart and three further items. They were asked to rate on 10 point scales the extent to which they were reassured by the test, the extent to which they believed they needed further tests to determine the cause of their illness, and how accurate they thought the test for identifying heart problems. After reversing three of the negatively worded items we summed the scores for these five items to create a scale for reassurance, with higher scores indicating higher levels of reassurance. These items for reassurance have been used previously,[5] and the scale showed acceptable internal consistency (Cronbach's $\alpha$=0.80).

### One month follow-up

At one month a researcher blind to the allocation group telephoned the patients and completed the reassurance scale. Patients were also asked if they still had chest pain and if they were still taking cardiac drugs.

### Statistical analysis

We defined participants as reassured when their reassurance level was above the median for the total sample. Our study is thus conservatively powered to detect a medium (Cohen W=0.38) effect size using PASS.[9] This effect size is achieved with a sample size of 66 (80% power, 5% significance level) split between three groups. We therefore recruited 90 participants to compensate for losses to follow-up.

We used SAS statistical software v 9.1 for the analyses. We brought post-test data forward as a proxy for the assessment at one month of the five participants lost to follow-up. To examine differences between the groups on the reassurance scale we used a repeated measures mixed model analysis. We used Tukey post-hoc tests to determine differences between individual groups and across time. To determine potential differences in the proportion of patients reassured (above median at post-testing and follow-up) between the groups and over time we used a categorical modelling procedure (SAS Proc Catmod). McNemar's test was used to assess whether there were within group differences in reported chest pain. All tests were two tailed and we considered $P<0.05$ as significant.

## RESULTS

Of 97 eligible adults with chest pain referred for a diagnostic exercise stress test, five declined to participate (fig 1). Overall, 28 were randomised to receive standard information on the test, 30 to receive an information pamphlet and explanation of the meaning of normal test results, and 34 to receive the pamphlet and a brief discussion about the meaning of normal test results. Fifteen had a positive test result and were excluded. All patients completed the brief post-test assessment. Five patients could not be traced at one month.

Table 1 lists the baseline personal characteristics and clinical details of the experimental groups, including waiting times for the investigation, experience of a previous exercise stress test, and pain. The groups were

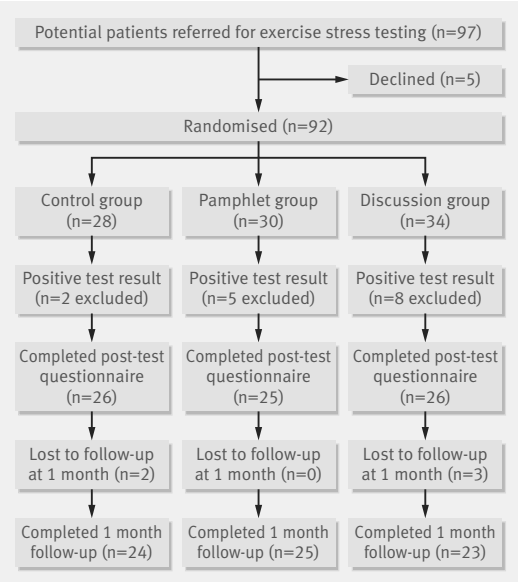



**Fig 1** | Participant flow through study

**Table 1** | Personal and clinical characteristics of groups at baseline. Values are numbers (percentages) unless stated otherwise

| Variable | Control group (n=28) | Pamphlet group (n=30) | Discussion group (n=34) |
|---|---|---|---|
| Mean (SD) age (years) | 52.2 (13.2) | 56.9 (13.6) | 52.2 (13.3) |
| Men | 19 | 16 | 17 |
| Women | 9 | 14 | 17 |
| Ethnicity: | | | |
| European | 18 | 22 | 23 |
| Other | 10 | 8 | 11 |
| Education: | | | |
| Secondary | 16 | 15 | 19 |
| Post | 12 | 15 | 15 |
| Employment: | | | |
| Full time | 14 | 13 | 20 |
| Other | 14 | 17 | 14 |
| Previous stress test: | | | |
| Yes | 4 | 9 | 8 |
| No | 24 | 21 | 26 |
| Mean (SD wait for investigation (weeks) | 8.1 (10.0) | 6.7 (6.7) | 8.4 (10.4) |
| Mean (SD) pain severity (1-10)* | 3.2 (2.2) | 3.4 (2.1) | 3.2 (2.4) |
| Mean (SD) pain limitation (1-10)† | 2.2 (2.4) | 2.6 (2.2) | 2.1 (2.0) |
| Mean (SD) score for "How worried are you about your health?" (1-10)‡ | 4.4 (2.9) | 6.2 (2.7) | 5.4 (2.5) |
| Mean (SD) score for "Do you believe something is seriously wrong with your heart?" (1-10)§ | 7.4 (2.5) | 8.6 (1.5) | 7.3 (2.2) |

*1=no pain; 10=severe pain.
†1=not at all; 10=extremely limited.
‡1=not at all; 10=extremely.
§1=not at all; 10=strongly believe.

well balanced. The repeated measures analysis showed a significant difference between the groups on the reassurance scale after the test and at the one month follow-up (table 2; P=0.002) and no interaction with time (P=0.25). A retrospective analysis showed a significantly higher level of reassurance in the discussion group than in the control group.

Significant differences were found between the groups in reassurance after testing and at one month follow-up ($\chi^2$=7, df=2, P=0.03). In the retrospective analysis the discussion group had a higher proportion of reassured patients (65%) after testing than the control (50%) and pamphlet (44%) groups (P=0.03 and P=0.02). At one month this difference was maintained ($\chi^2$=1.92, df=2, P=0.38), with 69% of patients still reassured in the discussion group compared with 40% in the pamphlet group and 35% in the control group. No significant difference was found between the control and pamphlet groups (P=0.99). More participants in the control and pamphlet groups had lower levels of reassurance at both time points than those in the discussion group (fig 2).

All patients needed to have symptoms of chest pain to be included in the trial. At one month the numbers of patients who mentioned chest pain had reduced significantly to 4 (17%) in the discussion group (P<0.001) and to 7 (28%) in the pamphlet group (P=0.005) but the reduction in the control group to 9 (36%) was not significant (P=0.09). Consistent with these findings was a trend for fewer patients in the discussion group to be taking cardiac drugs: 6 (25%) patients in the control group, 2 (8%) in the pamphlet group, and 1 (4.3%) in the discussion group ($\chi^2$=5.3, df=2, P=0.07).

**Table 2** | Reassurance scores after exercise stress test and at one month follow-up in groups

| Group | Mean (95% CI) post-test scores | Mean (95% CI) follow-up scores |
|---|---|---|
| Control | 35.8 (31.6 to 39.9) | 34.4 (30.5 to 38.4) |
| Pamphlet | 39.2 (36.1 to 42.3) | 38.4 (35.4 to 41.4) |
| Discussion | 42.0 (39.7 to 44.2) | 43.4 (41.0 to 45.8) |

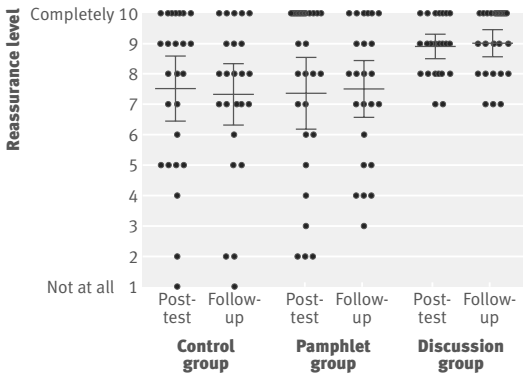



**Fig 2** | Dot plot for item asking patients how reassured they were by the exercise stress test after testing and at one month follow-up in experimental groups, including means (95% confidence intervals)

**WHAT IS ALREADY KNOWN ON THIS TOPIC**

Reassurance from doctors is a common medical intervention

Even after the completion of investigations and reassurance from doctors, many patients remain anxious about their symptoms

**WHAT THIS STUDY ADDS**

Written information and a discussion about normal results before testing improved rates of patients' reassurance

## DISCUSSION

Providing patients with chest pain who have been referred for exercise stress testing with information about the test and an explanation of normal test results before testing improved rates of reassurance and reduced the likelihood of future reports of chest pain. One month after the test, patients who had been randomised to an information pamphlet on the test and a brief discussion about normal results had fewer reports of chest pain, were more reassured by the test, and tended not to be taking cardiac drugs than patients who had been randomised to standard advice (control group) or to a pamphlet explaining the test and normal results. At one month most patients in the control group were not reassured by the investigation. The results of the study suggest that a simple low cost intervention that explains the meaning of normal results before investigations is likely to reduce patients' concerns about symptoms and unnecessary future medical care and investigations.

Previous research shows that a large number of patients without disease remain worried or uncertain after medical investigations.[10-12] We also found that half of the patients in the control group were not reassured by the standard advice they received before testing, and initial reassurance in this group tended to decline over time. An earlier study of patients with non-cardiac chest pain noted that many are unprepared for the possibility of negative findings and lack a context in which to interpret such results.[7] Providing prior information about the test and the meaning of a normal test result seemed to lead to better assimilation of reassuring messages. Furthermore, in patients prepared for a normal test result, reports of subsequent chest pain were reduced.

The strength of this study was that we were able to collect information on patients' concerns about their symptoms before testing, immediately after testing, and at one month. Also, only a few patients were lost at the follow-up assessment. It would be useful if future research collected information over an extended follow-up period, which would enable the assessment of any differences in future medical investigations in the study groups and an estimate of the economic benefits of the intervention. We used a health psychologist to engage patients in a discussion about the test and it remains to be established whether similar results can be obtained with a clinic nurse or registrar.

This relatively small study may best be considered as a proof of principle study, the results of which need replication in a larger sample. The implication of the study for clinicians is that an increase in patients' reassurance after clinical testing can be expected if more time is spent explaining the meaning of normal test results before the test. Improvements in reassurance are also likely to impact on unnecessary future investigations and patients' anxieties about their symptoms.

**Contributors:** KJP conceived and designed the study, drafted the manuscript, and is guarantor. JTM, FS, and LD helped develop the study materials and run the trial. GG, EB, CJE, and WR helped with data interpretation. CJE, WR, EB, and GG helped draft the paper. All authors contributed to and approved the final version.
**Funding:** University of Auckland.
**Competing interests:** None declared.
**Ethical approval:** This study was approved by the New Zealand Ministry of Health Ethics Committee (AKY/04/05/121).

1 Howard L, Wessely S. Reappraising reassurance: the role of investigations. *J Psychosom Res* 1996;41:307-11.
2 McDonald IG, Daly J, Jelinek VM, Panetta F, Gutman JM. Opening Pandora's box: the unpredictability of reassurance by a normal test result. *BMJ* 1996;313:329-32.
3 Potts SG, Bass CM. Psychological morbidity in patients with chest pain and normal or near normal coronary arteries: a long-term follow-up study. *Psychol Med* 1995;25:339-47.
4 Fitzpatrick R, Hopkins A. Referrals to neurologists for headaches not due to structural disease. *J Neurol Neurosurg Psychiatry* 1981;44:1061-7.
5 Donkin L, Ellis CJ, Powell R, Broadbent E, Gamble G, Petrie KJ. Illness perceptions predict reassurance following negative exercise testing result. *Psychol Health* 2006;21:421-30.
6 Nijher G, Weinman J, Bass C, Chambers J. Chest pain in people with normal coronary arteries. *BMJ* 2001;323:1319-20.
7 Sanders D, Bass C, Mayou RA, Goodwin S, Bryant BM, Tyndel S. Non-cardiac chest pain: why was a brief intervention apparently ineffective? *Psychol Med* 1997;27:1033-40.
8 Bruce RA, Hossack KF, DeRouen TA, Hofer V. Enhanced risk assessment for primary coronary heart disease events by maximal exercise testing: 10 years' experience of Seattle Heart Watch. *J Am Coll Cardiol* 1983;2:565-73.
9 Hintze J. *NCSS and PASS software*. Kaysville, UT: Number Cruncher Statistical Systems, 2004.
10 Lucock MP, Morley S, White C, Peake MD. Responses of consecutive patients to reassurance after gastroscopy: results of a self administered questionnaire survey. *BMJ* 1997;315:572-5.
11 Channer KS, James MA, Papouchado M, Rees JR. Failure of a negative exercise test to reassure patients with chest pain. *Q J Med* 1987;63:315-22.
12 Meechan GT, Collins JP, Moss-Morris R, Petrie KJ. Who is not reassured following benign diagnosis of breast symptoms? *Psychooncol* 2005;14:239-46.

**Accepted:** 12 December 2006

6. Stone J, Carson A, Sharpe M. Functional symptoms in neurology: management. J Neurol Neurosurg Psychiatry 2005;76 (suppl I): i13-i21.

# FUNCTIONAL SYMPTOMS IN NEUROLOGY: MANAGEMENT

J Stone, A Carson, M Sharpe

*J Neurol Neurosurg Psychiatry* 2005;**76**(Suppl I):i13–i21. doi: 10.1136/jnnp.2004.061663

In this article we offer an approach to management of functional symptoms based on our own experience and on the evidence from other specialities (because the evidence from neurology is so slim). We also tackle some of the most difficult questions in this area. What causes functional symptoms? Does treatment really work? What about malingering?

We give two example cases adapted from real patients to illustrate our approach.

## WHAT CAUSES FUNCTIONAL SYMPTOMS?

Table 1 is not comprehensive but it summarises a large literature on the suggested causes of functional symptoms. This is a question that has been approached from many angles—biological, cognitive, psychoanalytic, psychological, social, and historical. The factors shown have been found to be more common in patients with functional symptoms than in patients with similar symptoms clearly associated with disease pathology. Tables like this can help you to make a *formulation* of the aetiology of the patient's symptoms rather than just a diagnosis. An important feature of the table is the recognition of biological as well as psychological and social factors in the production and persistence of functional symptoms.

Most of the factors in table 1 have also been associated with other types of functional somatic symptoms such as irritable bowel syndrome and chronic pain as well as with depression or anxiety. Consequently they should be regarded more as vulnerability factors for developing symptoms, than as specific explanations for why some patients develop certain symptoms such as unilateral paralysis and others have attacks that look like epilepsy. Recent functional imaging studies of patients with functional motor and sensory symptoms[1] are beginning to offer biological clues (fig 1); they also challenge the idea that that such symptoms are ''all in the mind''—they are in the brain too.[2]

We will discuss the factors listed in table 1 later when considering an approached treatment. A fuller discussion of aetiology can be found elsewhere.[3]

### EXPLAINING THE DIAGNOSIS

In the first of these two articles we described our own approach to history taking and examination designed to be an efficient way of assessing the problem. The findings of this assessment will help you to *tailor* the explanation you give to individual patients. There is no ''one size fits all'' solution, but certain ways of saying things seem to work better than others.

Most people who develop symptoms want to know what is causing them. Explaining the diagnosis in a clear, logical, transparent, and non-offensive way is the key to management by the neurologist. It may sometimes be sufficient to produce improvement.

There are several reasons, both pragmatic and scientific, why we prefer the word ''functional'' in diagnoses such as ''functional weakness'' or ''functional sensory disturbance''. It is a diagnosis that: (1) replaces an erroneous physical versus psychological debate, allowing for a more productive functional/reversible versus structural/irreversible dichotomy; (2) provides a rationale for any treatment designed to improve the functioning of the nervous system—in particular, it allows the use of both physical and psychological strategies; (3) avoids offence (fig 2) and thus can be used transparently with the patient. We do acknowledge, however, that all diagnostic terms have limitations. The usefulness of the term with patients would diminish if ''functional'' became seen as a euphemism for psychogenic (as it is by some doctors now).

The word you use is probably not as important as the way that you use it. We probably underestimate our patients' ability to detect an unconvincing explanation (or one that the doctor does not really believe).

At a first encounter, our explanation of the diagnosis to the patient includes the following key points:

- *Explain what they do have*—For example, ''You have 'functional weakness'—this is a common problem. Your nervous system is not damaged but it is not working properly. That is why you cannot move your arm''.

See end of article for authors' affiliations

Correspondence to: Dr Jon Stone, Department of Clinical Neurosciences, Western General Hospital, Crewe Road, Edinburgh EH4 2XU, UK; Jon.Stone@ed.ac.uk

**Table 1** A scheme for thinking about the aetiology of functional symptoms in neurology

| Factors | Biological | Psychological | Social |
|---|---|---|---|
| Predisposing | ▶ Genetic factors affecting personality<br>▶ Biological vulnerabilities in nervous system?<br>▶ Disease | ▶ Poor ''attachment'' to parents and others<br>▶ Personality/coping style | ▶ Childhood neglect/abuse<br>▶ Poor family functioning |
| Precipitating | ▶ Abnormal physiological event or state (e.g. hyperventilation, sleep deprivation, sleep paralysis)<br>▶ Physical injury/pain | ▶ Perception of life event as negative, unexpected<br>▶ Depression/anxiety<br>▶ Acute dissociative episode/panic attack | ▶ Symptom modelling (via media or personal contact)<br>▶ Life events and difficulties |
| Perpetuating | ▶ Plasticity in CNS motor and sensory (including pain) pathways<br>▶ Deconditioning (e.g. lack of physical fitness in chronic fatigue, deconditioning of vestibular responsiveness in patients with dizziness who hold their head still)<br>▶ Neuroendocrine and immunological abnormalities similar to those seen in depression and anxiety | ▶ Perception of symptoms as being outwith personal control/due to disease<br>▶ Anxiety/catastrophisation about cause of symptoms<br>▶ Not being believed<br>▶ Avoidance of symptom provocation (e.g. exercise in fatigue) | ▶ Fear/avoidance of work or family responsibilities<br>▶ The presence of a welfare system<br>▶ Social benefits of being ill<br>▶ Availability of legal compensation<br>▶ Stigma of ''mental illness'' in society and from medical profession |

- *Indicate you believe the patient*—We pay particular attention to overcoming the patient's fear that you do not believe them or think they are mad, imagining or putting on their symptoms. If you have found that this is something they might be concerned about, simply saying ''I don't think you're mad, imagining or putting on your symptoms'' can be very effective.
- *Explain what they don't have*—For example, ''You do not have multiple sclerosis, epilepsy, etc''.
- *Emphasise that it is common*—For example, ''I see lots of patients with similar symptoms''.
- *Emphasise reversibility*—For example, ''Because there is no damage you have the potential to get better''.
- *Emphasise that self-help is a key part of getting better*—For example, ''I know you didn't bring this on but there are things you can do to help it get better''.

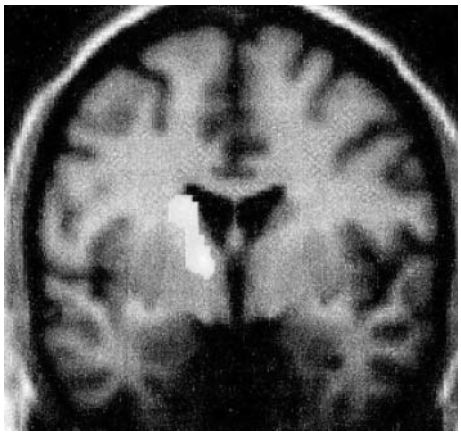


**Figure 1** A composite scan of four patients with functional hemimotor and sensory symptoms compared to recovery. There was hypoactivation of the contralateral thalamus, caudate, and putamen during the symptomatic state. Activations on a scan do not tell us how the symptom came to be there (or even if it was fabricated or not), but along with studies of endocrine and immunological abnormalities challenge a purely ''psychogenic'' view of the problem. Reproduced from Vuilliemier *et al*,[1] with permission from Oxford University Press.

- *Metaphors and comparisons may be useful*—For example, ''The hardware is alright but there's a software problem''; ''It's like a car/piano that's out of tune, all the parts are there, they just aren't working right together''; ''It's like a short circuit of the nervous system'' (non-epileptic attacks); ''It's like the opposite of phantom limb—they feel a limb that is not there, you cannot feel a limb that is (functional weakness)''.
- *Show the patient their positive signs*—For example, a patient (and their family) can be shown their own Hoover's sign or talked through a video of their non-epileptic attack. Explain how this confirms the diagnosis that the nervous system is working at some times but not at others.
- *Introducing the role of depression/anxiety*—Use your judgement about whether this will be helpful or harmful at an early stage. For example, ''If you have been feeling low/worried that will tend to make the symptoms even worse''.
- *Use written information*—Using this approach makes it much easier to send the patient their clinic letter. This in turn can do a lot to persuade them of your point of view. Leaflets perform a similar function (fig 3).
- *Talk to the family and friends*—Reinforce the diagnosis with family or friends.
- *Making a psychiatric referral*—For example, ''Dr X has a lot of experience and interest in helping people manage and overcome symptoms like this. Referring you to him does not mean that I think you are mad''.

Why not be more ''psychological''? Our clinical experience is that an approach which does not force the issue paradoxically actually *increases* the subsequent emergence and discussion of relevant psychological symptoms and life problems. When they emerge we avoid the temptation to simply re-attribute the symptoms to them (for example, ''…ah now I know that your husband is having an affair, that explains you weak leg'') but instead emphasise how important they may be as a factor making the symptoms worse.

The patient may want to know why this has happened to them. An honest answer might be that you do not know or that the reasons are probably complicated. But just because

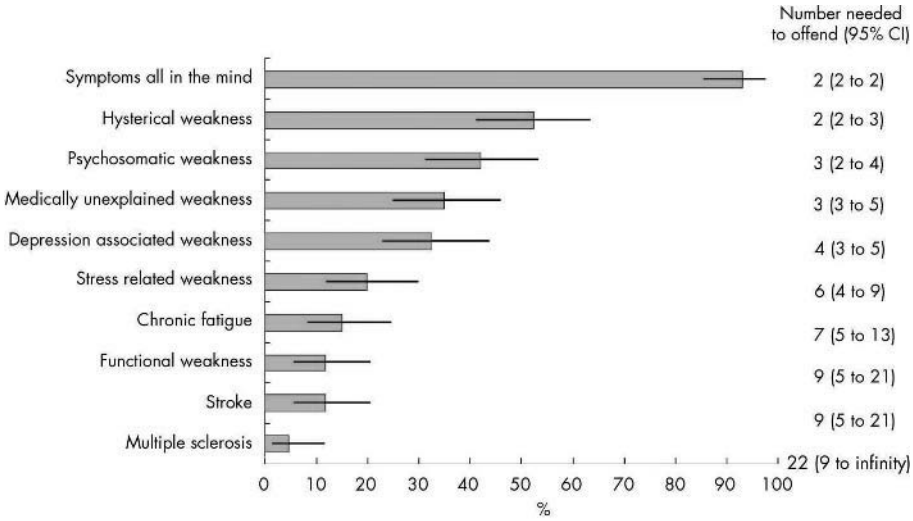



**Figure 2** Many words we use to describe symptoms unexplained by disease are potentially offensive to patients. These 86 general neurology patients were asked to imagine that they had a weak leg with normal tests and they were being given a diagnosis. The figure illustrates the percentage who would equate the diagnosis with being ''mad'', ''putting on'' symptoms, or ''imagining symptoms'', along with the number needed to offend—the number of patients that have to be given the diagnosis before one is offended. Reproduced from Stone *et al*,[13] with permission of the BMJ Publishing Group.

you do not know why something happens does not mean you cannot diagnose it, or treat it. You can explain to the patient that you would have just as much trouble trying to provide a *cause of their illness* if they had multiple sclerosis, migraine, or Parkinson's disease.

For patients with mild symptoms, explanation and reassurance with encouragement to resume normal activity may be sufficient. In those with more resistant symptoms one or more of the following treatments may be helpful.

## PHYSICAL REHABILITATION

Patients with physical problems often need physical treatments. Some of our best treatment successes have been accomplished by experienced neuro-physiotherapists who are able to combine hands on, physical treatments with explanation and encouragement. There have been encouraging results from studies of physical rehabilitation in patients with functional disability, but none of them has been randomised and few report long term outcome.[3] In patients with chronic fatigue there is systematic review evidence that graded exercise is helpful overall.

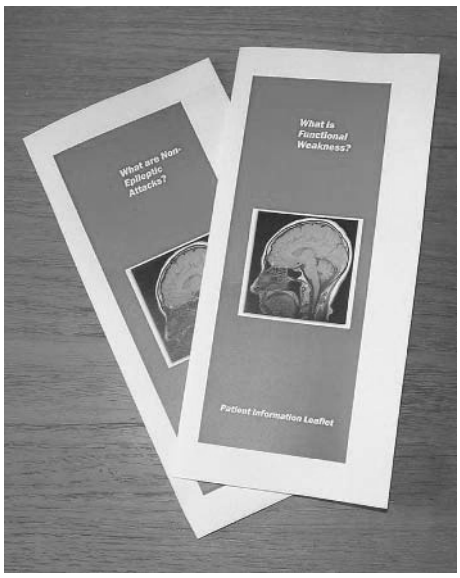



**Figure 3** Written information helps transparency and may help patient recovery.

## COGNITIVE BEHAVIOURAL THERAPY: HELPING THE PATIENT THINK AND BEHAVE DIFFERENTLY

Many neurologists regard cognitive behavioural therapy (CBT) as a mysterious treatment. It is not. Essentially it is an extension of the explanation, a way of helping the patient to become aware of, examine, and if appropriate revise the way they think, respond emotionally and behave in response to symptoms. The aim is to maximise function and reduce symptoms—but not necessarily to abolish them. In formal CBT the patient meets a therapist every one or two weeks and practices new ways of thinking about and responding to their symptoms between these sessions. In common with most neurological services we do not have ready access to specialist CBT therapists. We therefore try to incorporate the principles of CBT into medical care. Before you dismiss this suggestion as unrealistic you should be aware that it was well described by neurologists practising 100 years ago. They called it ''rational persuasion'' or ''re-education''.[2]

CBT emphasises the interaction of cognitive, behavioural, emotional, and physiological factors in perpetuating symptoms (fig 4).[4] The patient's cognitive interpretation of bodily symptoms is key. This will depend on their knowledge and experience of disease. For example, it is hardly surprising if someone with paralysis misinterprets their symptoms as multiple sclerosis or a stroke and as evidence of irreversible damage to their nervous system. In keeping with these beliefs the patient may behave in a way that seems sensible but which actually make the problem worse—for example, by avoiding behaviours or situations that exacerbate the symptom (such as exercise) or endlessly seeking a medical diagnosis before considering treatment. These factors have been demonstrated to be relevant to both the aetiology and treatment of chronic pain and chronic fatigue.

Evidence exists at systematic review level that CBT is effective for a wide range of functional somatic symptoms.[5] Its use has also been described (although not properly tested) in patients with non-epileptic attacks,[6] motor symptoms,[3] and severe and multiple functional symptoms. How can you do it? Think back to the 35 year old patient with disabling unilateral weakness, fatigue, and pain (as well as anger about being told that her symptoms are ''psychological'') described

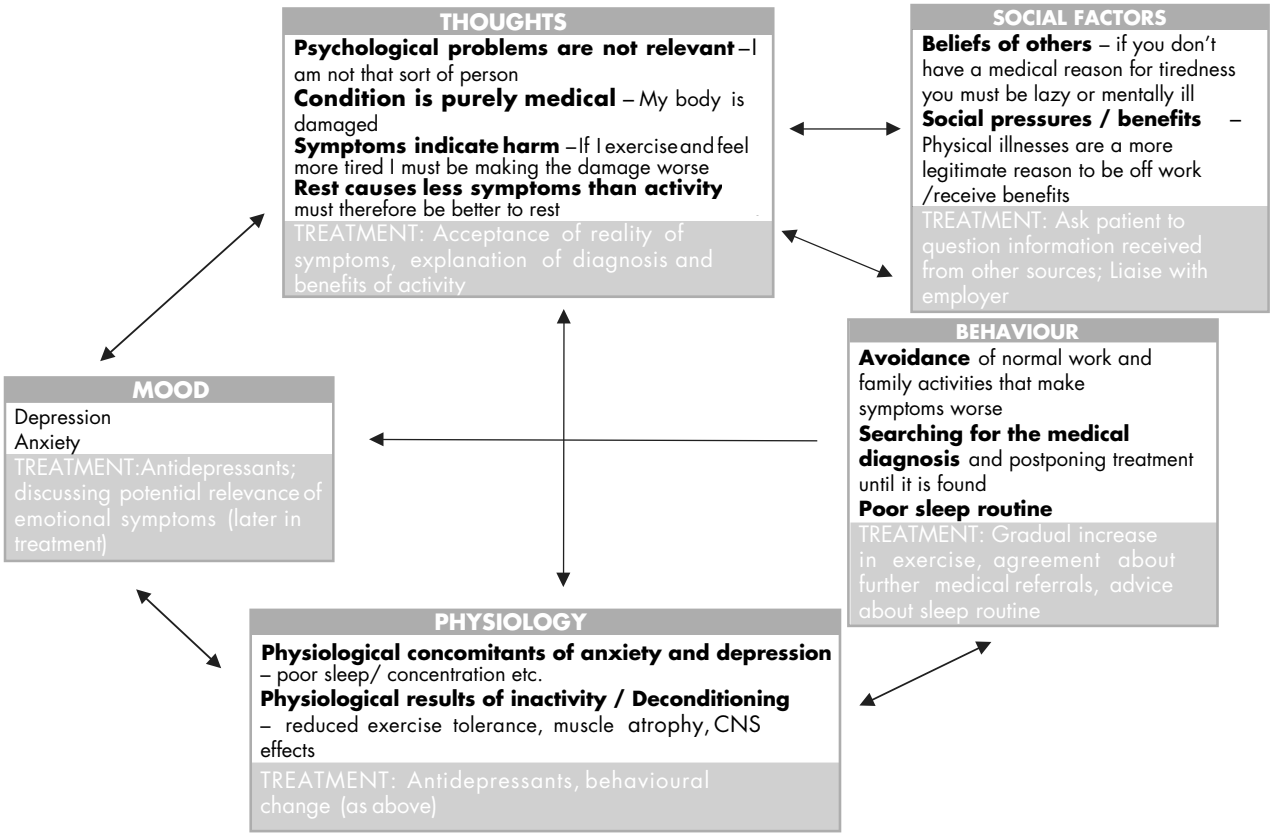



**Figure 4** A model of perpetuating factors in functional symptoms (in this example, fatigue) illustrating some targets for treatment with cognitive behavioural therapy and antidepressants.

at the start of our first article. The following are some of the elements of CBT that you could incorporate into the consultation:

- Accept all symptoms at face value and give a positive explanation.
- Persuade the patient that change is possible, they are not ''damaged'', and they do have the potential to recover.

**Figure 5** Faking injury. Rivaldo, the Brazilian football player, was fined £5180, half a day's wages, for ''faking'' an injury after a Turkish player kicked the ball at his leg and he collapsed clutching his face. The Turkish player was sent off and Brazil went on to win the 2002 world cup.

- Give the patient a rationale for treatment—for example, exercise will help recondition your muscles and ''tune up'' your nervous system.
- Encourage activity, warning the patient that they may feel temporarily worse afterwards but that there will be benefits in the long term. Warn against ''over doing it'' on good days as much as ''under doing it'' on bad days. Explain that symptoms tend to be cyclical anyway and predict days when they will feel ''back to square one''. Start with small goals such as walking around the garden once and build up.
- Establish a sleep routine. Give simple advice such as avoiding sleep during the day, getting up at a specified time, and getting out of bed for 15 minutes rather than lying worrying awake at night.
- Encourage the patient to reconsider unhelpful and negative thoughts. For example, patients with pain commonly think ''If I go for a walk I'll feel much worse afterwards and I'll make the 'wear and tear' that my GP told me about worse''. Instead the patient should be encouraged to consider and test out an alternative possibility such as ''If I go for that walk, I will be sore afterwards for a bit but in fact it will be strengthening my bones and muscles not making them worse and it will help build my confidence''.
- Look for modifiable physiological/dissociative trigger factors. If you can find evidence of dissociative or autonomic symptoms before symptom onset this may provide an additional extra rationale for this type of treatment. For example, if a patient with non-epileptic

### Case 1: A twisted ankle that remained twisted

A 32 year old man was admitted for investigation of an abnormal foot posture. Twelve months earlier he had been in a car accident when he had twisted his right ankle. Although there was no head injury he only has a hazy memory of the accident and remembers feeling spaced out and shocked that he had crashed. There was no bony injury but instead of making a normal recovery his ankle gradually inverted until it remained in a fixed position (fig 6). In addition he complained of a lot of back pain and had gradually developed weakness of the whole leg. On questioning he said he felt constantly exhausted and had trouble sleeping. There was a history of recurrent groin pain which had not been diagnosed but had resolved. He was taking dihydrocodeine which only ''took the edge off'' his back pain. He had been working long hours as a van driver and said that he really enjoyed his job which he had been unable to do since the accident. He said he was not depressed but he found it hard to describe anything he enjoyed doing. His family had seen a big change in his personality. They said his ankle remained like that when he was asleep. There was no compensation case.

On examination he looked miserable. He had collapsing weakness of all the muscles in his right arm and leg and Hoover's sign was strongly positive. The ankle was in a fixed inverted position. Attempts to straighten it produced irregular shaking in the whole leg. Vibration sense and light touch were altered over the whole of the right side of his body including his face. Magnetic resonance imaging, neurophysiology, and other tests were normal.

**What would you do? How would you explain the diagnosis?**

**What kinds of treatment might help?**

*Read the outcome of case 1 at the end of the article*

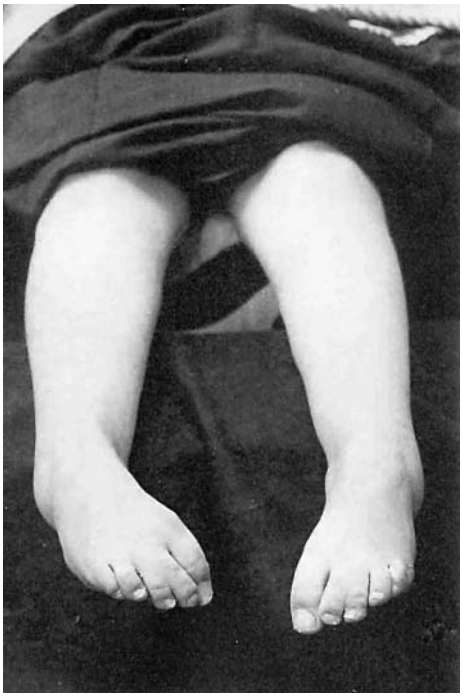


**Figure 6** This photograph shows a patient from a 19th century textbook with bilateral fixed ankle inversion, similar to the unilateral ankle inversion described in case 1.

attacks discloses such symptoms a CBT approach similar to that used for panic (in which the patient is encouraged to be aware of ''catastrophic'' interpretations of symptoms and to consider more benign ones) can be used.[6] In our experience, patients with paralysis, who also often report these symptoms just before onset and can have a relapsing course, can be helped by explaining the link between these ''warning symptoms'' and their weakness.

- Looking at obstacles to recovery. Rather than focus on possible ''causes'' from the past such as an unhappy childhood, focus on obstacles to recovery. These may include the hated job which they will have to return to, the legal case that may not settle for five years, and the benefit trap. It still surprises us how often these issues can discussed openly and frankly and the patient encouraged to actively address them once you have won the patient's trust.
- Provide information about more detailed written material. There is so far little available but a self-help book on fatigue[7] can help the patient get started by themselves. If you think they'll accept it there are plenty of CBT based self help books for depression and anxiety.[8]
- Further elements of treatment could involve spending more time discussing these explanations with key family members, discussing with the patient how to explain their improvement to other people, and negotiating a phased return to work.

As a final stage it may be possible to start making links between symptoms and stressful events—for example, ''When you had that terrible day what were you thinking about—do you think that was relevant?''. This final stage is often the one that many doctors think should be at the ''front end'' of treatment, not appreciating that it can be one of the most difficult to pull off and, in our experience, not always essential in order to make a recovery.

Reading this you may think ''This is impossible to fit in to my busy neurology clinic''. But if you are in the habit of providing constructive explanations for symptoms and some pointers about what to do about them, you are probably already practising a CBT approach.

## ANTIDEPRESSANTS AND OTHER DRUGS

Antidepressants can help many patients with functional symptoms, even those who are not depressed. A recent systematic review of antidepressant treatment in patients with a range of functional symptoms found a number needed to treat of only three (comparing favourably with many treatments in neurology).[9] In practice, their use may be difficult because of: (1) psychiatric stigma; (2) a perception that they are addictive or harmful; and (3) side effects. All these therefore need to be discussed before prescribing—for example, saying to the patient: ''So-called antidepressants often help these symptoms even in patients who are not feeling depressed. They have wider actions than treating depression—for example on pain, sleep, and appetite—and can reverse the abnormalities in brain function we have talked about''.

The patient is best warned that they will probably experience side effects but that these will tend to wear off eventually so the treatment should be persisted with for at

### Case 2: Uncontrollable shaking

A 24 year old auxiliary nurse working in a care home for the learning disabled was admitted with a three month history of shaking attacks. These were occurring up to five times a day and lasting between 2–10 minutes (fig 7). She initially did not report any prodromal symptoms but on direct questioning admitted to strong and frightening depersonalisation and a warm feeling in the minute or so before some attacks. During some of the attacks she reported being able to hear people around her but being unable to respond. She had been getting the feeling that ward staff thought she was ''making it up''. She sometimes slept after the attack, but could be tearful. Otherwise she reported exhaustion, poor sleep, and poor concentration. She had a history of chronic pelvic pain and laparoscopic surgery. She was on anticonvulsants from a different hospital and had been told that she had epilepsy. Her case notes documented a history of childhood sexual abuse and previous contact with psychiatrists for depression. She angrily denied feeling low or anxious or any recent life stresses. She said that although her job could be challenging it was also very rewarding and she was upset to be off work. She had just left the parental home to live with her boyfriend. Examination was normal. During a witnessed attack, her eyes were observed to be shut, she had an increasingly fast respiratory rate, and limb movements were asynchronous. EEG during one of the attacks was normal.

**How are you going to explain the diagnosis?**

**How are you going to handle the issue of her history of sexual abuse?**

**What would you do to help?**

*Read the outcome of case 2 at the end of the article*

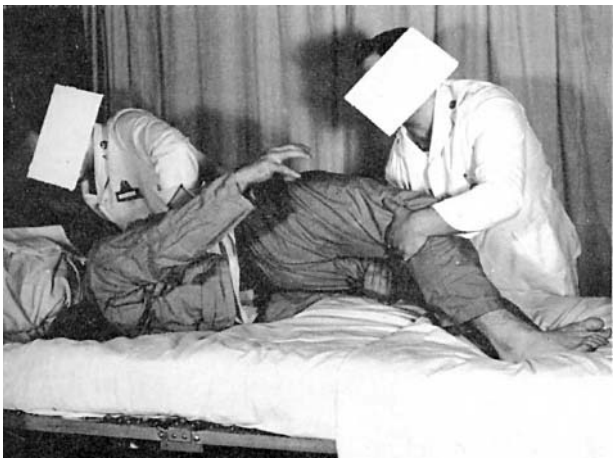


**Figure 7** This US army photograph shows a man being restrained during a non-epileptic attack with back arching, similar to that described in case 2.

least four weeks. They should also be warned not to expect any benefit until this time.

There is not much evidence to guide us in the choice of antidepressant agent. Tricyclic antidepressants are particularly helpful in patients with insomnia and pain but can cause unacceptable drowsiness and a dry mouth. Selective serotonin reuptake inhibitors (SSRIs) and other more selective drugs have similar efficacy and are probably preferable in the medically unwell and elderly, but also have side effects such as nausea. They also may not be so good at treating pain. Other drugs used in chronic pain such as gabapentin should also be considered. Antidepressants do not help the symptom of depersonalisation. We generally explain to patients that they can get better without the tablets—which is true—but that they are worth trying if they want to ''explore every therapeutic avenue''.

## OTHER MANAGEMENT TECHNIQUES

### Hypnosis and intravenous sedation

There is some evidence for the use of hypnosis in patients with functional motor symptoms. Alternatively, *examination* under sedation can be used therapeutically to demonstrate to the neurologist, and by means of video to the patient, that an apparently paralysed limb can move or a fixed dystonic foot is not fixed. These methods merit more systematic study.

### Psychodynamic and other types of psychotherapy

In classical psychodynamic theory, conversion disorder implies that distress resulting from intolerable mental conflict is converted in to a somatic symptom with consequent relief of distress. Another facet of this theory is symbolism—for example, where a symptom like a pseudo-seizure may be said to represent a symbolic re-enactment of childhood sexual abuse. Symbolism is hard to test but the evidence overall is not consistent with ''conversion''. For example, the more somatic symptoms a patient has the *more* emotional symptoms they will also tend to have. In addition, in our experience, when a patient looks as if they are not distressed, they often are— they just do not want to tell you about it.

More recent psychodynamic theory has moved on from the ideas above and instead highlights the importance of early relationships and their effect on the relationships people form as adults. For example, it is plausible that poor parenting could produce interpersonal dependency in adulthood. If this dependency and excessive ''attachment behaviour'' was to a doctor or a family member only interested in physical problems you can begin to see how a tendency to repeatedly present somatic complaints might develop. Adverse experience in childhood may also influence the person's tendency to develop certain symptoms. For example, chronic pelvic pain complaints are more common in women who have been sexually abused.

In non-specialist practice awareness of these factors may help to make patients' otherwise inexplicable symptoms more understandable. But they are hard to make use of in treatment. For some patients a more in depth psychotherapy that helps the patient to make links with these antecedents of symptoms may be of value, but has not been evaluated in randomised trials.

## WHICH PATIENTS SHOULD BE REFERRED TO A PSYCHIATRIST?

Remember that if you sent everyone you see with functional symptoms to a psychiatrist, you will be sending one third of your neurology clinic to them. Patients with mild symptoms, symptoms that respond very positively to the initial explanation, or those with a good GP will probably not need (or accept) the complication of another referral. However, a patient who is unimproved after a receiving a careful explanation, a trial of antidepressants and physiotherapy should probably be referred. A patient who has previously not been helped by experienced psychiatric intervention is

probably unlikely to benefit. If you do need to refer to a psychiatrist it is often easier to do this at a second appointment. Ideally, the psychiatrist will have be interested and experienced in managing patients with somatic symptoms. A liaison or neuropsychiatrist, if available, is often best.

## DIFFICULT MANAGEMENT ISSUES

### Litigation

There is no doubt that simulation in order to make money (fig 5) does occur (as discussed in more detail below). Furthermore, very few of us would refuse the opportunity to make some money if it was suggested we might be entitled to it. Leaving aside the financial motivation, compensation is a potent obstacle to recovery for all patients regardless of whether their symptoms are functional or not. Seeking compensation implicitly involves a commitment to: (1) the idea that there is physical injury/damage; (2) the notion that someone else is to blame for the symptoms now and in the past; and (3) a desire on the patients part to ''prove'' that they really do have the symptom. Many patients with functional symptoms who are *not* seeking compensation share these feelings about their symptoms, often because they feel no-one believes them and they are being accused of imagining their symptoms. It is hardly surprising then that in the context of an injury many should get involved with a legal process that is prepared to back them up and get them some money in the process. These complex issues and the negative role that lawyers and doctors can play in this process are discussed in a highly readable if polemical book, W*hiplash and other useful illnesses*.[10]

### Disability and incapacity benefits

Should a patient with functional symptoms receive disability benefits? Such benefits can be substantial, be more than previous earnings, and can lead to a situation where a patient will lose money if they get better. This dilemma may be usefully discussed openly with the patient. Remember that there is little research to support the idea that secondary gain is a greater factor in patients with functional symptoms than in those with disease.

### Aids and appliances

A similar dilemma arises when thinking about aids such as sticks and wheelchairs. They can be both helpful, in improving independence and confidence, and harmful, leading to dependence on them and decreased activity. Each case must be evaluated on it merits.

## THE PATIENT WHO DOES NOT GET BETTER

Many patients with functional symptoms are hard to treat and follow a lifelong course of symptoms, disability, and medical consultations. It is important to have realistic expectations about who can be helped and to accept that you may have to treat several to make a big difference to one. If you have made an effort with a particular patient but it is clear that they do not really believe that things can change (or you do not have any more resources to help) then this may be the point at which to ask the GP to take over their long term chronic care. Rather then ending contact on a negative note you may wish to tell the patients that they are coping well with a difficult illness and that you are sorry you have not been able to help more.

If the patient has a history of repeated presentation to secondary care with the associated risk of iatrogenic harm, a positive plan to ''contain'' the patient in primary care may help—for example, by the GP making regular monthly appointments regardless of whether there are new symptoms or not. This in itself may reduce the number of new symptoms and will enable more optimal management of things like recurrent depression, but the GP may still need to ask you to review the patient from time to time.

## PROGNOSIS

### Symptomatic recovery and other measures of outcome

The natural history of functional symptoms in neurology has not been well described. In outpatients, a third to a half of patients can be expected to be unchanged or worse a year after diagnosis. Symptom persistence is more likely for those with motor symptoms or pseudoseizures than just sensory symptoms. Some patients will develop other functional symptoms and attend multiple medical specialities; iatrogenic harm from unwarranted surgery and drugs is a major problem. Known predictors of poor outcome are long symptom duration and personality disorder.[3]

### Misdiagnosis

Since an influential paper by Slater in the 1960s, many doctors have been worried that a high proportion of patients with functional symptoms, like paralysis and non-epileptic attacks, will go on to develop disease that with hindsight explains their symptoms. In fact, a number of recent studies have reported rates of misdiagnosis of around 5% in regional and tertiary neurological centres.[3 11] This misdiagnosis rate is similar to that of other neurological disorders. In practice this means that occasionally you will get the diagnosis wrong and so you should be willing to re-evaluate the patient. When misdiagnosis does occur, it is most common in gait and movement disorder and where the clinician has placed too much emphasis on a bizarre or ''psychiatric'' presentation.

## MALINGERING AND FACTITIOUS DISORDER: IS THE PATIENT MAKING IT UP?

Discriminating between consciously produced and unconsciously produced functional symptoms is difficult, if not impossible. Patients' awareness of control over a symptom like paralysis is probably not ''all or nothing'' but rather on a continuum. It may also vary over time so that a patient may begin an illness with little awareness about what is happening but gradually gain a degree of conscious control with time (or vice versa).

Doctors are almost certainly worse at detecting deception by a patient than we would like to think. A recent study where examiners were blind to whether subjects were feigning paralysis or genuinely experiencing it during a hypnotic state showed no greater than chance performance. As Miller put it, the detection of malingering is ''nothing more infallible than one man's assessment of what is probably going on in another man's mind''. The only investigations that reliably tell you that someone is malingering are covert surveillance demonstrating a *major discrepancy* in function or a direct confession, but these are rarely obtained outside medicolegal scenarios. Functional imaging of the brain is opening up new possibilities of detecting differences in intention and action that are not visible clinically, but these remain experimental and may never be reliable. When conscious intention is discovered or confessed, a distinction must be made between those patients generating symptoms or behaviour merely to gain ''medical

### Case 1: Outcome

**What would you do? How would you explain the diagnosis?**
The clinical features were consistent with a functional dystonia associated with back pain, fatigue, and functional right sided weakness. However, the dystonia part of the diagnosis was left as ''probably functional'' initially. We were also concerned about the possibility of a contracture. We opted to treat the other aspects of his illness first to see what would happen. He was given a positive explanation for the symptoms and shown a video of another patient with the same problem. We explained the role of physiotherapy and of antidepressants (even though we agreed that he was not feeling depressed). As a result of this he fairly quickly felt confident about the diagnosis and his back pain, fatigue, and weakness began to slowly improve. He started to make plans for the future and discussed changing his job if and when he could recover. He admitted that he had been feeling very frustrated and low in recent months. His foot, however, remained stubbornly inverted.

**What kinds of treatment would you attempt?**
Six months later, in the absence of any improvement in his ankle posture, he was brought in for videotaped sedation. In a highly clinical environment he was given a titrated dose of propofol so that he was awake but drowsy. In this state he was able to move his ankle normally for the first time in one and a half years. He was also able to watch the video afterwards. A few days later there was a ''breakthrough'' in his treatment and his foot straightened and started moving normally. He now is back at work and walking normally. Although he is not completely asymptomatic he is very grateful for his recovery. Asked what had made the difference he said he was terrified that something in his medial ankle would ''snap'' if it ever came back to a normal position. We made a diagnosis of definite functional dystonia.

### Case 2: Outcome

**How are you going to explain the diagnosis?**
The history and investigations are typical for non-epileptic attacks. This is exactly the kind of patient in whom it can be counterproductive to wade in with a psychological explanation. She has angrily denied any emotional symptoms and has already been given a diagnosis of epilepsy, a valid diagnosis that may be difficult to take away. Non-epileptic attacks can be explained as a common and treatable temporary ''short circuit'' of the nervous system but without the abnormal electrical activity of epilepsy. Repeated reminders that you do not think she is making the symptoms up will probably be necessary. Written information including a leaflet and a copy of your letter will help confirm to the patient and her family that this is a real diagnosis and that you are not just waffling.

**How are you going to handle the issue of childhood sexual abuse?**
It is usually inadvisable to make connections between remote events such as this and current symptoms unless you have established a good relationship with the patient over a period of time. Even if you think there is a connection between her abuse, her pelvic pain, and the non-epileptic attacks—and there may be—the patient is unlikely to want to hear your thoughts on the matter or be helped by them, at least for some time.

**What would you do to help?**
The most important first step is to undo the diagnosis of epilepsy and make a clear and unequivocal diagnosis of non-epileptic attacks. Her anticonvulsants should be stopped. The presence of prodromal symptoms is helpful. Explain that depersonalisation is common and not life threatening. Ask her to try rebreathing into a paper bag over her mouth and nose the next time she feels it coming on. Explain how patients can learn to stop attacks like these in time with practice. Make a careful referral to a liaison psychiatrist explaining you don't think she's crazy, but Dr X has special expertise in these kinds of symptoms. If you manage to get her to turn up for the appointment the psychiatrist ought to be able to engage her, begin to explore psychological aspects, and monitor progress. Ideally the neurologist should make sure the patient believes/understands the diagnosis at follow up as well.

care'' (factitious disorder) and those who simulate for financial or other material gain (malingerers).[12]

A bad experience of discovering that you have been ''deceived'' by a patient may lead to cynicism about all patients, since the true proportion of patients who malinger is unknown. When you feel like this it may be useful to ask yourself the following questions: Why do so many patients present such similar stories of bafflement and fear about their symptoms? Why do follow up studies show persistence of symptoms in the majority in the long term? Why are patients with these symptoms so keen to have investigations to ''hunt down'' an organic cause for their symptoms?—if they knew they were malingerers they would know that this could weaken their case. When you see a patient clearly exaggerating a symptom or groaning heavily during an examination, is this exaggeration to deceive or exaggeration to convince? Finally, you may want to ask yourself—is it your job to detect malingering anyway since it is not a medical diagnosis but a moral problem?

## CONCLUSION

Early management of functional symptoms involves demonstrating to the patient that you believe them and that you recognise their symptoms as being common and potentially reversible. A lot more research is needed in to the optimum approach but our experience is that using the ''functional model'' of symptom generation allows a transparent explanation and interaction with the patient that can facilitate later physical and psychological treatments. Much of the core of a cognitive behavioural approach to treatment is in fact simple advice about exercise, sleep, and ways about thinking about symptoms that can be given effectively by a neurologist. While it is unreasonable to expect everyone to get better, it is also a mistake to think that a neurologist cannot make a difference, even in a limited time.

## ACKNOWLEDGEMENTS

We would like to thank Dr Adam Zeman and Professor Charles Warlow for their valuable collaboration and support of this work.

Thanks also to Will Whitely, Rustam Al-Shahi, Malcolm McLeod, and Patrick Fox for comments on the manuscript.

## Authors' affiliations

**J Stone, A Carson, M Sharpe,** NHS Department of Clinical Neurosciences, Western General Hospital, and School of Molecular and Clinical Medicine, University of Edinburgh, Edinburgh, UK

## REFERENCES

1 **Vuilleumier P**, Chicherio C, Assal F, *et al*. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain* 2001;**124**:1077–90.
► **The most convincing functional imaging study of patients with functional motor and sensory symptoms.**
2 **Sharpe M**, Carson A. ''Unexplained'' somatic symptoms, functional syndromes, and somatization: do we need a paradigm shift? *Ann Intern Med* 2001;**134**:926–30.
► **Why symptoms unexplained by disease should be remedicalised and not seen as just psychiatric.**
3 **Halligan P**, Bass C, Marshall JC. *Contemporary approaches to the science of hysteria: clinical and theoretical perspectives*. Oxford: Oxford University Press, 2001.
► **The most comprehensive book available on this subject with a wide variety of approaches including chapters on physical and cognitive behavioural treatment, neuroimaging, history, and prognosis.**
4 **Mayou RA**, Bass C, Sharpe M. *Treatment of functional somatic symptoms*. Oxford: Oxford University Press, 1995.
► **One of the very few books dedicated to this subject.**
5 **Kroenke K**, Swindle R. Cognitive-behavioral therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. *Psychother Psychosom* 2000;**69**:205–15.
► **A meta-analysis of all CBT trials for functional somatic symptoms and syndromes.**
6 **Goldstein LH**, Deale AC, Mitchell-O'Malley SJ, *et al*. An evaluation of cognitive behavioral therapy as a treatment for dissociative seizures: a pilot study. *Cogn Behav Neurol* 2004;**17**:41–9.
► **Describing promising results with CBT for non-epileptic attacks. Includes a detailed description of the treatment given.**
7 **Sharpe M**, Campling F. *Chronic fatigue syndrome (CFS/ME): the facts*. Oxford: Oxford University Press, 2000.
► **A self help book for people with persistent fatigue written with a patient. Consistent with methods described in this article. One to recommend to patients!**
8 **Butler G**, Hope T. *Manage your mind: the mental fitness guide*. Oxford: Oxford University Press, 1995.
► **A self help book based on CBT principles. Suitable only for patients willing to accept a diagnosis of depression or anxiety.**
9 **O'Malley PG**, Jackson JL, Santoro J, *et al*. Antidepressant therapy for unexplained symptoms and symptom syndromes. *J Fam Pract* 1999;**48**:980–90.
► **Systematic review providing evidence for the use of antidepressants for functional symptoms.**
10 **Malleson A**. *Whiplash and other useful illnesses*. Montreal and Kingston: McGill-Queen's University Press, 2002.
► **Entertainingly written although sometimes polemical look at the medicolegal industry, the development of ''new illnesses'', and the effect all this has had on symptom reporting and disability.**
11 **Crimlisk HL**, Bhatia K, Cope H, *et al*. Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. *BMJ* 1998;**316**:582–6.
► **Drives a nail in the coffin of studies from the 1960s that said neurologists often got the diagnosis of functional motor and sensory symptoms wrong.**
12 **Halligan PW**, Bass C, Oakley DA. *Malingering and illness deception*. Oxford: Oxford University Press, 2003.
► **The most comprehensive text on malingering with a wide perspective of contributors.**
13 **Stone J**, Wojcik W, Durrance D, *et al*. What should we say to patients with symptoms unexplained by disease? The ''number needed to offend''. *BMJ* 2002;**325**:1449–50.

7. Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry 2005; 76 (suppl I): i2-i12.

# FUNCTIONAL SYMPTOMS AND SIGNS IN NEUROLOGY: ASSESSMENT AND DIAGNOSIS

J Stone, A Carson, M Sharpe

*J Neurol Neurosurg Psychiatry* 2005;**76**(Suppl I):i2–i12. doi: 10.1136/jnnp.2004.061655

It's a Tuesday morning at 11.30 am. You are already 45 minutes behind. A 35 year old woman is referred to your neurology clinic with a nine month history of fatigue, dizziness, back pain, left sided weakness, and reduced mobility. Her general practitioner documents a hysterectomy at the age of 25, subsequent division of adhesions for abdominal pain, irritable bowel syndrome, and asthma. She is no longer able to work as a care assistant and rarely leaves the house. Her GP has found some asymmetrical weakness in her legs and wonders if she may have developed multiple sclerosis. She looks unhappy but becomes angry when you ask her whether she is depressed. On examination you note intermittency of effort and clear inconsistency between her ability to walk and examination on the bed. She has already had extensive normal investigations. The patient and her husband want you to ''do something''. As you start explaining that there's no evidence of anything serious and that you think it's a psychological problem, the consultation goes from bad to worse….

In this article we summarise an approach to the assessment and diagnosis of functional symptoms in neurology, paying attention to those symptoms that are particularly ''neurological'', such as paralysis and epileptic-like attacks. In the second of the two articles we describe our approach to the management of functional symptoms bearing in mind the time constraints experienced by a typical neurologist. We also address difficult questions such as: ''What causes functional symptoms?'', ''Are they real?'', and ''Is there anything that can be done?''

We emphasise the need for a transparent and collaborative approach. As we will explain this depends on giving up a purely ''psychological'' view of functional symptoms in favour of a biopsychosocial view of causation in which dysfunction of the nervous system is the final common pathway.

## SYMPTOMS AND DISEASE

It is important to keep in mind the difference between symptoms and disease. Symptoms, like fatigue, are the patient's subjective experience. Doctors are trained to find a disease, such as multiple sclerosis, to explain the symptoms. When there is no disease it becomes tempting to suggest that the symptom must be ''not real'' or psychogenic. In fact, symptoms appear for multiple reasons of which disease is only one (fig 1). Symptoms arise from physiological factors (for example, physiological tremor), psychological factors (for example, paraesthesia during a panic attack), behaviours (for example, excessive rest), and cultural or external factors (for example, compensation and the welfare state). For some patients disease pathology is a major (but not the only) factor in causing symptoms and in others it is minor or absent entirely.

A crucial implication of this approach is that the patient does not have to have a ''genuine'' disease in order to have a ''genuine'' symptom.

### WHAT SHOULD WE CALL THEM?

The large number of terms to describe symptoms unexplained by disease is a reflection of the diverse concepts that have been used to understand them. They include:

- Pure symptomatic labels (for example, chronic fatigue, low back pain)
- Symptom syndromes (for example, chronic fatigue syndrome)
- ''Non-diagnoses'' that describe what the diagnosis is not rather than what it is (for example, non-epileptic attacks, non-organic, medically unexplained)
- Diagnoses that imply an as yet unestablished disease cause (for example, reflex sympathetic dystrophy)
- Diagnoses that imply an as yet unestablished psychological cause (psychogenic, psychosomatic, ''all in your mind'')
- Historic diagnoses that do not fit in to any of these categories (for example, ''hysteria'', ''functional'')
- ''Official'' psychiatric diagnoses. These are found in psychiatric glossaries which are rarely used by neurologists and include:

See end of article for authors' affiliations

Correspondence to: Dr Jon Stone, Department of Clinical Neurosciences, Western General Hospital, Crewe Road, Edinburgh EH4 2XU, UK; Jon.Stone@ed.ac.uk

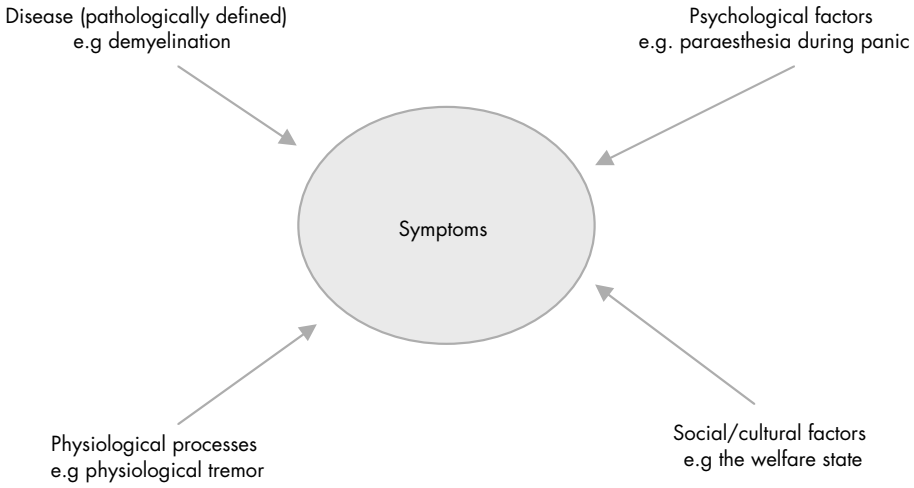



**Figure 1** Disease is only one cause of symptoms.

- conversion disorder: a psychoanalytic concept that describes the occurrence of motor or sensory neurological symptoms other than pain and fatigue that cause distress, are not explained by disease, not malingered but are thought to relate to psychological factors
- somatisation disorder (Briquet's syndrome): refers to patients with lifelong functional symptoms including pain, neurological, gastrointestinal and sexual symptoms, again with the implication that psychological problems have been somatised or converted
- dissociative motor disorder: in which dissociation (or a failure of integration of psychological processes) is the putative mechanism
- hypochondriasis: a distressing state of anxiety about disease
- factitious disorder: symptoms consciously simulated in order to gain medical care
- malingering: a term (and not a medical diagnosis) for symptoms which are simulated for clear financial or material gain.

The terminology you use is important. It will not only reflect how you think about the problem, but will also determine the patients' reaction to your diagnosis. For reasons that we explain in the second article we prefer the term ''functional'' and will use it in these articles.

## HOW COMMON ARE FUNCTIONAL SYMPTOMS?

Around one third of new neurological outpatients have symptoms regarded by neurologists as ''not at all'' or only ''somewhat'' explained by disease.[1] This finding is not unique to neurology and has also been reported in primary and secondary medical care worldwide. Table 1 illustrates some of the different functional somatic symptoms and syndromes that have been described by various medical specialties. Although superficially disparate, there is substantive overlap in the symptoms, epidemiology, and response to treatments of these functional somatic syndromes.[2]

Contrary to popular belief, even the more dramatic functional symptoms are surprisingly common. The incidence of functional paralysis is probably similar to that of multiple sclerosis (around 5/100 000). Non-epileptic attacks make up around 10–20% of the patients referred to specialist epilepsy clinics with intractable seizures and up to 50% of patients admitted to hospital in apparent status epilepticus.[3] In movement disorders clinics up to 5% of new referrals may have functional symptoms.[4]

## FUNCTIONAL SYMPTOMS: WHY BOTHER?

When faced with a clinic full of patients with epilepsy and multiple sclerosis, many neurologists cannot help thinking that patients with functional symptoms should be at the bottom of their priorities. There are a variety of views. Many doctors believe that patients often exaggerate or make up their symptoms in order to gain sympathy or financial benefit. Alternatively, some doctors believe the patients' symptoms, but simply view the problem as ''not neurological'' and one that should be dealt with by a psychiatrist and not a neurologist.

One argument relates to the patients themselves. When patients with functional neurological symptoms are compared to those whose symptoms are associated with disease, they are found to have similar disability and even more distress.[1] Their symptoms tend to persist at follow up but only rarely become explained by disease. Distress and disability are by their nature subjective but we argue that ultimately it is the subjective that matters most.

The second argument relates to the work of a neurologist. Whether you like it or not, functional symptoms account for one third of your workload. If you allow yourself to become interested in the problem rather than irritated by it, you may find it has an effect on how much you enjoy your job generally.

**Table 1** Examples of ''functional'' somatic symptoms and syndromes from different medical specialities

| | |
|---|---|
| ► Neurology | Functional weakness, non-epileptic attacks, hemisensory symptoms |
| ► Gastroenterology | Irritable bowel syndrome, non-ulcer dyspepsia, chronic abdominal pain |
| ► Gynaecology | Chronic pelvic pain, premenstrual syndrome |
| ► ENT | Functional dysphonia, globus pharynges |
| ► Cardiology | Atypical chest pain, unexplained palpitations |
| ► Rheumatology | Fibromyalgia |
| ► Infectious diseases | (Post-viral) chronic fatigue syndrome |
| ► Immunology/allergy | Multiple chemical sensitivity syndrome |

## TAKING A HISTORY FROM SOMEONE WITH FUNCTIONAL SYMPTOMS: A PRACTICAL APPROACH

If you suspect that a patient's presenting symptoms are functional, there are ways of adapting the history to make it more efficient, more interesting for you, and more helpful for the patient. For the patient with functional symptoms, a good assessment is also the beginning of treatment.

### ''Drain the symptoms dry''

If your patient has a lot of symptoms, begin by making a list of all of them. Here is where you can save most time by resisting the urge to interrogate the features and onset of every symptom as you go. Instead, leave a few lines between each symptom on the list so that you can return to them as required. This allows the patient to unburden themselves quickly of all their symptoms, gives you a broad picture early on, and prevents new symptoms ''cropping up'' late in the consultation. Fatigue, sleep disturbance, memory and concentration problems, and pain can be routinely enquired about at this stage. However, for reasons we will explain, questions about mood are often better left to later.

The more physical symptoms a patient presents with the more likely it is that the primary presenting symptom will not be explained by disease.[2] A long list of symptoms should therefore be a ''red flag'' that the main symptom is functional.

### Asking about disability

Ask the patient to describe ''What's a typical day like?''. Follow up questions such as ''How much of the day do you spend in bed?'' and ''How often do you leave the house?'' are more useful than the traditional disability questions about dressing and walking distance. Pay particular attention to *why* they are disabled—for example, someone may have a very mild hemiparesis which really does not impair gait but be very worried about falling which is why they do not go outside.

### Finding out more about onset and course

Although you may want to take a detailed history of the course of some symptoms, if a patient has had the symptoms for many years it may be more useful to obtain the overall course of the illness by drawing a graph with time on the x axis and severity on the y axis (fig 2). This can be a quick way of condensing a large amount of information— the line of the graph demonstrates how the illness has gradually worsened, cycled, or perhaps just been static over the period in question. To find the starting point, a useful question is ''When did you last feel well?''. Other events can then be added using arrows—for example, to indicate when the patient stopped working, life events, or medical interventions.

### Asking about dissociation

Dissociative symptoms include depersonalisation (feeling detached from oneself) and derealisation (feeling that the world is no longer real) and can be unfamiliar territory for neurologists. However, they commonly occur in patients with neurological disease (such as epilepsy and migraine), in patients with functional symptoms, particularly those with paralysis and non-epileptic attacks, and less commonly in healthy individuals. People find it difficult to describe dissociation and may just say they felt ''dizzy''. The following descriptions give an indication of what sort of thing to look for:

- ''I felt as if I was there, but not there, as if I was outside of myself''
- ''I was spaced out, in a place all of my own''
- ''Things around me didn't feel real, it was like I was watching everything on television''
- ''My body didn't feel like my own''
- ''I couldn't see but I could hear everyone, I just couldn't reply''.

Dissociative symptoms are not diagnostic of a functional problem, but are worth looking for, particularly in patients with functional paralysis or non-epileptic attacks, because:

- they are frightening to patients who are often relieved to discover that the symptom is common and does not indicate ''madness''
- where there is dissociation, there is a reasonable chance of finding that the patient has panic attacks (episodic severe anxiety)
- they can offer an extra way of explaining to patients the link between their experiences and the development of unusual symptoms such as a limb that no longer feels as if its part of them.

### What happened with previous doctors?

Ask your patient to tell you about doctors who they saw previously. They may complain bitterly about Dr X or Y who ''didn't listen'' to them or who told them it was ''nothing serious''. You do not need to say whether you agree with Dr X or Y but hearing about this serves two important purposes. Firstly, it can warn you about explanations and treatments that are likely to be rejected. Secondly, by letting the patient talk openly about previous disappointing medical encounters you are showing them that you are interested in their suffering and understand their frustration.

### Asking about illness beliefs

What does the patient think is causing their symptoms? What do they think should be done about them? Do they think they are irreversible or reversible? There is evidence that patients with functional neurological symptoms are *more* likely to be convinced that their symptoms are caused by disease than patients whose symptoms are actually caused mainly by disease—perhaps because they are trying to convince others that their symptoms are ''real''. These

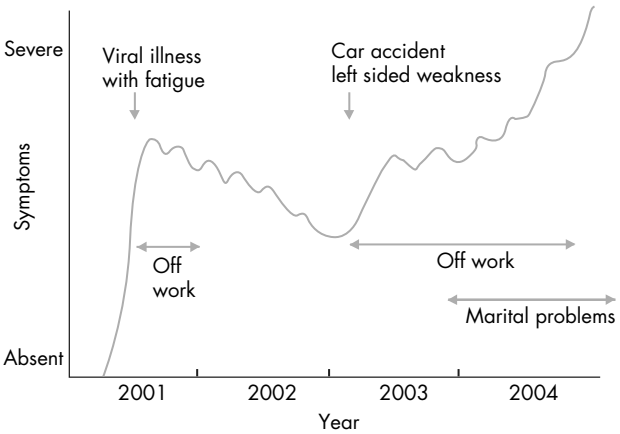



**Figure 2** Using a graph to take a history from a patient with functional symptoms.

questions also guide the final explanation. For example, if a patient expresses fear that their symptoms are due to multiple sclerosis a specific explanation of why this is not the case will be needed.

### Past medical history: ''get the notes''

Apart from the overall number of symptoms, the other general *diagnostic* red flag is whether there is a history of previous functional symptoms (table 1). The more functional symptoms they have had in the past, the more likely it is that the current symptom is also functional.[2] This reflects the fact that some people are more prone to developing symptoms than others, for reasons we will discuss later. There may also be a history of medical attempts to treat these symptoms with surgical operations (for example, hysterectomy at a young age, appendicectomy, laparoscopy to investigate abdominal pain). Patients may have forgotten previous problems or they may just sense that the more they tell you about previous medical encounters that ended in no diagnosis, the less likely you are to take their current symptoms seriously. That is why you need the notes. Previous psychiatric diagnoses may be particularly unforthcoming in the history. If the patient already has a neurological or other disease diagnosis, ask yourself if the evidence recorded in the notes justifies it—it may not. Alternatively, they may have a disease but one which is insufficient to explain the current symptoms.

### Social history: work, money, the law, and marriage

An unpleasant job, being in a ''benefit trap'' (where money received on benefits is comparable to that earned at work), and involvement in a legal case should not be seized on as ''the cause'' of symptoms. This is just another form of unhelpful oversimplification. They could, however, be highly relevant obstacles to recovery. For example, patients with motor symptoms who got married or divorced have been found to do better than those patients whose marital status does not change.

### Modelling

A history of similar illness in friends or family or contact with illness through work may lead to another simplistic explanation that the patient is copying or ''modelling'' their symptoms on others. Although plausible, there is little evidence to support or refute the idea that this occurs.

### Asking about emotional symptoms: go carefully

Depression, anxiety, and panic are more common in patients with functional symptoms than those with disease. However, asking about psychological symptoms in the wrong way can make the patient defensive because they think that you are about to dismiss them as ''psychiatric''. We therefore suggest that you:

- make sure you have already asked about all the associated ''somatic'' symptoms first—for example, fatigue, poor concentration, poor sleep
- leave questions about emotions until the end of the history
- when you do ask, frame the question in terms of the symptom they are presenting with
- avoid, initially at least, psychiatric terms like depression, anxiety and panic.

For example, instead of ''Have you been feeling depressed?'' try ''Do your symptoms ever make you feel down or frustrated?''. Instead of ''Do you enjoy things any more?'' try ''How much of the time do your symptoms stop you enjoying things?''. When the patient replies that they can't enjoy things because they can't walk, etc, ask them how often they can enjoy the things they can do.

If you suspect your patient has been having panic attacks or is agoraphobic ask ''Do you ever have attacks where you have lots of symptoms all at once? When do these happen? Is it when you're outside or in certain situations?''.

Reading this you may ask yourself: why not just ask the patient directly about depression and anxiety? Many patients, and not just those with functional symptoms, regard anything ''psychological'' as mental weakness, madness, or an accusation that they are ''making up'' their symptoms. Being careful about how you ask questions about psychological symptoms and deferring them to later in the interview allows the patient to gain more confidence in you as a doctor. We find that once a patient trusts you are not going to use emotional symptoms ''against'' them they often will tell you important things they might otherwise not have done.

### History of abuse: to ask or not to ask?

Childhood abuse and neglect is another factor that makes people more prone to functional symptoms. But unless you have a long time to spend with the patient or they volunteer the information, we would suggest leaving questions about early life experiences and abuse until subsequent consultations (or to someone else). The evidence from primary care currently does not support the idea that quickly ''getting to the bottom of things'' in this way improves outcome.

### How long should all this take?

Like surgery, there is a limit to how quickly this can be done in a very complicated patient, even with the efficiencies we have suggested. Doing it in 10 minutes may be worse than not doing it all.

## EXAMINATION

The diagnosis of motor and sensory symptoms discussed below depends on demonstrating positive functional signs as well as the absence of signs of disease.[5] Most of these signs relate to inconsistency, either internal (for example, Hoover's sign reveals discrepancies in leg power) or external (for example, tubular field defect is inconsistent with the laws of optics).

When considering functional motor or sensory signs remember that:

- inconsistency is evidence that signs are functional, but does not tell you whether they are consciously or unconsciously produced
- the presence of a positive functional sign does not exclude the possibility that the patient also has disease—they may have both
- all physical signs have limited sensitivity, specificity, and inter-rater reliability.

## GENERAL SIGNS

### La belle indifférence

''La belle indifférence'', an apparent lack of concern about the nature or implications of symptoms or disability, is a clinical feature that continues to receive prominence in standard descriptions of conversion disorder. However, it has no discriminatory value. Furthermore, in our experience most

patients who are said to have ''la belle indifférence'' are either: (1) making an effort to appear cheerful in a conscious attempt to not be labelled as depressed; or (2) factitious (because they are deliberately making up the symptom they are not concerned about it).

### The laterality of the symptoms

Although often considered left sided, a recent systematic review found only a slight left sided preponderance (55–60%) for functional motor and sensory symptoms.

## FUNCTIONAL WEAKNESS

### Preliminary observation

Look for evidence of *inconsistency*. For example, compare their gait when they leave the consulting room to when they came in? What happens to their weakness when they have to take their clothes on or off or when they have to get something from their bag?

### Hoover's sign and other tests of ''complemental opposition''

Hoover's sign, described in 1908, is the most useful test for functional weakness and the only one that has been found in controlled studies to have good sensitivity and specificity.[6] It is a simple, repeatable test, which does not require skilled or surreptitious observation. The test relies on the principle that we extend our hip when flexing our contralateral hip against resistance (you can test this out on yourself). It can be performed in two ways:

- *Hip extension*—Look for a discrepancy between voluntary hip extension (which is often weak) and involuntary hip extension (which should be normal) when the opposite hip is being flexed against resistance (fig 3). It is important when testing involuntary hip extension to ask the patient to concentrate hard on their good leg.
- *Hip flexion*—Test hip flexion in the weak leg while keeping your hand under the good heel. Look for the absence of downward pressure in the good leg.

A similar principle can be used to examine weakness of hip abduction which may initially be weak but then come back to normal if tested simultaneously with the ''good side''.

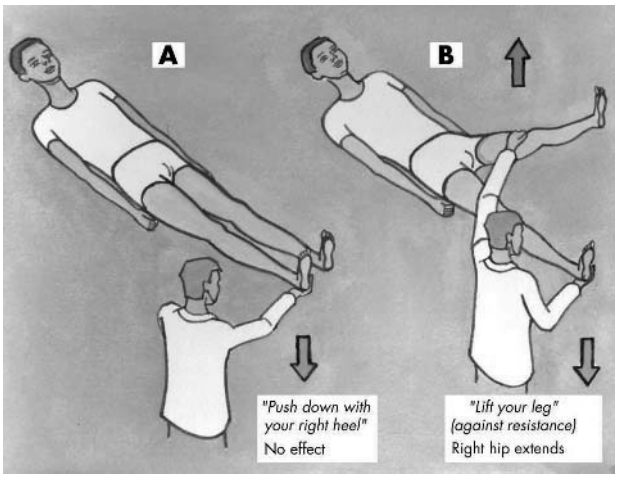



**Figure 3** Hoover's sign. (A) Hip extension is weak when tested directly. (B) Hip extension is normal when the patient is asked to flex the opposite hip. Reproduced from Stone *et al*,[5] with permission of the BMJ Publishing Group.

These tests, although useful, should be interpreted cautiously for the following reasons:

- Pain in the affected hip may produce greater weakness on direct, compared with indirect, testing as a result of attentional phenomena (related to pain rather than weakness)
- Cortical neglect can cause a positive Hoover's sign
- The test may be mildly positive in normal individuals because of a splinting effect
- None of the studies testing its utility were blinded and none mention the problem of neglect.

### Collapsing weakness

''Collapsing weakness'', the phenomenon in which a limb collapses from an instructed position with a light touch, is a common finding in patients with functional weakness. It is often associated with power that comes and goes or ''intermittency''. This should be not be described as ''intermittency of effort'' since you cannot directly assess someone's effort. Normal power can often be achieved transiently with encouragement, for example by saying to the patient, ''At the count of three, stop me from pushing down…''. Alternatively, gradually increase the force applied to the limb starting gently and building imperceptibly up to normal force.

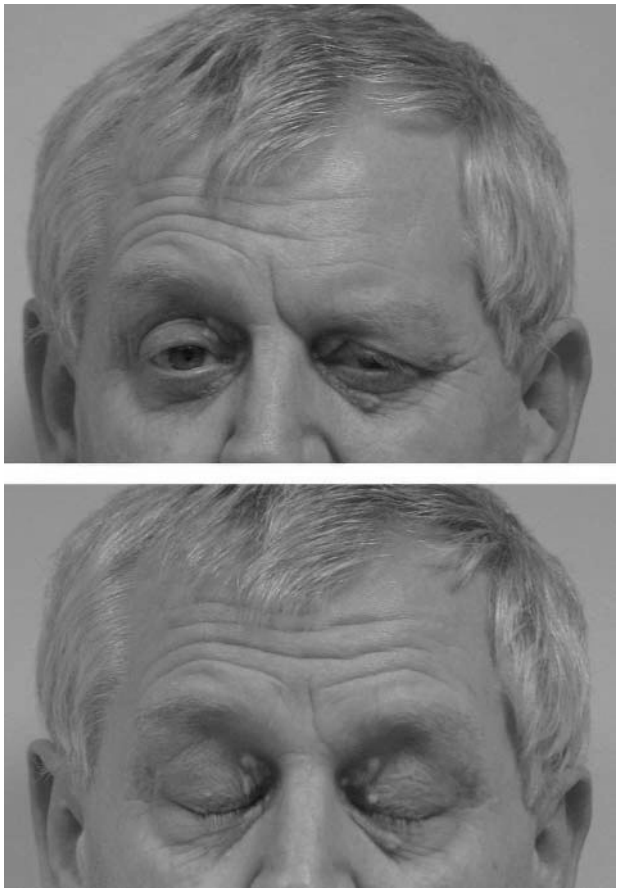


**Figure 4** Pseudoptosis. This man presented with photophobia and difficulty elevating the right side of his forehead. The photograph shows his normal resting state (upper panel) and normal movement of his forehead with his eyes shut (lower panel). There is overactivity of his orbicularis oculis which had been incorrectly interpreted as ptosis. It improved with gradual exposure to light. Reproduced from Stone,[17] with permission of Blackwells Publishing.

An inability to understand the instruction, pain in the relevant joint, being generally unwell, and a misguided eagerness of some patients to ''convince the doctor'' may cause a false result. These concerns have been vindicated in the small number of validity studies of this sign which have found that it is a rather poor discriminator between functional and disease related symptoms.[7]

### Functional weakness of the face, pseudoptosis, and ''wrong way tongue deviation''

Organic unilateral ptosis is usually associated with frontalis overactivity, whereas in pseudo-ptosis a *persistently* depressed eyebrow with a variable inability to elevate frontalis, overactivity of orbicularis, and photophobia is characteristic (fig 4). Apparent functional weakness of the lower half of the face and tongue deviation towards the normal rather than paretic side may occur because of overactivity of the affected side rather than underactivity.

### Other signs of functional weakness

- ''Co-contraction'' describes the contraction of an antagonist muscle—for example, triceps, when the agonist muscle, biceps, is being tested.
- When carrying out the ''arm-drop'', look for an unusually slow and jerky descent of the arm from an outstretched position on to the lap (better and less aggressive than dropping the arm on to the patient's face).
- Occasionally when the ''arm-drop'' test is performed the arms remain inexplicably elevated, so called ''pseudo waxy flexibility'', a phenomenon akin to that seen under hypnosis.
- It may be worth examining the strength of the sternocleidomastoid which is rarely weak in disease but may often be weak in unilateral functional weakness.

### Using sedation/hypnosis

In the altered mental state induced by sedative drugs or hypnosis, patients with functional weakness may begin to move their limbs normally again. Showing a video recording of this to the patient can be helpful in demonstrating to them the potential for reversibility.

### Important absent signs in functional weakness

Although the conventional examination of tone and reflexes should be normal, pain may increase tone, anxiety can increase reflexes, and in the patient with unilateral symptoms there may be mild reflex asymmetry, particularly if there is attentional interference from the patient. Pseudoclonus can occur, with irregular and variable amplitude. The plantar response should not be upgoing, but do not be surprised if the plantar response is mute on the affected side in functional weakness, particularly if there is pronounced sensory disturbance.

## FUNCTIONAL SENSORY DISTURBANCE

Functional sensory disturbance may be reported as a symptom or may be detected first by the examiner. While a number of functional sensory signs have been described, none appear to be specific and they should not therefore be used to make a diagnosis.

### Demarcation at the groin or shoulder

Patients may describe sensory loss that ends where the leg or arm ends, at the shoulder or groin.

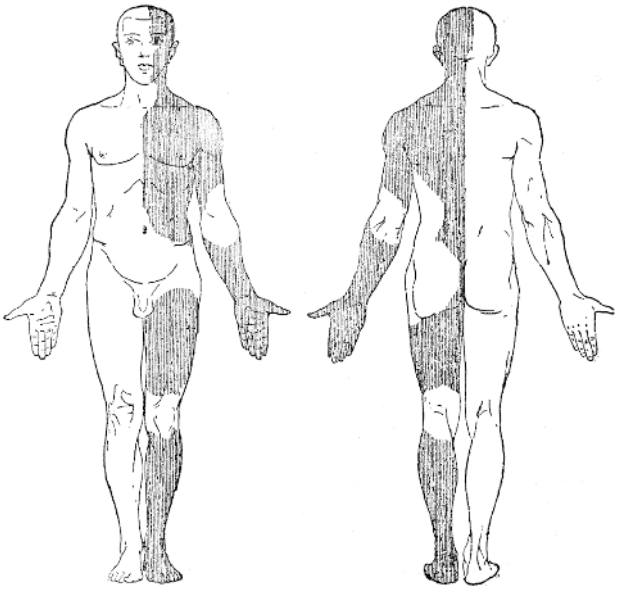


**Figure 5** Hemisensory disturbance. From *Charcot's clinical lectures on diseases of the nervous system*, volume 3.[18]

### The ''hemisensory syndrome'', midline splitting, and splitting of vibration sense

The hemisensory syndrome has been described for over a century and continues to be a well known but rarely studied clinical problem in neurology (fig 5). The intensity of the sensory disturbance often varies, and while it may be complete it is usually rather patchy, but with a distinct complaint from the patient that something is ''not right'' down one side or that they feel ''cut in half''.

Patients with hemisensory disturbance frequently complain of intermittent blurring of vision in the ipsilateral eye (asthenopia) and sometimes ipsilateral hearing problems as well. Hemisensory symptoms are increasingly recognised in patients with chronic generalised and regional pain.

''Midline splitting'', the exact splitting of sensation in the midline, is said to be a functional sign because cutaneous branches of the intercostal nerves overlap from the contralateral side, so organic sensory loss should be 1 or 2 cm from the midline. However, midline splitting can also occur in thalamic stroke. Therefore the finding of reversible contralateral thalamic and basal ganglia hypoactivation using single photon emission computed tomography (SPECT) in patients with unilateral functional sensory symptoms is intriguing in relation to this sign.[8]

Similarly, patients with disease should not report a difference in the sensation of a tuning fork placed over the left compared to the right side of the sternum or frontal bone, as the bone is a single unit and must vibrate as one. Studies of both midline splitting and splitting of vibration sense have found they are common in patients with disease and so cannot be recommended.[7]

### Tests involving doctor trickery

If you ask a patient to ''Say 'Yes' when you feel me touch you and 'No' when you don't'' they may indeed say ''no'' in the affected area. The problem in interpreting this test is firstly that the patient may be using ''no'' to mean ''not as much'', and secondly many patients will work out (at least in hindsight) that they were being tricked. This makes this test

**Table 2** Attack features that can help to distinguish non-epileptic attacks from epileptic seizures. Reproduced from Reuber and Elger,[3] with permission

| Observation | Non-epileptic seizures | Epileptic seizures |
|---|---|---|
| Situational onset | Occasional | Rare |
| Gradual onset | Common | Rare |
| Precipitated by stimuli (noise, light) | Occasional | Rare |
| Undulating motor activity | Common | Very rare |
| Asynchronous limb movements | Common | Rare |
| Purposeful movements | Occasional | Very rare |
| Rhythmic pelvic movements | Occasional | Rare |
| Opisthotonus, ''arc de cercle'' | Occasional | Very rare |
| Side-to-side head shaking | Common | Rare |
| Tongue biting (tip) | Occasional | Rare |
| Tongue biting (side) | Rare | Common |
| Prolonged ictal atonia | Occasional | Very rare |
| Ictal crying | Occasional | Very rare |
| Closed mouth in ''tonic phase'' | Occasional | Very rare |
| Vocalisation during ''tonic–clonic'' phase | Occasional | Very rare |
| Closed eyelids | Very common | Rare |
| Convulsion >2 minutes | Common | Very rare |
| Resistance to eyelid opening | Common | Very rare |
| Pupillary light reflex | Usually retained | Commonly absent |
| Reactivity during ''unconsciousness'' | Occasional | Very rare |
| Lack of cyanosis | Common | Rare |
| Rapid postictal reorientation | Common | Rare |

unhelpful if you want to adopt the transparent approach we favour.

## NON-EPILEPTIC ATTACKS

There is a stronger evidence base for approaching the diagnosis of functional/non-epileptic attacks or pseudoseizures.[3] As for functional weakness, the history may be suggestive, but will usually not be in itself diagnostic.

### Semiology

Non-epileptic attacks vary widely in their semiology but have been broadly divided into hyperkinetic/thrashing attacks and akinetic/motionless attacks. Table 2 lists some of the signs which have been tested in studies of both patients with non-epileptic attacks and epilepsy. In our experience symptoms of panic and dissociation are common in the prodromal phase, although patients may be reluctant to describe them.

As table 2 shows, there are no clinical signs of non-epileptic attacks which *never* occur in epilepsy, and apart from ictal electroencephalogram (EEG) abnormalities, there are no signs unique to epilepsy. For this reason, it is dangerous to use any of the listed signs in isolation to make a diagnosis.

There is a wide differential diagnosis for attacks that look ''odd''. ''Strangeness'' in itself should not lead you to a diagnosis of pseudoseizures. Frontal lobe seizures can look particularly bizarre. Paroxysmal movement disorders are another potential catch.

### Prolactin measurement

Serum prolactin is often elevated 15–20 minutes after a generalised tonic–clonic seizure and should be normal after a non-epileptic attack. However, prolactin rise has been demonstrated after syncope and found to be normal after partial seizures. The test can be useful but in our experience it is often carried out badly in practice, with no baseline sample and a post-ictal specimen that is either measured too early or too late. For this reason, we do not advocate its use outside specialist units.

### EEG and videotelemetry

EEG with videotelemetry remains the ''gold standard'' investigation for non-epileptic attacks. However, patients with partial epilepsy, particularly frontal lobe epilepsy, may not show any abnormalities on surface EEG recording when there is a deep ictal focus. In addition, some patients may not have attacks during monitoring.

### Using placebo and suggestion to induce attacks

The use of intravenous placebo, such as giving a bolus of intravenous saline with the suggestion that it will bring on an attack, is controversial as it may involve deception by the doctor (depending how the procedure is explained to the patient). Verbal suggestion alone may be effective.[9]

## FUNCTIONAL OR ''PSYCHOGENIC'' MOVEMENT DISORDERS

The diagnosis of a functional movement disorder is particularly challenging because of the unusual nature of some organic movement disorders. This is illustrated in the disproportionate number of movement disorders in cases where structural disease has been misdiagnosed as functional.

Further description of the features below can be found elsewhere[4] and useful video material can be found accompanying a recent textbook of movement disorders.[10]

There are some general features common to all functional movement disorders. These include:

- *Rapid onset*—This is more unusual in patients with organic movement disorder.
- *Variability*—Variability in frequency, amplitude, or distribution may be obvious during an examination or during observation at other times. It must be remembered that all movement disorders vary to some degree and will get worse during times of stress or worry, so minor variability is not helpful.
- *Improvement with distraction*—Distracting tasks include asking the patient to perform tests of mental concentration (for example, serial subtraction) or physical tasks

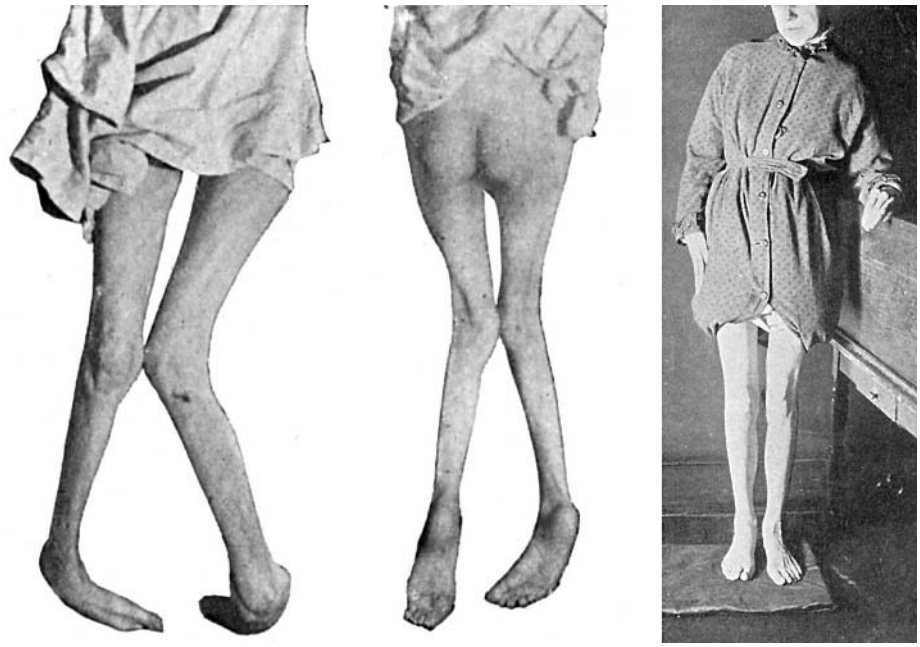


**Figure 6** A patient with paraplegia and psychogenic/functional dystonia of 14 years duration before (left and middle panels) and after (right panel) treatment with psychotherapy. Reproduced from Purves-Stewart and Worster-Drought.[19]

with their normal limbs (such as rapid alternating hand movements). The inverse, worsening with attention, may also occur. Again, organic movement disorders may be susceptible to these factors to a degree.

### Tremor

#### Entrainment

When testing for entrainment, a type of distraction task, the patient is asked to make a rhythmical movement with their normal hand or foot. Either the normal limb ''entrains'' to the same rhythm as the abnormal side or, more commonly, the requested rhythmical movement is irregular or incomplete. There is reasonable evidence for the reliability of this test from several controlled studies. A tapping frequency of 3 Hz may be more discriminant, and produce more variation, than a faster 5 Hz rate.

#### Tremor amplitude change with weights/co-activation sign

When weights are added to the affected limb, patients with functional tremor tend to have greater tremor amplitude whereas in those with organic tremor the tremor amplitude tends to diminish. This may be because of co-activation of agonists and antagonist, the so-called ''co-activation sign''. Related to this, patients with functional tremor may shake their limb more vigorously if it is held still.

#### Dystonia

Patients with hysterical contracture have been described since the late 19th century alongside organic dystonia (fig 6). Psychodynamic interpretations of dystonia (such as torticollis representing a ''turning away from responsibility'') encouraged misdiagnosis. When this error was realised there was a backlash and the diagnosis of psychogenic dystonia almost disappeared. More recently it is being recognised again and is included in the spectrum of ''fixed dystonia''.[11] The diagnosis is difficult but useful features include: an inverted foot or ''clenched fist'' onset in an adult, a fixed posture which is apparently present during sleep, and the presence of severe pain.

The ''gold standard'' for the diagnosis of functional dystonia is to demonstrate complete remission after administration of general anaesthesia, a suggestion, or placebo. Such a procedure, if handled carefully, may also be therapeutic. Be aware, however, that some types of organic dystonia may also remit spontaneously. A high proportion of patients with psychogenic dystonia have had an injury to the affected limb. There is an overlap between dystonia seen in relation to complex regional pain and psychogenic dystonia.

### Other movement disorders

Psychogenic myoclonus is described as a myoclonus with variable amplitude and frequency. It may be strikingly stimulus sensitive—for example, to fluorescent lighting or with elicitation of deep tendon reflexes—in which case the latency between stimulus and jerk is often long and variable. Laborious research methods may demonstrate the presence of a ''Bereitschaftspotential'' one second before the jerk whereas in cortical myoclonus of organic origin there may be a cortical spike around 20 ms before the movement.

Psychogenic hemifacial spasm, parkinsonism, and paroxysmal movement disorders (some of which are like ''partial'' non-epileptic attacks) are also described.[4]

## FUNCTIONAL GAIT DISTURBANCE

Several case series describe the features of functional gait disturbance[12] including one with video recordings.[13] Variability and improvement with distraction are noted but, as with movement disorders, just because a gait looks ''bizarre'' or ''ridiculous'' does not mean it is functional.

Unilateral functional weakness of a leg, if severe, tends to produce a characteristic gait in which the leg is dragged behind the body as a single unit, like a ''log'' (fig 7). The hip is either held in external or internal rotation so that the foot points inwards or outwards. This may be associated with a tendency to haul the leg on to an examination couch with both hands.

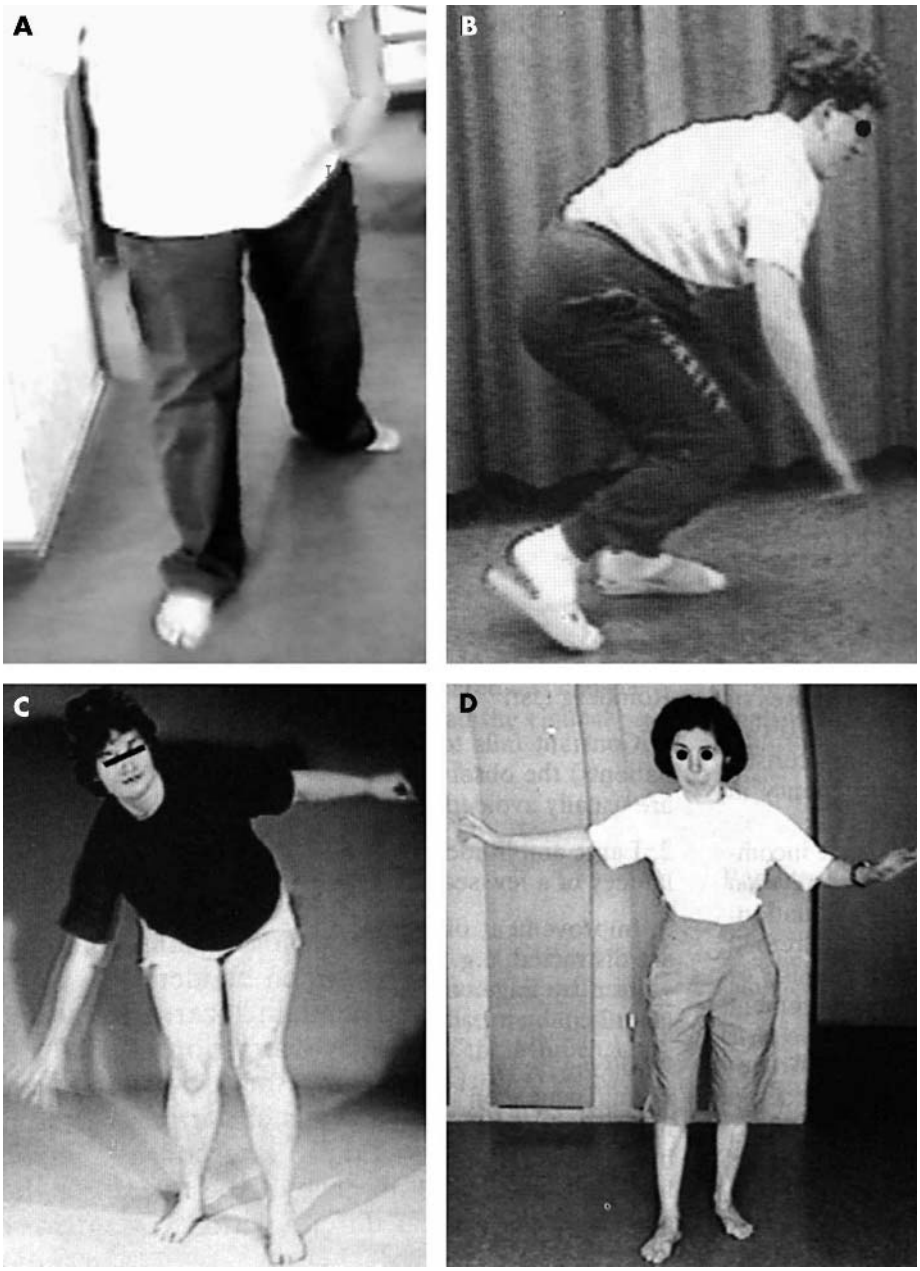



**Figure 7** Functional gait disorders. (A) Dragging monoplegic gait. (B) Uneconomic posture. (C) Pseudoataxia. (D) ''Walking on ice'' gait. Fig 7A reproduced from Stone *et al*,[5] with permission of the BMJ Publishing Group. Fig 7B–D reproduced from Lempert *et al*,[12] with permission of Karger Publishing.

Other features suggestive of a functional gait (fig 7) include:

- *Excessive slowness*—Dramatic delay in gait initiation and subsequent ''foot-sticking'' without the subsequent improvement seen in extrapyramidal disorders.
- *Falling towards or away from doctor*
- *''Walking on ice'' pattern*—The gait pattern of a normal person walking on slippery ground. Cautious, broad based steps with decreased stride length and height, stiff knees and ankles. Arms sometimes abducted as if on a tightrope.
- *Uneconomic postures with waste of muscle energy*—A gait with an eccentric displacement of centre of gravity such as standing and walking with flexion of hips and knees. Often associated with fear of falling.
- *Sudden knee buckling*—Patients usually prevent themselves from falling before they touch the ground. Knee buckling can occur in Huntington's chorea and cataplexy.
- *Pseudoataxia*—A gait characterised by crossed legs with or a generally unsteady gait with sudden sidesteps.

## OTHER SYMPTOMS

A brief summary of other symptoms (excluding cognition, pain, and fatigue) is given here mainly in order to direct the interested reader to the relevant literature

### Dizziness

A full discussion of how to determine whether dizziness is predominantly functional, and indeed whether such a distinction can be made, can be found elsewhere.[14] A variety of terms have been used to describe the intersection of vestibular and psychogenic factors in dizziness including, phobic postural vertigo, ''excessive awareness of normal sensation'', and space and motion discomfort. Some key points are:

- Anxiety and phobic avoidance of situations or head positions that bring on dizziness does not necessarily indicate a ''psychogenic'' aetiology
- On the other hand, such phobic avoidance may continue after the initial pathology has resolved
- Panic attacks presenting somatically with dizziness should be considered in the differential diagnosis of dizziness—look for a fear of embarrassment and inability to escape from situations in which it is likely to occur, such as supermarkets, as well as for other autonomic symptoms
- Physiological vestibular sensitivity to particular visual stimuli such as patterned lines or bright lights (sometimes called visual vertigo) may lead to symptoms that also come on in crowded places
- Depersonalisation and derealisation may be described by the patient as ''dizziness''. If this sensation is there all the time, the patient may have depersonalisation disorder (a chronic form of dissociation)
- Asking the patient to hyperventilate to see if that reproduces the symptoms might appear straightforward, but it has a high false positive rate in patients with dizziness cause by disease.

A full assessment of vestibular abnormalities, provoking stimuli and emotional symptoms can lead to tailored treatment in the form of vestibular rehabilitation and/or a cognitive behavioural approach regardless of the aetiology.

### Speech and swallowing symptoms

Typically, functional dysarthria resembles a stutter or is extremely slow with long hesitations that are hard to interrupt. The speech may be telegrammatic consisting only of the main verbs and nouns in a sentence. In its extreme form the patient may become mute. Be careful though, as these types of speech disturbance can also be seen in patients with disease.

Word finding difficulty is a common symptom in anyone with significant fatigue or concentration problems and may compound a functional dysarthria. True dysphasia as a more severe functional symptom, however, is rare.

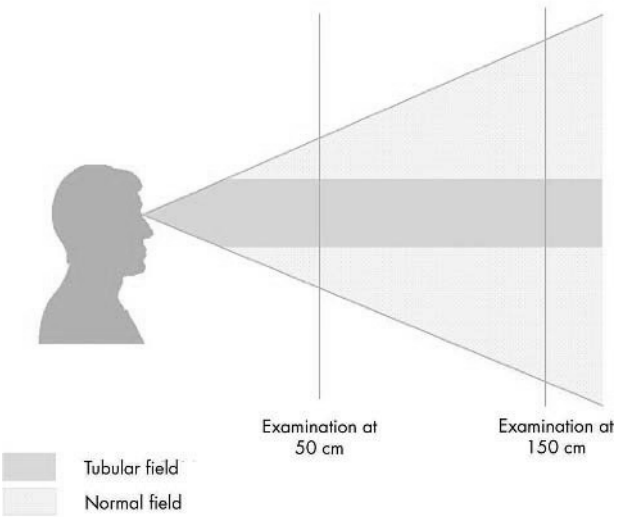



**Figure 8** A ''tubular'' field deficit is inconsistent with the laws of optics and eye physiology. You can detect striking tubular field at the bedside.

Dysphonia is a much more common functional speech complaint and there is now quite a large literature outlining approaches to diagnosis and management.[15] Often the clinical presentation is of whispering or hoarse speech that is initially thought to be laryngitis by the patient but then persists for months or years. The possibility of spasmodic adductor or abductor dysphonia must always be considered.

Globus pharyngis or functional dysphagia is common and there is also a sizeable literature about it. The patient normally complains of a sensation of a ''ball in the throat'' and investigations do not reveal a cause. There is controversy regarding what constitutes a full set of investigations for this symptom.

### Visual symptoms

Intermittent blurring of vision that returns to normal if the patient screws up their eyes tight then relaxes them again is commonly reported. Some of these patients have convergence or accommodative spasm, with a tendency for the convergence reflex to be transiently overactive, either unilaterally or bilaterally. In this situation lateral gaze restriction can sometimes appear to be present, but the presence of miosis may help to confirm the diagnosis. Voluntary nystagmus is described and appears to be a ''talent'' possessed by around 10% of the population.

Tests for functional visual acuity problems are described in detail elsewhere.[16] Simple bedside tests for a patient complaining of complete blindness are to ask them to sign their name or bring their fingers together in front of their eyes (which they should be able to do). They may have a normal response to menace and optokinetic nystagmus with a rotating drum (which equates to acuity of greater than 6/60). Decreased acuity in one eye can be assessed with a ''fogging test'' in which ''plus'' lenses of increasing power are placed in front of the ''good'' eye until the patient can only be using their ''bad'' eye to see.

Spiral or tubular fields are commonly seen clinically, are often asymptomatic, and can be elicited at the bedside. Remember to test the visual fields at two distances when looking for a tubular field (fig 8). Patients with functional hemianopia have been described who have homonymous hemianopia with both eyes open and then, inconsistent with this, have a monocular hemianopia in one eye with full fields in the other eye. Monocular diplopia or polyopia may be functional but can be caused by ocular pathology.

### Auditory symptoms

Basic tests for deafness rely on a startle response such as making a loud unexpected ''clap'' out of sight of the patient. Auditory brainstem evoked responses or evoked otoacoustic emissions may be necessary to fully investigate a patient with this symptom.

## INVESTIGATIONS

Even after finding clear positive evidence of functional symptoms, investigations are necessary in many (but not all) patients. Our criteria for performing tests are either (1) we are uncertain of the diagnosis, or (2) the patient remains uncertain of the diagnosis even though we are (and have done our best to explain it to them). Some patients really do not want tests; they just want a confident opinion. Others are only interested in the opinion of the scanner. As a general rule of thumb, if you are carrying out investigations to convince or reassure the patient, remember that this may

only be temporarily effective in patients with severe health anxiety who can become ''addicted'' to the reassurance of investigations. Similarly, patients who are convinced they have a certain disease like multiple sclerosis, but are not in the least anxious about this possibility, will not necessarily accept a negative investigation anyway. In a sizeable number of patients, normal investigations will be helpful and can speed recovery.

Preferably investigations should be performed as quickly as possible, as protracted testing maintains a focus on looking for disease rather than on rehabilitation. The need to look for disease also needs to be balanced against the risk of uncovering laboratory or radiological abnormalities that have nothing to do with the symptoms but which may delay or disrupt positive management. If tests are abnormal and relevant then positive functional signs should not necessarily be ignored. It may be necessary to make two diagnoses—one of an organic disease such as multiple sclerosis and another of additional functional weakness or disability.

## CONCLUSIONS: ASSESSMENT AND DIAGNOSIS

Functional symptoms are one of the most common reasons for consulting a neurologist. The assessment of such patients can be made more efficient and interesting by adapting the history, obtaining all the symptoms early on, asking about illness beliefs, and being careful about how and when you ask about psychological symptoms. In making the diagnosis the presence of positive functional signs are of key importance but should be used cautiously. Finally, be prepared to make a diagnosis of additional functional disability in someone with a known organic disorder.

## ACKNOWLEDGEMENTS

We would like to thank our colleagues in Edinburgh, especially Dr Adam Zeman and Professor Charles Warlow, for their valuable collaboration and support of this work. Thanks also to Will Whitely, Rustam Al-Shahi, Malcolm McLeod, and Patrick Fox for comments on the manuscript.

### Authors' affiliations

**J Stone, A Carson, M Sharpe,** NHS Department of Clinical Neurosciences, Western General Hospital, and School of Molecular and Clinical Medicine, University of Edinburgh, Edinburgh, UK

## REFERENCES

1 **Carson AJ**, Ringbauer B, Stone J, *et al*. Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. *J Neurol Neurosurg Psychiatry* 2000;**68**:207–10.
► **Providing that ''one third'' figure and showing that disability and distress are comparable to patients with disease.**
2 **Wessely S**, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? *Lancet* 1999;**354**:936–9.
► **A review article examining the similarities between symptom syndromes in different medical specialities.**
3 **Reuber M**, Elger CE. Psychogenic nonepileptic seizures: a review and update. *Epilepsy and Behaviour* 2003;**4**:205–16.
► **A thorough recent review of the exponentially increasing literature on non-epileptic attacks.**
4 **Thomas M**, Jankovic J. Psychogenic movement disorders: diagnosis and management. *CNS Drugs* 2004;**18**:437–52.
► **A summary of the literature on functional movement disorders.**
5 **Stone J**, Zeman A, Sharpe M. Functional weakness and sensory disturbance. *J Neurol Neurosurg Psychiatry* 2002;**73**:241–5.
► **A review of physical signs of functional weakness and sensory disturbance.**
6 **Ziv I**, Djaldetti R, Zoldan Y, *et al*. Diagnosis of ''non-organic'' limb paresis by a novel objective motor assessment: the quantitative Hoover's test. *J Neurol* 1998;**245**:797–802.
► **A neurophysiological study supporting the validity of Hoover's sign. Proof that science is possible in this area.**
7 **Gould R**, Miller BL, Goldberg MA, *et al*. The validity of hysterical signs and symptoms. *J Nerv Ment Dis* 1986;**174**:593–7.
► **An often quoted study showing that so called ''hysterical signs'' such as ''la belle indifférence'', collapsing weakness and midline splitting were often found in patients presenting with acute stroke.**
8 **Vuilleumier P**, Chicherio C, Assal F, *et al*. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain* 2001;**124**:1077–90.
► **The most convincing functional imaging study of patients with functional motor and sensory symptoms.**
9 **McGonigal A**, Oto M, Russell AJ, *et al*. Outpatient video EEG recording in the diagnosis of non-epileptic seizures: a randomised controlled trial of simple suggestion techniques. *J Neurol Neurosurg Psychiatry* 2002;**72**:549–51.
► **Describing a method of inducing non-epileptic attacks during videotelemetry using simple suggestion rather than deceptive placebo.**
10 **Sawle G**, Brown P. *Movement disorders in clinical practice*. Oxford: Isis Medical Media, 1999.
► **Contains a CD Rom with several videos of patients with functional movement disorders including a demonstration of tremor entrainment.**
11 **Schrag A**, Trimble M, Quinn N, *et al*. The syndrome of fixed dystonia: an evaluation of 103 patients. *Brain* 2004;**127**:2360–72.
► **A clinical exploration of patients with permanently clenched fists, inverted feet and the like.**
12 **Lempert T**, Brandt T, Dieterich M, *et al*. How to identify psychogenic disorders of stance and gait. A video study in 37 patients. *J Neurol* 1991;**238**:140–6.
► **A practical and detailed review of functional gait disorder with plenty of photos.**
13 **Hayes MW**, Graham S, Heldorf P, *et al*. A video review of the diagnosis of psychogenic gait: appendix and commentary. *Mov Disord* 1999;**14**:914–21.
► **A very helpful ''video article'' of 22 gaits, half of whom have functional gaits. Great teaching material as it also includes ''standard'' gait abnormalities.**
14 **Furman JM**, Jacob RG. Psychiatric dizziness. *Neurology* 1997;**48**:1161–6.
► **A thoughtful review of the role of psychological factors in dizziness.**
15 **Roy N**. Functional dysphonia. *Curr Opin Otolaryngol Head Neck Surg* 2003;**11**:144–8.
► **An review of functional dysphonia.**
16 **Beatty S**. Non-organic visual loss. *Postgrad Med J* 1999;**75**:201–7.
► **An review of clinical signs used in the detection of functional visual symptoms.**
17 **Stone J**. Pseudoptosis. *Practical Neurology* 2002;**2**:364–5.
18 **Charcot JM**. *Clinical lectures on diseases of the nervous system*. London: New Sydenham Society, 1889.
19 **Purves-Stewart J**, Worster-Drought C. The psychoneuroses and psychoses. In: *Diagnosis of nervous diseases*. Baltimore: The Williams and Wilkins Company, 1952:661–758.

# 8. Casuïstiek formulieren

Om de bijeenkomsten zo effectief mogelijk te benutten en de rollenspelen zo realistisch mogelijk te laten verlopen, is het van belang dat u eigen casuïstiek voorbereid (welke u 'speelt' wanneer u de patiëntenrol vertolkt). Hiervoor maakt u gebruik van onderstaand casuïstiekformulier, zowel voor uzelf, om de patiënt beter 'neer te zetten', als voor uw collega die de huisartsenrol vertolkt en voor de observator.
Kies bij voorkeur een patiënt, niet de moeilijkste, die regelmatig uw spreekuur bezoekt met klachten waarvoor u geen duidelijke oorzaak kunt vinden.

# Casuïstiek formulier

## Biografische gegevens:

(fictieve) naam: _____

leeftijd: _____

geslacht: _____

samenlevingsverband: _____

beroep: _____

arbeidssituatie: _____

## Medische en psychosociale status:

*voorgeschiedenis*: _____

actuele situatie (reden van consult, klachten, hulpvraag, omstandigheden): _____

*onderzoeksbevindingen:* _____

(voorlopige) diagnose: _____

*hypotheses:* _____

*indrukken, gedachten, gevoelens, aandachtspunten en vragen:* _____

# Casuïstiek formulier

## Biografische gegevens:

(fictieve) naam: _____

leeftijd: _____

geslacht: _____

samenlevingsverband: _____

beroep: _____

arbeidssituatie: _____

## Medische en psychosociale status:

*voorgeschiedenis*: _____

actuele situatie (reden van consult, klachten, hulpvraag, omstandigheden): _____

*onderzoeksbevindingen:* _____

(voorlopige) diagnose: _____

*hypotheses:* _____

*indrukken, gedachten, gevoelens, aandachtspunten en vragen:* _____

# Casuïstiek formulier

## Biografische gegevens:

(fictieve) naam: _____

leeftijd: _____

geslacht: _____

samenlevingsverband: _____

beroep: _____

arbeidssituatie: _____

## Medische en psychosociale status:

*voorgeschiedenis*: _____

actuele situatie (reden van consult, klachten, hulpvraag, omstandigheden): _____

*onderzoeksbevindingen:* _____

(voorlopige) diagnose: _____

*hypotheses:* _____

*indrukken, gedachten, gevoelens, aandachtspunten en vragen:* _____

# 9. Oefeningen

1. **Eigen reflectie op SOLK en persoonlijke leerdoelen**
2. **Doorvragen over klachten conform SCEGS analyse**
3. **Samenvatten van bevindingen**
4. **Uitleg vicieuze cirkel/neerwaartse spiralen**
5. **Warming up**
6. **Effectief geruststellen**
7. **Afstemmen van verwachtingen**
8. **Omgaan met onzinnige verwijzingen (+ vb. brief)**
9. **Terugrapportage naar de huisarts**
10. **Integratie vaardigheden – patiënt met lastige SOLK**
11. **Bespreking terugrapportage huisarts**
12. **Implementatie opdracht tbv dagdeel 3**
13. **Huiswerkopdracht 'zelfevaluatie'**

## Notities

## Oefening 1 Eigen reflectie op solk + persoonlijke leerdoelen

| Reflectie huidige werkwijze door beantwoording van volgende vragen: |
|---|
| 1. Wat is uw mening over patiënten die naar u verwezen zijn met lichamelijk onverklaarde klachten. |
| |
| |
| 2. Verloopt het contact met patiënten met lichamelijk onverklaarde klachten anders dan met patiënten met lichamelijk verklaarde klachten? Zo ja, waarin dan? |
| |
| |
| 3. Bent u goed toegerust voor de benadering/aanpak van (patiënten met) lichamelijke klachten waarvoor geen lichamelijke oorzaak te vinden? |
| |
| |

Schrijf vervolgens uw **persoonlijke leerdoelen** op en check tijdens de training of u 'op koers' zit.

1.

2.

3.

## Notities

## Oefening 2 Doorvragen over klachten (scegs analyse) (33 min)

**Gebruik eigen casus.**

Tijdsbewaking:

| | |
|---|---|
| Voorbereiding (rolverdeling, doorlezen casus) | 3 min. |
| Rollenspel 1 | 7 min. |
| Nabespreking | 3 min. |
| Rollenspel 2 | 7 min. |
| Nabespreking | 3 min. |
| Rollenspel 3 | 7 min. |
| Nabespreking | 3 min. |

**Opdracht:** Vraag enkele minuten (maximaal zeven) actief door op de klachten (met SCEGS analyse als leidraad).

**Aandachtspunten:**

- Laat de patiënt de klacht concretiseren, vraag naar details.
- Laat alle elementen van de SCEGS analyse aan de orde komen.

**Valkuilen:**

- De arts neigt bij veel klachten tot inperken en bagatelliseren.
- De arts oppert te snel eigen interpretaties.
- De arts vraagt onvoldoende door en geeft te snel informatie.

## Notities

## Oefening 3 Samenvatten van bevindingen (25 min)

**Gebruik dezelfde casus als bij oefening 2.**

| | |
|---|---|
| Tijdsbewaking en rolverdeling afspreken | 1 min. |
| ▪ Rollenspel 1 | 5 min. |
| ▪ Nabespreking | 3 min. |
| ▪ Rollenspel 2 | 5 min. |
| ▪ Nabespreking | 3 min. |
| ▪ Rollenspel 3 | 5 min. |
| ▪ Nabespreking | 3 min. |

**Opdracht:** Vat de informatie uit de exploratiefase samen in positief-neutrale taal (anamnese, LO evenals Cognities v pt over de klachten als de impact van klachten op Emoties, Gedrag/activiteiten, Sociale Omgeving). Geef uitleg over uw verklaring en beloop van klachten.

**Aandachtspunten:**

- Benoem normale bevindingen in positieve termen.
- Vermijd termen die als ziektelabel gezien kunnen worden.
- Vat zowel klachten als cognities en emoties samen.
- Vat samen zonder af te zwakken.
- Benadruk dat de klacht reëel is.
- Sluit aan bij de informatiebehoefte van de patiënt en bij eventuele tegenstrijdigheden die naar voren zijn gekomen in de exploratieve fase.

**Valkuilen:**

- De arts zegt 'niets' gevonden te hebben.
- De arts suggereert, dat 'niets somatisch' betekent: 'dus psychisch'.
- De arts bagatelliseert de ernst van de klacht.
- De arts geeft uitleg in algemene termen en sluit te weinig aan bij de termen die de patiënt gebruikt.

## Notities

## Oefening 4 Uitleg vicieuze cirkels /neerwaartse spiralen (25 min)

**Gebruik dezelfde casus als bij oefening 2 en 3.**

| | |
|---|---|
| Tijdsbewaking en rolverdeling afspreken: | 1 min. |
| ▪ Rollenspel 1 | 5 min. |
| ▪ Nabespreking | 3 min. |
| ▪ Rollenspel 2 | 5 min. |
| ▪ Nabespreking | 3 min. |
| ▪ Rollenspel 3 | 5 min. |
| ▪ Nabespreking | 3 min. |

**Opdracht:** Leg de patiënt uit hoe vicieuze cirkels een rol kunnen spelen bij het in standhouden of verergeren van klachten, bijvoorbeeld door ongerustheid of een andere factor (emotie of cognitie uit de klachtenanalyse). Maak gebruik van een passende metafoor, die aansluit bij de informatie en beleving van de patiënt.

**Aandachtspunten:**
- Sluit aan bij de informatie van de patiënt uit de exploratieve fase.
- Gebruik papier en pen om de cirkel te kunnen tekenen.

**Valkuil:**
- De arts maakt de cirkel te ingewikkeld.

## Notities

## Oefening 5 Warming up (5 min)

**Gebruik een eigen casus.**

**Opdracht:** Neem patiënt voor je uit de afgelopen week, waarbij je onderzoek is afgerond en je werkhypothese SOLK is. Zoek bij deze patiënt naar in stand houdende factoren van de klachten. Teken vervolgens de klachten én de factoren die de klachten in stand houden of verergeren in een cirkel/ster/visgraat.

**Aandachtspunten:**

- Noteer voor jezelf welke informatie je eventueel mist, als je de cirkel niet rond kunt krijgen.
- Wees specifiek en aansprekend in het benoemen van de instand houdende factoren.

**Valkuil:**

- Je gebruikt te abstracte en/of te algemene termen voor de instand houdende factoren.

## Notities

## Oefening 6 Effectief geruststellen (20 min)

**Gebruik hiervoor de casus uit oefening 5.**

Tijdsbewaking: Oefening in tweetallen

| | |
|---|---|
| Rollenspel 1 | 5 min. |
| Nabespreking | 2 min. |
| Voorbereidingen rollenspel 2 | 1 min. |
| Rollenspel 2 | 5 min. |
| Nabespreking | 2 min. |

**Opdracht:** Start het gesprek met een samenvatting van het vorige consult (SCEGS) en vertel aan patiënt je bevindingen van LO en aanvullend onderzoek -> werkhypothese SOLK. Stel deze patiënt gerust volgende de techniek van het effectief geruststellen (pag. 36 onderaan). Als ongerustheid niet duidelijk is, exploreer deze dan eerst verder.

**Aandachtspunten:**

- Laat merken dat de bezorgdheid van de patiënt is overgekomen.
- Geef de bezorgdheid liever versterkt dan afgezwakt weer.
- Richt de geruststellende uitleg op punten waarover de patiënt ongerust is.
- Benoem uw ervaring met dit soort problemen.

**Valkuilen:**

- De arts vergeet de geruststelling te onderbouwen.
- De arts zwakt de bezorgdheid van de patiënt af.

## Notities

## Oefening 7 Afstemmen van verwachtingen (20 min)

**Gebruik een casus waarbij een medisch onderzoek relevant is.**

| | |
|---|---|
| Voorbespreking en rolverdeling | 1 min. |
| Rollenspel 1 | 5 min. |
| Nabespreken | 3 min. |
| Rollenspel 2 | 5 min. |
| Nabespreken | 3 min. |
| Rollenspel 3 | 5 min. |
| Nabespreken | 3 min. |

**Opdracht:** Vertel welk onderzoek je wilt gaan doen, wat het onderzoek inhoudt en wat een uitslag is bij een normaal testresultaat. Illustreer en visualiseer de informatie waar dat kan. Geef aanvullend toelichting bij informatiefolder of refereer aan (medische) website, waarop klachten en het betreffende onderzoek zijn beschreven.

**Aandachtspunten:**

- Geef aan wat de hulpvraag van de patiënt en de verwijzer aan jou als specialist is.
- Geef daarbij in eigen woorden terug waarover de patiënt zich zorgen maakt.
- Sluit in je uitleg aan bij de belevingswereld van de patiënt.

**Valkuil:**

- Je bent te technisch aan het uitleggen.
- Je informatie bereikt de patiënt niet.

## Oefening 8 Omgaan met onzinnige verwijzingen (15 min)

**Voorbereiding:** Lees de casus op de pagina hiernaast.

**Opdracht:** Welke reacties levert het lezen van de casus bij je op? Welke ideeën heb je over deze verwijzing?

**Aandachtspunten:**

- Sluit aan bij de patiënt
- Wees duidelijk in je aanpak
- Geef aan dat er verschillende partijen zijn met mogelijk verschillende verwachtingen creëer daarin de ruimte die je nodig voor een goede patiëntenzorg.

**Valkuil:**

- Je raakt geïrriteerd en laat dat merken (meestal non-verbaal)
- Je bent te meegaand met de patiënt en komt in de knel met je eigen beroepsopvatting

## Casus Bij oefening 8 over onzinnige verwijzing

**Een 60-jarige vrouw wordt naar de poli Neurologie verwezen door haar huisarts met als motivatie onderstaande brief van haar chiropractor:**

**"Geachte heer/mevrouw,**
Na een erg lang traject is deze patiënte naar u doorverwezen.
Patiënte is bij mij met herhaling getest op VBAI (de Klyne's Manouvre), Nystagmus (Cardinale velden) en evenwicht/proprioceptie (Rhomberg) en bijna altijd is vertigo en/of misselijkheid het resultaat.
Daarnaast was duidelijke vertigo en bijna in elkaar zakken door evenwichtsverlies, elke keer te zien bij cervicale compressie, distractie, bewegingsvrijheid zonder weerstand en bewegingsvrijheid met weerstand (maar juist bij het loslaten/ontspannen).
Op mijn verzoek is er een X-CWK gemaakt die weinig duidelijkheid gaf ivm de klachten van hoofdpijn en duizeligheid dus er is daarna een functie X-CWK gemaakt waaruit bleek, dat C1 niet veel beweegt tov de schedel (C0). Deze bevinding was niet genoteerd in het radiologische rapport maar kan wel te maken hebben met occipitalizatie en is naar mijn opinie wel afwijkend.
Hiernaast waren er orthopedische bevindingen van suboccipitale spierspanning met subluxatie C3-4 en lumbale facet gewricht subluxatie met hypertone spieren suboccipitaal en thoracolumbaal.
Het resultaat van mijn therapie was tijdelijke verlichting van de spierspanning en hoofdpijn (zeer matig) en weinig tot geen effect aan de vertigo.
De behandelingen zijn zeer voorzichtig uitgevoerd met speciale aandacht voor de mogelijke kwalen en waren dus palliatief, in afwachting van het eindelijk vaststellen van de juiste afwijkende/vernauwde bloedvat(en) en daarna de nodige behandeling uitvoeren om blijvende verbetering aan te bieden.

**Verdere Toelichtingen:**
Patiënte heeft via de huisarts afspraken gekregen bij drie verschillende neurologen tot het moment van haar besluit om zelf ervoor te betalen en nog belangrijker, het akkoord gaan van het MRI Centrum Rotterdam om door mijn verwijzing het gewenste onderzoek uit te laten voeren. Alle drie neurologen weigerde een MRI/MRA te maken want ze zagen de indicatie niet. Als gevolg van de op eigen kosten gemaakte MRI en voornamelijk de MRA bij het MRI Centrum Rotterdam is er advies gegeven op een Duplex-Doppler te maken. Die is inmiddels gemaakt maar niet van de vertebralis als die door de foramen magnum gaat en ook niet bij de Cikel van Willis en belangrijke takken (namelijk AICA/PICA).
Ik vind het noodzakelijk en de neuroradioloog van de MRI Centrum ook, dat er als vervolg een angiographie met contrast plaats vindt ter bevestiging van de intracraniale problemen en dan van uw kant de juiste behandeling uit te voeren. Ik stel uw beoordeling en visie zeer op prijs en zou graag een verslag voor ons dossier hebben nadat patiënte bij u is geweest.
Ik hoop u hiermee voldoende te hebben geïnformeerd, voor verdere informatie sta ik altijd tot uw beschikking.

Met vriendelijke groet,
Chiropractor X "

## Notities

## Oefening 9 Terugrapportage naar huisarts (25 min)

**Gebruik een eigen casus, bij voorkeur uit een van de eerdere oefeningen van vandaag.**

| | |
|---|---|
| Voorbereiding en rolverdeling | 1 min. |
| Rollenspel 1 | 5 min. |
| Nabespreken | 3 min. |
| Rollenspel 2 | 5 min. |
| Nabespreken | 3 min. |
| Rollenspel 3 | 5 min. |
| Nabespreken | 3 min. |

**Opdracht:** Stel de terugrapportage naar de huisarts op en betrek hierin een concreet advies voor de patiënt, gericht op het aanpakken of beïnvloeden van klachtenbestendigende factoren. Kies een activiteit die aansluit bij wat in de SCEGS analyse naar voren is gekomen (zoals vermijdingsgedrag, reacties vanuit de omgeving, omgang met de klachten in de werksituatie).

**Aandachtspunten:**

- Vermeld duidelijk wat je bevindingen zijn op basis van jouw informatie
- Check of de patiënt zich hierin kan vinden
- Bespreek in hoofdlijnen de terugrapportage naar de huisarts met de patiënt
- Weerstand is een teken, dat de patiënt serieus bezig is met je voorstel, laat je niet afschrikken en ga op zijn overwegingen in.

**Valkuil:**

- Je denkt voor de patiënt ipv deze te activeren
- Je bent te vaag en algemeen in de terugrapportage
- Je gaat te snel voor de patiënt.

## Notities

## Oefening 10 Integratie vaardigheden - patiënt met lastige SOLK (45 min)

**Voorbereiding in drietallen:**
**Ieder noteert in leesbare steekwoorden:**

- Geslacht, leeftijd en andere relevante kenmerken SOLKpt
- Hoofdklacht uit verwijsbrief
- Relevante informatie uit VG
- Welk LO is verricht plus bevindingen
- Aanvullende diagnostiek plus eventuele uitslag

**Opdracht arts:**

Lees samenvatting patiënt
Exploreer adhv SCEGS-analyse (6 min)
Zet een vaardigheid naar keuze in (6 min), zoals bv
- samenvatten van bevindingen en uitleg geven over SOLK
- afstemmen van verwachtingen
- uitleg onderzoek en betekenis normaal testresultaat
- gezamenlijke besluitvorming ten aanzien van beleid

**Opdracht observator:**

Bewaak de tijd (12 min spel, 3 min feedback)
Regisseer de oefening:
- welke vaardigheid wil de arts oefenen na exploratie?
- waarop wil de arts feedback hebben?

Besluit zo nodig tot een korte time-out
Observeer en notuleer
Regisseer de feedbackronde:
- arts –patiënt -observator (wat is top en heb je een tip?)
- noteer leerpunten/vragen voor plenaire uitwisseling

**Opdracht patiënt:**

Speel je eigen patiënt
Reageer realistisch op de arts, niet hilarisch
Geef beknopte informatie in geval van time-out aan de arts
Registreer hoe je de aanpak van de arts ervaart

Iedereen oefent en vervult elke rol een keer.

## Notities

## Oefening 11 Bespreking terugrapportage (20 min)

**Voorbereiding: In tweetallen.**
**Benodigd een kopie van terugrapportage van specialist aan de huisarts.**

| | |
|---|---|
| Nogmaals lezen van elkaars brief | 3 min. |
| Noteren van onduidelijkheden bij elkaars brief | 3 min. |
| Bespreken van reacties/suggesties brief A | 5 min. |
| Toelichting van auteur hierop | 2 min. |
| Bespreken van reacties/suggesties brief B | 5 min. |
| Toelichting van auteur hierop | 2 min. |

**Opdracht:** Voorzie de terugrapportage van alle essentiële punten, die in een brief aan de huisarts nodig zijn voor SOLK patiënt:

- Verwoording van de verwijsvraag
- Verwijsvraag is expliciet beantwoord
- Hulpvraag van de patient is benoemd
- Hulpvraag van patiënt is expliciet beantwoord
- Beschrijving van lichamelijk onderzoek en diagnostiek
- Beschrijving van eventuele aanvullende diagnostiek
- Uitleg aan pt over in stand houdende factoren van klacht bij deze patiënt
- Advies ter uitwerking aan patiënt en huisarts

**Valkuil:**

- De brief biedt geen houvast voor huisarts of patiënt
- De uitleg is onduidelijk

## Oefening 12 Implementatie opdracht

**In tweetallen presentatie aan de groep hoe de vaardigheden in de praktijk al dan niet met succes zijn toegepast adhv eigen casuïstiek** (maximaal 15 min voor presentatie en discussie)
**Beide personen hebben een actieve inbreng. Dat kan ook zijn ondersteuning met literatuurstudie.**

- Introductie casus patiënt met SOLK
- Hulpvraag patiënt en de verwijzer
- Aantal consulten voor exploratie, informeren en beleid
- Gebruikte interventies (SCEGS, uitleg/plausibele verklaring/internetsites, vicieuze cirkel, klachtenregistratie, voorbereiden op uitkomst van de test, terugrapportage naar de huisarts/leefstijladvies voor patiënt).
- Eventuele opname ter illustratie
- Reflecties: leerpunten, discussiepunt, vragen, stellingen
- Groepsdiscussie

## Notities

## Oefening 13 Zelfevaluatie

**Reflecteer op je consulten met SOLK patiënten aan de hand van de lijst met aandachtspunten hieronder. Noteer wat je goed afgaat en wat nog moeilijk voor je is.**

AANDACHTSPUNTEN voor de communicatie met SOLK patiënten

**Ik ben in staat om het patiëntperspectief te verhelderen door:**

1. de cognities (gedachten) van de patiënt over zijn klachten te exploreren
2. te vragen naar de impact van de klachten op het sociale leven van de patiënt
3. te vragen naar de impact van de klachten op de activiteiten van de patiënt
4. te vragen naar de emoties van de patiënt over zijn klachten
5. fysieke klachten, zoals aangegeven door de patiënt, te exploreren
6. de klachten van de patiënt als reëel te erkennen

**Ik ben in staat om de patiënt te informeren door:**

7. een samenvatting te geven waarin ik klachten en cognities, emoties, gedrag en reacties van sociale omgeving van de patiënt betrek
8. mijn bevindingen naar de patiënt toe positief te etiketteren en daarbij gebruik te maken van positieve termen
9. uit te leggen dat de klachten géén symptomen van ziekte zijn
10. uitleg te geven over de factoren die de klachten in stand houden
11. bij mijn uitleg over SOLK gebruik te maken van tekeningen
12. het patiëntperspectief t.a.v. klachten en behandelopties te benoemen
13. mijn eigen perspectief als arts t.a.v. klachten en behandelopties te benoemen
14. verschillen en overeenkomsten tussen mijn zienswijze en die van patiënt te benoemen
15. de patiënt de nodige tijd te geven om de informatie te verwerken

**Ik ben in staat om met de patiënt vervolgbeleid af te spreken door:**

16. waar nodig toekomstige diagnostische testen uit te leggen
17. effectief na te vragen of de patiënt het heeft begrepen
18. de patiënt te betrekken bij de besluitvorming over het vervolgbeleid
19. commitment van patiënt aan beleid en vervolgafspraken te benoemen
20. de uitvoerbaarheid van de behandeling en vervolgafspraken te benoemen
21. een samenvatting te geven van het vervolgbeleid
22. Ik ben in staat om tijdens de verschillende fasen van het consult effectief non-verbaal te communiceren met de patiënt

**Het onderzoeksproject is gesubsidieerd door Fonds Nuts Ohra, CZ Fonds en ZonMw.**

**Erasmus MC**
**Afdeling inwendige geneeskunde**
Postbus 2040
3000 CA Rotterdam
http://www.lichamelijkonverklaardeklachten.nl